

NIH Public Access

Author Manuscript

Paediatr Perinat Epidemiol. Author manuscript; available in PMC 2012 September 1

Published in final edited form as:

Paediatr Perinat Epidemiol. 2009 May ; 23(3): 186-198. doi:10.1111/j.1365-3016.2008.01006.x.

Disparities in the Prevalence of Cognitive Delay: How Early Do They Appear?

Marianne M. Hillemeier¹, George Farkas², Paul L. Morgan³, Molly A. Martin², and Steven A. Maczuga⁴

¹Department of Health Policy and Administration and Population Research Institute, Pennsylvania State University

²Department of Sociology and Population Research Institute, Pennsylvania State University

³Department of Educational Psychology, School Psychology, and Special Education and Population Research Institute, Pennsylvania State University

⁴Population Research Institute, Pennsylvania State University

Summary

Cognitively delayed children are at risk for poor mental and physical health throughout their lives. The economically disadvantaged and some race/ethnic groups are more likely to experience cognitive delay, however the age at which delays first emerge and the underlying mechanisms responsible for disparities are not well-understood. The objective of this study is to determine when socio-demographic disparities in cognitive functioning emerge, and identify predictors of low cognitive functioning in early childhood. Data come from 7,308 singleton and 1,463 non-singleton children in the Early Childhood Longitudinal Study-Birth Cohort (ECLS-B), a nationally representative cohort of children born in 2001. Multiple logistic regression analyses examine associations between socio-demographic characteristics and low cognitive functioning at 9 and 24 months, and whether gestational and birth-related factors mediate these associations.

Socio-demographic characteristics are statistically significant predictors of low cognitive functioning among singletons at 24 months, including the three lowest quintiles of socioeconomic status (lowest quintile, OR = 2.7, 95% confidence interval (CI), [1.7,4.1], nonwhite race/ethnicity (African American OR=1.8, 95% CI [1.3,2.5]; Hispanic OR=2.3, 95% CI [1.6,3.2]), and gender (male OR = 2.1, 95% CI [1.7,2.5]). Gestational and birth characteristics, including very low and moderately low birthweight, and very preterm and moderately preterm delivery, have strong associations with low cognitive functioning at 9 months (VLBW OR=55.0, 95% CI [28.3,107.9]; MLBW OR=3.6, 95% CI [2.6,5.1]; VPTD OR=3.6, 95% CI [2.0,6.7]; MPTD OR=2.4, 95% CI [1.7,3.5]), but weaker effects by 24 months (VLBW OR=3.7, 95% CI [2.3,5.9]; MLBW OR=1.8, 95% CI [1.4,2.3]; VPTD OR=1.8, 95% CI [1.1,2.9]; MPTD OR=0.9, 95% CI [0.7,1.3]). Results for non-singletons were similar. Gestational and birth characteristics do not mediate the socio-demographic disparities in cognitive functioning at 24 months. Socio-demographic disparities in poor cognitive functioning emerge by 24 months of age. Further investigation of processes whereby social disadvantage adversely affects development prior to 24 months is needed.

Children with cognitive delays are at high risk for a range of negative social outcomes, including school drop-out, delinquency, and unemployment,¹ and have comparatively poor

Address Correspondence to: Marianne M. Hillemeier, Department of Health Policy and Administration, Pennsylvania State University, 604 Ford Building, University Park, PA 16802, telephone 814-863-0873, fax 814-863-2905, mmh18@psu.edu..

physical and mental health in adulthood.^{2, 3} Preventing these adverse long-term outcomes requires identification of those factors elevating children's risk of cognitive delay, as well as the timing at which these factors exert their negative effects. Certain subpopulations, particularly the economically disadvantaged and members of some race/ethnic groups including African Americans and Hispanics, are known to be at disproportionate risk for cognitive delays and disabilities, at least by school-entry.^{4–9} However, the age at which these delays first emerge, and the mechanisms responsible for disparities in risk are not well-understood.

Known risk factors for cognitive delay include the conditions of the child's gestation (e.g., length of gestation; whether the mother smoked or drank alcohol during pregnancy) and birth (e.g., birthweight and nutritional status; whether there were complications at delivery).^{10–12} Resnick et al.¹¹ found that birthweight, congenital anomalies, and complications of labor predicted the occurrence of learning disabilities, and Stein et al.¹² reported that even moderately low birthweight children were at significantly greater risk for cognitive disability than normal birthweight children. Low cognitive functioning is also associated with socio-demographic characteristics (e.g., child's gender, parents' socioeconomic status, race/ethnicity). Smith, Brooks-Gunn, and Klebanov,¹³ for example, found that school children living in poverty scored much lower on measures of verbal ability, mathematics skill, and word recognition.

Few studies have comprehensively analyzed the effects of gestation, birth conditions, and a child's socio-demographic characteristics and family conditions on his or her cognitive development in early childhood, focusing instead on predicting an older child's cognitive functioning at or after school entry.¹⁴ In addition, few studies have analyzed the effects of the different sets of factors using samples of non-singletons, even though multiple births are increasingly common¹⁵ and patterns of risk may differ between singleton and non-singleton children.

The purpose of this paper is to determine when socio-demographic disparities in cognitive delay emerge, and to identify significant predictors of low cognitive functioning in early childhood. We extend previous research by analyzing a recently released, nationally representative dataset, the Early Childhood Longitudinal Study Birth Cohort (ECLS-B). The ECLS-B includes complete birth certificate data, information gathered from parents, and direct assessment of children's mental and motor proficiency. This study identifies a range of socio-demographic characteristics and gestational and birth-related risk factors that are associated with low cognitive functioning at 9 and 24 months of age. Because twin and higher-order multiple pregnancies are at increased risk for medical complications and early delivery,¹⁶ we report results of analyses performed separately for singleton and non-singleton children.

Data and Research Methods

Sample

The ECLS-B is a nationally representative, longitudinal cohort study that enrolled 10,221 children born in 2001. The study sample is based on birth certificate records and includes oversamples of Asian and Pacific Islanders, Native Americans and Alaska Natives, low birthweight (1,500–2,500 grams) and very low birthweight (less than 1,500 grams) children, and twins. At approximately 9 months (2001–2002) and 24 months after the child's birth (2003), ECLS-B field staff administered developmental assessments of the children and interviewed the children's parents.

We restrict our analytic sample to those children who completed the 9 and 24 month developmental assessments. This yields a final analytic sample of 7,308 singleton children and 1,463 non-singleton children (1,404 twins, 52 triplets, and 7 quadruplets). Although the analytic sample excludes some of the children in the full ECLS-B sample, the two samples appear similar: examination of percent distribution in each quintile of socioeconomic status (SES) revealed no statistically significant differences.

Measures

Low Cognitive Functioning—Our dependent variable derives from the mental scale of the Bayley Short Form—Research Edition (BSF-R), a condensed version of the Bayley Scales of Infant Development, Second Edition (BSID-II) that was administered at the 9 and 24 month assessments. In both the BSID-II and the BSF-R, the mental score is based on trained interviewers' direct assessments of children's age-appropriate cognitive development as manifested in tasks demonstrating memory, habituation, preverbal communication, problem-solving and concept attainment. The initial ECLS-B study protocol included the full BSID-II, but in response to time constraints apparent in the initial field testing, the shortened BSF-R version was developed by an expert advisory panel for the ECLS-B study using item response theory (IRT) methodology. The BSF-R was extensively tested to ensure that the psychometric properties of the BSID-II were maintained and that it successfully measures children's abilities across the entire ability distribution. The overall IRT reliability coefficient for the BSF-R, and completed certification procedures that included both a written examination and observation of their administration of all items in the assessment.

Children were asked to complete specific age-appropriate tasks (e.g., "turn pages in a book," "look for contents of a box," "put three cubes in a cup," "respond to a spoken request"). Low-scoring children were asked to complete additional basal items (e.g., "play with a string," "vocalize three different vowel sounds," "retain two cubes for 3 seconds"). High-scoring children were asked to complete additional ceiling items (e.g., "retrieve a toy," "use words to make wants known," "put nine cubes in a cup"). A mental scale score is provided in the ECLS-B at each assessment point. Child age was recorded at the time of administration of the Bayley assessment was recorded as chronological age minus the number of weeks preterm.

We created a dichotomous variable that was given a value of 1 for children scoring in the lowest 10 percent of the BSF-R mental scale, a percentile cut-point that has been used to define developmental delay in previous research.¹⁸ We also tested alternative specifications using 5 and 15 percent as cut-points, and the patterning of our results was similar. We used these cut-off scores to determine developmental delay because while the original BSID-II assessment provides cut-off scores for identifying delays, the abbreviated BSF-R available in the ECLS-B does not have strictly comparable criteria. We also estimated multiple linear regression models using mental scale score as a continuous outcome, finding results consistent with the logistic results presented here (estimates available from the authors). We present results using the dichotomous measure because our focus is on disparities in cognitive delay, which have implications for screening and intervention that do not pertain to differences at the middle or upper ends of the cognitive performance distribution.

Child Age—Although the ECLS-B study design specified developmental assessments at 9 and 24 months, in practice children's ages varied on either side of these targets. Child age in months was included to account for increases in Bayley scores with age within these ranges.

Child Sex—Females were the reference category, with male children coded as 1.

Socioeconomic Status (SES)—A composite measure was created at each time point by ECLS-B project staff to reflect the socioeconomic status of the household. This measure incorporated five SES components:

- 1) Father/male guardian's educational attainment
- 2) Mother/female guardian's educational attainment
- **3)** Father/male guardian's occupation. Occupation was coded using the Standard Occupational Classification Manual,¹⁹ and the codes were collapsed into 23 aggregated categories with one additional category for unemployed, retired, disabled, and unclassified workers. Occupation was then recoded to reflect the average of the 1989 General Social Survey prestige score, computed as the average of the corresponding prestige scores for the 2000 Census occupational categories covered by the ECLS-B occupation.¹⁷
- 4) Mother/female guardian's occupation. Occupation was computed as described above for father/male guardian's occupation.
- 5) Household income. Parents were asked whether their annual household income was either \$25,000 and less or greater than \$25,000, and they were also asked detailed range questions within the broad range they specified (e.g., for those in the upper range, they were asked whether their household income was \$25,001 to \$30,000, \$30,001 to \$35,000, etc.). The midpoint of the detailed range was used in the computation of the composite SES variable.

Not all parents responded to every question related to SES, with the proportion of data missing for each of the education and occupation measures less than 2 percent and the proportion missing detailed income information approximately 9 percent. Missing values were imputed using a hot deck methodology whereby the value reported by a respondent is assigned to a similar person with missing data on that item.¹⁷

For each component of SES, a z-score was computed with a mean of 0 and a standard deviation of 1, and then the z-scores were averaged to create the composite SES measure. In households where only one parent was present, not all components were defined and in those cases the composite measure represents the average of available components. A five category SES variable was created that represents the quintile of the distribution for the value of the composite SES of each child. The first quintile (SES1) represents the lowest category, and the fifth quintile (SES5) represents the highest category. In logistic regression modeling, we created four dummy variables to represent SES, with SES5 designated as the reference category. The 9 month models include SES information collected at that time point, and updated SES information provided by parents at 24 months was incorporated into the 24 month analyses.

Advanced Maternal Age—We created a dichotomous variable with a value equal to 1 for mothers aged 35 years or older at the time of the child's birth.

Marital Status—We created an indicator of the mother's marital status at the child's birth, where married mothers are the reference category and unmarried mothers (those reporting that they were separated, divorced, widowed, or never married) are coded as 1.

Race/Ethnicity—Race/ethnicity is included in the analyses because members of some race/ethnic groups including African Americans and Hispanics, have previously been found

to be at disproportionate risk for cognitive delay.^{5, 7} Understanding the mechanisms underlying this risk is of theoretical and policy interest, and therefore the present analyses incorporate social class and other potentially relevant covariates in addition to race/ethnicity. Race/ethnicity of the child was ascertained from parent responses to questions providing a fixed set of race/ethnic categories, with the option to choose more than one race. In our analyses non-Hispanic White is the reference category. The other categories are as follows: 1) African American; 2) Korean, Chinese, Indian, or Japanese; 3) Other Asian; 4) Native American; 5) Hawaiian/Pacific Islander; 6) mixed race; and 7) Hispanic. We were able to separate Korean, Chinese, Asian Indian, and Japanese infants from other Asian infants using parent report supplemented with information from birth certificates, and we considered these Asian groups separately because these children traditionally score higher on cognitive tests.²⁰

Medical Risk Factors—Children's outcomes may be influenced by numerous maternal prenatal medical conditions and risk factors.^{21–25} To account for these risk factors without adding numerous variables that could lead to multicollinearity problems in statistical modeling, we constructed a count of the medical risk factors present during pregnancy as recorded on the birth certificate from the following list: incompetent cervix, acute or chronic lung disease, chronic hypertension, pregnancy-induced hypertension, eclampsia, diabetes, hemoglobinopathy, cardiac disease, anemia, renal disease, genital herpes, oligohydramnios, uterine bleeding, Rh sensitization, previous birth weighing 4000+ grams, or previous preterm birth.

Behavioral Risk Factors—Adverse child outcomes have been associated with risk behaviors during pregnancy including alcohol use^{26, 27} and smoking.^{26, 28} Data from the birth certificate included information about whether or not the mother used alcohol during pregnancy, and whether or not she used tobacco during pregnancy. For each of these behaviors, a variable was coded 1 if use was reported, and 0 if it was not. The behavioral risk scale consisted of the sum of the two coded values, and ranged from 0 to 2.

Obstetric Procedures—As discussed above for medical risk factors, we created a count of the following obstetric procedures occurring during pregnancy, labor and/or delivery as recorded on the birth certificate: induction of labor, stimulation of labor, tocolysis, amniocentesis, and cesarean section.

Labor Complications—Labor complications are also implicated in adverse developmental outcomes,^{29, 30} and we constructed a count of the number of labor complications experienced as recorded on the birth certificate from the following list: abruptio placenta, anesthetic complications, dysfunctional labor, breech/malpresentation, cephalopelvic disproportion, cord prolapse, fetal distress, excessive bleeding, fever of >100 degrees, moderate/heavy meconium, precipitous labor (<3 hours), prolonged labor (>20 hours), placenta previa, or seizures during labor.

Preterm Delivery—We constructed two indicators regarding preterm delivery. The first indicates very preterm births. This was equal to 1 for births occurring at 32 weeks completed gestation. The second indicates moderately preterm births. This was equal to 1 for birth occurring between 33 and 36 weeks completed gestation.

Birthweight—We constructed two indicators for the child's birthweight. Very low birthweight was a dichotomous variable equal to 1 for births weighing 1500 grams. Moderately low birthweight was a dichotomous variable equal to 1 for births weighing 1,501–2,500 grams.

Congenital Anomaly—A dichotomous variable is set equal to 1 if any congenital anomaly was present at birth.

Analyses

We performed descriptive analyses for study variables in singleton and non-singleton children, and tested for statistically significant differences between the two groups using t-tests. We then used logistic regression models to test whether socio-demographic characteristics entered simultaneously (i.e., gender, socioeconomic status, marital status, maternal age over 35, race/ethnicity) are associated with cognitive delay, measured at both 9 months and 24 months of age. We also tested whether these associations are mediated by gestational and birth-related risk factors. Separate analyses were conducted for singleton and non-singleton children. These models incorporated sampling weights and design effects to appropriately account for oversampling of some population subgroups and the stratified cluster design of the ECLS-B. All analyses were performed with SAS version 9.1 statistical software.

Results

Table 1 shows descriptive statistics for the samples of singleton and non-singleton births, weighted to adjust for survey design. The age and gender distributions of the two groups are similar. However, likely due to their greater use of fertility-enhancing drugs, mothers who are older (age 35 or older), married, with higher socioeconomic status, and of White nonHispanic race/ethnicity are over-represented among the multiple births. These multiple births also involved more medical risk factors, obstetric procedures, labor complications, and pre-term and low birthweight births, and had a higher rate of congenital anomalies. At both 9 and 24 months, slightly under 10 percent of singletons fell into the low cognitive functioning group. This is because singletons are the great majority of all births, and we defined cognitive delay as falling into the bottom 10 percent of scores. Among multiple births, 24.6 percent fell into the bottom 10 percent at approximately 9 months of age, but only 17.5 percent were in the bottom 10 percent at approximately 24 months. Non-singletons, who are more likely to be preterm, have low birthweight, and experience other risk factors, also appear to make strong progress in overcoming the deleterious consequences of these factors as they age, at least from 9 to 24 months.

Table 2 displays the results of logistic regression analyses predicting cognitive delay for singletons, separately at 9 and 24 months. For each time period we present two regression models. The first set of regressions uses only socio-demographic variables as predictors; the second adds gestational and birth characteristics to the models. The numbers shown are the effects of each predictor on the odds of a child displaying cognitive delay, after statistically controlling the other variables in the equation.

We begin by examining the first and third columns of this table, showing the results using only exogenous socio-demographics as predictors at both 9 and 24 months of age. These regressions show the total effects associated with these variables on predicting cognitive delay at 9 and 24 months.

Males were more likely than females to fall into the cognitively delayed group at both 9 and 24 months of age. At 9 months, socioeconomic status is generally not statistically significantly associated with low cognitive functioning except for those in the second lowest quintile (SES2), who have significantly higher risk (OR=1.7, 95% CI, 1.2 to 2.5) relative to the highest SES group. By 24 months, however, significantly higher risks are seen in each of the lower quintiles compared with the reference group, with odds ratios ranging from 1.9 to 2.7. With SES variables controlled, whether or not the mother is 35 or older, and whether or

not she is unmarried do not exert significant effects. African-American children, both groups of Asian children, Hispanic children, and children of mixed race show significant and strongly increased risks of low-functioning at 24, but not at 9 months.

The full regression models are displayed in columns 2 and 4. Here gestational and birth characteristics are added to the equations. Doing so does not appreciably reduce the negative effects of the socio-demographic characteristics. This suggests that the effects of socio-demographic characteristics do not operate via gestational and birth characteristics, but rather occur over and above any such causal mechanisms, and are related to other factors that differ across socio-demographic groups.

A number of the gestational and birth characteristics show reasonably large effects at 9 months of age. The most powerful of these is very low birthweight, which increases a child's odds of low cognitive functioning at 9 months by a factor of 55.0. Next in size are very preterm and moderately low birthweight, which multiply a child's odds of low cognitive functioning by 3.6. Significantly increased 9-month odds are also observed for moderately preterm births and those with congenital anomalies.

Many of these effects decline dramatically by 24 months of age. Most importantly, the odds for very low birthweight decline from 55.0 to 3.7. Those for very pre-term and moderately low birthweight also decline substantially, although they remain statistically significant. The odds ratio for moderately preterm is no longer significant. While being born very pre-term and at both very low and moderately low birthweight still involve an increased risk of low cognitive functioning at 24 months, many children are apparently able to overcome these biophysical risk factors as they age.

The findings in Table 2 may be summarized as follows. First, a range of socio-demographic variables elevate a child's risk of low cognitive functioning at 24 months, but not at 9 months of age. Noteworthy among these risk-factors are being in three of the lower SES categories, and being African-American, Asian, Hispanic and mixed race. These effects are *not* in general attributable to the association between these socio-demographic characteristics and either gestational or birth characteristics.

Second, several of the gestational and birth characteristics increase a child's risk of low cognitive functioning. Noteworthy among these are being born very and moderately low birthweight, and very preterm. However, in general the magnitudes of effect of these factors decline strongly as the infant ages from 9 to 24 months. By 24 months of age, these effects are of similar magnitude to the independent risk factors associated with being male, having lower SES, and being African-American, Other Asian, or Hispanic.

Table 3 repeats these analyses for non-singleton births. Children who are also either very low birthweight, moderately low birthweight, and very or moderately preterm are particularly likely to display low cognitive functioning at 9 months, with odds of 14.8, 3.0, 4.3, and 3.6, respectively. However, and consistent with the results for singletons, these odds all decline substantially by 24 months, and in some cases lose statistical significance. (The loss of statistical significance is likely partly due to the smaller sample size for non-singletons.) Thus, as with singletons, when non-singletons age from 9 to 24 months they tend to overcome much of the risk associated with a pre-term and/or low birthweight delivery.

Non-singletons also show many of the patterns of socio-demographic effects observed for singletons - increased risk for males, low SES groups, and for African-Americans and Hispanics at 24 as compared with 9 months, and a failure of these risks to be accounted for by gestational- and birth-related risk factors. In sum, the results for non-singletons show

The results in Tables 2 and 3 are based on defining "low cognitive functioning" as falling into the bottom 10% of all student performance scores. To check on the robustness of these results to changes in this 10% cutoff value, we replicated these regressions using both 5% and 15% as cutoff values. In general, the overall pattern of results is the same. For singletons, with the 5% cutoff the medical risk factor and very preterm effects become stronger at 9 months. With the 15% cutoff, the most noticeable change is that, at 24 months, Native Americans have significantly elevated odds of low cognitive functioning, with a significant odds ratio of approximately 2 in both estimated equations. For non-singletons, with the 5% cutoff the male effect at 24 months becomes larger, rising to odds ratios of 2.7 in the first model and 2.9 in the second model. With the 15% cutoff, the most noteworthy differences are that the male effect at 24 months becomes smaller (but remains statistically significant), declining to odds ratios of 1.5 in each model. Also the SES3 coefficient at 24 months increases to odds of 1.9 in the first model and 2.0 in the second model. In addition, the African-American coefficient at 24 months remains significant, but the odds ratios decrease to 3.1 in the first model and 3.3 in the second model. Finally, the odds ratio associated with very low birthweight at 24 months decreases to 1.7 and becomes statistically insignificant.

We also tested the robustness of our results to alternative measures of SES by repeating the regressions in Tables 2 and 3, using each of the subscales composing SES (mother's education, father's education, mother's occupational prestige score, father's occupational prestige score, and household income) as predictors, one variable at a time. The results supported the principal patterns observed for SES effects in Tables 2 and 3 - that low SES increases the prevalence of cognitive delay more at 24 than at 9 months, particularly for non-singleton children - for some of these subscales, but not for others. Most supportive were the results for household income, which closely resembled those for the overall SES scale. The other SES subscales showed one or another deviation from the overall pattern. For mother's education, the effect for non-singletons was not stronger than that for singletons. For father's education, and for mother's occupational prestige, the effect for singletons was not stronger at 24 than at 9 months.

Discussion

This study sought to identify the nature and patterning of risks—both biological and sociodemographic--for cognitive delay among children during the first two years of life. Capitalizing on the availability of a new, nationally representative dataset containing both complete birth certificate information and standardized developmental assessments, we examined the effects of a range of biophysical and socio-demographic characteristics on the likelihood of low cognitive functioning. Consistent with previous research on smaller samples drawn from clinical settings, 3^{1-33} we found that biophysical factors including shortened gestation and low birthweight are strongly associated with elevated risk for low cognitive functioning. In contrast to many previous studies, cognitive assessments were available at two points during very young childhood, allowing for an examination of change over time. The effects of most gestational and birth characteristics, while still important, decline in magnitude over the interval between 9 and 24 months. Concomitantly, the influence of socio-demographic characteristics including SES and race/ethnicity increase, and by the time children reach 2 years of age, become roughly equal in size to the effects of the gestation and birth-related factors. Race/ethnic groups with elevated odds at the latter time point include African Americans, Hispanics, those reporting mixed race, and both

groups of Asians studied. While the results for Korean, Chinese, Indian and Japanese children are surprising because their cognitive performance at older ages has generally been shown to be favorable,²⁰ the results presented here should be interpreted with caution given the relatively small number of these children included in the ECLS-B.

Although rates of preterm birth and low birthweight are consistently elevated among socioeconomically disadvantaged and non-white women, ^{15, 34} these socio-demographic effects on risk of low cognitive functioning were not attributable to an increased likelihood of experiencing these gestational and birth characteristics. Instead, the risks associated with a child's socio-demographic characteristics operated largely independently from those mechanisms. This finding underscores the importance of determining the processes through which socioeconomic disadvantage adversely affects cognitive development in very early childhood. As Wachs ³⁵ documents, the range of possible influences on individual development is very broad. For example, multiple aspects of a child's physical health are related to developmental functioning and are more likely to be compromised among low income populations. Nutritional status has been repeatedly shown to be an important factor, with suboptimal caloric intake and deficiencies in specific nutrients including iron, iodine, and essential fatty acids known to adversely affect central nervous system functioning.³⁶⁻³⁹ Hazardous environmental exposures are more likely to occur in socio-economically disadvantaged populations,⁴⁰⁻⁴² and in many cases can lead to cognitive delay. Lead is the most well-studied environmental toxin in this regard,43 however exposure to other toxins including PCBs,^{44, 45} dioxins,⁴⁶ and mercury⁴⁷ have also been found to be problematic. The health of socioeconomically disadvantaged children is also at elevated risk due to inadequate access to high-quality health care.⁴⁸ Multiple factors contribute to this impaired access including lack of affordable health insurance,^{49, 50} an inadequate supply of health care providers practicing in low-income and minority communities,⁵¹ and perceived discrimination in health care facilities ill-equipped to treat culturally diverse and non-English-speaking families.^{52, 53} Characteristics of the home environment have also been shown to be associated with cognitive development,⁵⁴ and factors such as parenting practices differ across socio-demographic groups in ways that can impact a child's cognitive development.⁵⁵ Brooks-Gunn and Markman,²⁸ for example, find evidence of race/ethnic differences in several aspects of parenting including nurturance, discipline, teaching, and language, and suggest that these differences are a significant contributor to gaps in school readiness. The quality of substitute child care also has an important influence on developmental outcomes,⁵⁶ and socioeconomically disadvantaged families are much less likely to have access to optimal child care arrangements.⁵⁷

As rates of multiple births continue to rise, cognitive outcomes among this potentially at-risk population are of considerable interest. We found that, as was the case among singletons, both gestational/birth characteristics and socio-demographic factors were associated with increased risk of low cognitive functioning at 9 months, with the strongest effects associated with gestation 32 weeks. By 24 months, socio-demographic factors become increasingly important, and lower SES and African American and Hispanic race/ethnicity are associated with risks of low cognitive functioning that in many cases are higher than those seen among singletons. Further research is needed to explore the mechanisms underlying the heightened risks for these subgroups.

A limitation of our study is that the analyses rely heavily upon information reported on birth certificates. Although widely used, these data may be subject to error, particularly with regard to complications of pregnancy and delivery and substance use during pregnancy.⁵⁸ Also, the outcome of interest, low cognitive functioning, may have been measured with more precision at 24 months than at 9 months, since a wider range of behaviors can be observed as the child matures.

The study findings have important implications. Most significantly, assessment of risks for cognitive delay in early childhood should include not only well-recognized biological factors such as length of gestation and birthweight, but also multiple features of the child's social environment. While elevated odds of poor cognitive functioning at 9 months are most strongly associated with very low birthweight and being very preterm, by 24 months social factors are equally important in predicting cognitive delay. The change in risk patterns from 9 to 24 months suggests that early childhood is a crucial transition period for the promotion of cognitive growth, and that policy interventions focused on maximizing social, educational, and economic resources among disadvantaged families with young children may serve to reduce subsequent disparities in school readiness and academic achievement. In addition, the recommended developmental assessment schedule during this period, which includes routine surveillance during well child visits and a standardized assessment at 9 and 18 months,⁵⁹ may be insufficient in socio-economically disadvantaged populations. Moreover, a recent survey of pediatricians indicates that only about one-fourth adhere to current screening guidelines.⁶⁰ While much remains to be learned about the mechanisms through which social disadvantage translates into developmental delay even at the earliest ages, appropriate developmental screening and provision of high-quality remedial services when indicated are likely to have an important positive impact on subsequent cognitive growth.

Acknowledgments

Acknowledgements: This research was supported by the Eunice Kennedy Shriver National Institutes of Child Health and Human Development, grants 2R24HD041025 and R21HD058124.

References

- 1. Blackorby J, Wagner M. Longitudinal postschool outcomes of youth with disabilities. Findings from the National Longitudinal Transition Study. Exceptional Children. 1996; 62:399–414.
- Martin LT, Fitzmaurice GM, Kindlon DJ, Buka SL. Cognitive performance in childhood and early adult illness: a prospective cohort study. Journal of Epidemiology and Community Health. Aug; 2004 58(8):674–679. [PubMed: 15252070]
- Clark DO, Stump TE, Miller DK, Long JS. Educational disparities in the prevalence and consequence of physical vulnerability. Journals of Gerontology Series B: Psychological Sciences and Social Sciences. May; 2007 62(3):S193–197.
- 4. Brooks-Gunn J, Duncan GJ. The effects of poverty on children. The Future of Children. Summer-Fall;1997 7(2):55–71. [PubMed: 9299837]
- Donovan, S.; Cross, CC. Minority students in special and gifted education. National Academies Press; Washington DC: 2002.
- 6. Fryer RG, Levitt SD. The black-white test score gap through third grade. American Law and Economics Review. 2006; 8(2):249–281.
- Ferguson, R. Why America's black-white school achievement gap persists. In: Loury, GC.; Modood, T.; Teles, SM., editors. Ethnicity, social mobility, and public policy: comparing the US and UK. Cambridge University Press; Cambridge: 2005. p. 309-341.
- Jencks, C.; Phillips, M. The black-white test score gap. Brookings Institution Press; Washington DC: 1998.
- 9. Yeargin-Allsopp M, Drews CD, Decoufle P, Murphy CC. Mild mental retardation in black and white children in metropolitan Atlanta: a case-control study. American Journal of Public Health. Mar; 1995 85(3):324–328. [PubMed: 7892913]
- Reichman NE. Low birth weight and school readiness. The Future of Children. Spring;2005 15(1): 91–116. [PubMed: 16130543]
- Resnick MB, Gueorguieva RV, Carter RL, et al. The impact of low birth weight, perinatal conditions, and sociodemographic factors on educational outcome in kindergarten. Pediatrics. Dec. 1999 104(6):e74. [PubMed: 10586008]

- Stein RE, Siegel MJ, Bauman LJ. Are children of moderately low birth weight at increased risk for poor health? A new look at an old question. Pediatrics. Jul; 2006 118(1):217–223. [PubMed: 16818568]
- Smith, JR.; Brooks-Gunn, J.; Klebanov, PK. The consequences of living in poverty for young children's cognitive and verbal ability and early school achievement. In: Duncan, GJ.; Brooks-Gunn, J., editors. Consequences of growing up poor. Russell Sage Foundation Press; New York, NY: 1997.
- Stanton-Chapman TL, Chapman DA, Bainbridge NL, Scott KG. Identification of early risk factors for language impairment. Research in Developmental Disabilities. Nov-Dec;2002 23(6):390–405. [PubMed: 12426008]
- Hamilton BE, Minino AM, Martin JA, Kochanek KD, Strobino DM, Guyer B. Annual summary of vital statistics: 2005. Pediatrics. Feb; 2007 119(2):345–360. [PubMed: 17272625]
- Sutcliffe AG, Derom C. Follow-up of twins: health, behaviour, speech, language outcomes and implications for parents. Early Human Development. Jun; 2006 82(6):379–386. [PubMed: 16690232]
- Andreassen, C.; Fletcher, P.; Park, J. Early childhood longitudinal study, birth cohort (ECLS-B) psychometric report for the 2-year data collection. National Center for Educational Statistics, Institute of Educational Sciences, US Department of Education; Washington DC: 2007.
- Dale PS, Price TS, Bishop DV, Plomin R. Outcomes of early language delay: I. Predicting persistent and transient language difficulties at 3 and 4 years. Journal of Speech, Language, and Hearing Research. Jun; 2003 46(3):544–560.
- US Office of Management and Budget. Standard Occupational Classification Manual. Available at: http://www.bls.gov/soc/2000
- Goyette K, Xie Y. Educational expectations of Asian American youths: determinants and ethnic differences. Sociology of Education. 1999; 72(1):22–36.
- Ornoy A. Growth and neurodevelopmental outcome of children born to mothers with pregestational and gestational diabetes. Pediatric Endocrinology Reviews. Dec; 2005 3(2):104– 113. [PubMed: 16361984]
- Fischer MJ, Lehnerz SD, Hebert JR, Parikh CR. Kidney disease is an independent risk factor for adverse fetal and maternal outcomes in pregnancy. American Journal of Kidney Diseases. Mar; 2004 43(3):415–423. [PubMed: 14981599]
- 23. Habli M, Levine RJ, Qian C, Sibai B. Neonatal outcomes in pregnancies with preeclampsia or gestational hypertension and in normotensive pregnancies that delivered at 35, 36, or 37 weeks of gestation. American Journal of Obstetrics and Gynecology. Oct; 2007 197(4):406, e401–407. [PubMed: 17904980]
- Khairy P, Ouyang DW, Fernandes SM, Lee-Parritz A, Economy KE, Landzberg MJ. Pregnancy outcomes in women with congenital heart disease. Circulation. Jan 31; 2006 113(4):517–524. [PubMed: 16449731]
- Whitley R. Neonatal herpes simplex virus infection. Current Opinion in Infectious Diseases. Jun; 2004 17(3):243–246. [PubMed: 15166828]
- 26. Shankaran S, Lester BM, Das A, et al. Impact of maternal substance use during pregnancy on childhood outcome. Seminars in Fetal and Neonatal Medicine. Apr; 2007 12(2):143–150. [PubMed: 17317350]
- Kartin D, Grant TM, Streissguth AP, Sampson PD, Ernst CC. Three-year developmental outcomes in children with prenatal alcohol and drug exposure. Pediatric Physical Therapy. Fall;2002 14(3): 145–153. [PubMed: 17053697]
- Raatikainen K, Huurinainen P, Heinonen S. Smoking in early gestation or through pregnancy: a decision crucial to pregnancy outcome. Preventive Medicine. Jan; 2007 44(1):59–63. [PubMed: 16959307]
- Molkenboer JF, Roumen FJ, Smits LJ, Nijhuis JG. Birth weight and neurodevelopmental outcome of children at 2 years of age after planned vaginal delivery for breech presentation at term. American Journal of Obstetrics and Gynecology. Mar; 2006 194(3):624–629. [PubMed: 16522389]

- Spinillo A, Fazzi E, Stronati M, Ometto A, Capuzzo E, Guaschino S. Early morbidity and neurodevelopmental outcome in low-birthweight infants born after third trimester bleeding. American Journal of Perinatology. Mar; 1994 11(2):85–90. [PubMed: 7515239]
- Pinto-Martin JA, Whitaker AH, Feldman JF, Van Rossem R, Paneth N. Relation of cranial ultrasound abnormalities in low-birthweight infants to motor or cognitive performance at ages 2, 6, and 9 years. Developmental Medicine and Child Neurology. Dec; 1999 41(12):826–833. [PubMed: 10619281]
- 32. Hack M, Klein NK, Taylor HG. Long-term developmental outcomes of low birth weight infants. The Future of Children. Spring;1995 5(1):176–196. [PubMed: 7543353]
- Ment LR, Vohr B, Allan W, et al. Change in cognitive function over time in very low-birth-weight infants. Journal of the American Medical Association. Feb 12; 2003 289(6):705–711. [PubMed: 12585948]
- 34. Alexander GR, Slay M. Prematurity at birth: trends, racial disparities, and epidemiology. Mental Retardation and Developmental Disability Research Reviews. 2002; 8(4):215–220.
- 35. Wachs, TD. Necessary but not sufficient: the respective roles of single and multiple influences on individual development. American Psychological Association; Washington DC: 2000.
- Krassner MB. Diet and brain function. Nutrition Reviews. May; 1986 44(Suppl):12–15. [PubMed: 2908796]
- Lozoff B. Iron deficiency and child development. Food and Nutrition Bulletin. Dec; 2007 28(4 Suppl):S560–571. [PubMed: 18297894]
- Schurch B. Malnutrition and behavioral development: the nutrition variable. Journal of Nutrition. Aug; 1995 125(8 Suppl):2255S–2262S. [PubMed: 7542707]
- Uauy R, Peirano P, Hoffman D, Mena P, Birch D, Birch E. Role of essential fatty acids in the function of the developing nervous system. Lipids. Mar; 1996 31(Suppl):S167–176. [PubMed: 8729114]
- Chakraborty J, Zandbergen PA. Children at risk: measuring racial/ethnic disparities in potential exposure to air pollution at school and home. Journal of Epidemiology and Community Health. Dec; 2007 61(12):1074–1079. [PubMed: 18000130]
- Borrell LN, Factor-Litvak P, Wolff MS, Susser E, Matte TD. Effect of socioeconomic status on exposures to polychlorinated biphenyls (PCBs) and dichlorodiphenyldichloroethylene (DDE) among pregnant African-American women. Archives of Environmental Health. May; 2004 59(5): 250–255. [PubMed: 16201671]
- Apelberg BJ, Buckley TJ, White RH. Socioeconomic and racial disparities in cancer risk from air toxics in Maryland. Environmental Health Perspectives. Jun; 2005 113(6):693–699. [PubMed: 15929891]
- 43. Bellinger DC. Very low lead exposures and children's neurodevelopment. Current Opinion in Pediatrics. Apr; 2008 20(2):172–177. [PubMed: 18332714]
- Stewart PW, Reihman J, Lonky EI, Darvill TJ, Pagano J. Cognitive development in preschool children prenatally exposed to PCBs and MeHg. Neurotoxicology and Teratology. Jan-Feb;2003 25(1):11–22. [PubMed: 12633733]
- 45. Boersma ER, Lanting CI. Environmental exposure to polychlorinated biphenyls (PCBs) and dioxins. Consequences for longterm neurological and cognitive development of the child lactation. Advances in Experimental Medicine and Biology. 2000; 478:271–287. [PubMed: 11065080]
- 46. Vreugdenhil HJ, Lanting CI, Mulder PG, Boersma ER, Weisglas-Kuperus N. Effects of prenatal PCB and dioxin background exposure on cognitive and motor abilities in Dutch children at school age. Journal of Pediatrics. Jan; 2002 140(1):48–56. [PubMed: 11815763]
- Jedrychowski W, Jankowski J, Flak E, et al. Effects of prenatal exposure to mercury on cognitive and psychomotor function in one-year-old infants: epidemiologic cohort study in Poland. Annals of Epidemiology. Jun; 2006 16(6):439–447. [PubMed: 16275013]
- Flores G, Tomany-Korman SC. Racial and ethnic disparities in medical and dental health, access to care, and use of services in US children. Pediatrics. Feb; 2008 121(2):e286–298. [PubMed: 18195000]
- 49. Fairbrother GL, Emerson HP, Partridge L. How stable is medicaid coverage for children? Health Affairs (Millwood). Mar-Apr;2007 26(2):520–528.

- 50. Bloom B, Cohen RA. Summary health statistics for U.S. children: National Health Interview Survey, 2006. Vital and Health Statistics 10. Sep.2007 (234):1–79.
- 51. Devoe JE, Baez A, Angier H, Krois L, Edlund C, Carney PA. Insurance + access not equal to health care: typology of barriers to health care access for low-income families. Annals of Family Medicine. Nov-Dec;2007 5(6):511–518. [PubMed: 18025488]
- Clemans-Cope L, Kenney G. Low income parents' reports of communication problems with health care providers: effects of language and insurance. Public Health Reports. Mar-Apr;2007 122(2): 206–216. [PubMed: 17357363]
- 53. Blendon RJ, Buhr T, Cassidy EF, et al. Disparities in health: perspectives of a multi-ethnic, multi-racial America. Health Affairs (Millwood). Sep-Oct;2007 26(5):1437–1447.
- Tong S, Baghurst P, Vimpani G, McMichael A. Socioeconomic position, maternal IQ, home environment, and cognitive development. Journal of Pediatrics. Sep; 2007 151(3):284–288. 288, e281. [PubMed: 17719939]
- Power C, Jefferis BJ, Manor O, Hertzman C. The influence of birth weight and socioeconomic position on cognitive development: Does the early home and learning environment modify their effects? Journal of Pediatrics. Jan; 2006 148(1):54–61. [PubMed: 16423598]
- 56. Bradley RH, Vandell DL. Child care and the well-being of children. Archives of Pediatrics and Adolescent Medicine. Jul; 2007 161(7):669–676. [PubMed: 17606830]
- 57. Magnuson KA, Waldfogel J. Early childhood care and education: effects on ethnic and racial gaps in school readiness. The Future of Children. Spring;2005 15(1):169–196. [PubMed: 16130546]
- DiGiuseppe DL, Aron DC, Ranbom L, Harper DL, Rosenthal GE. Reliability of birth certificate data: a multi-hospital comparison to medical records information. Maternal and Child Health Journal. Sep; 2002 6(3):169–179. [PubMed: 12236664]
- American Academy of Pediatrics. Identifying infants and young children with developmental disorders in the medical home: an algorithm for developmental surveillance and screening. Pediatrics. 2006; 118(1):405–420. [PubMed: 16818591]
- Sand N, Silverstein M, Glascoe FP, Gupta VB, Tonniges TP, O'Connor KG. Pediatricians' reported practices regarding developmental screening: do guidelines work? Do they help? Pediatrics. Jul; 2005 116(1):174–179. [PubMed: 15995049]

Table 1

Singleton and Non-Singleton Demographics, Gestational and Birth Factors, and Cognitive Test Scores,^a Early Childhood Longitudinal Study-Birth Cohort (ECLS-B)

	Singleton	is (n=7,308)		Non-Singleto	ns (n=1,463)
	Mean	SD		Mean	SD
Child Age, Round 1 (months)	10.5	4.5		10.4	3.1
Child Age, Round 2 (months)	24.4	2.4		24.3	1.7
Male	51.2%			49.4%	
SES, First Quintile, Round 1	18.2%			15.6%	
SES, Second Quintile, Round 1	25.1%		*	19.6%	
SES, Third Quintile, Round 1	21.7%		*	18.7%	
SES, Fourth Quintile, Round 1	15.1%			17.5%	
SES, Fifth Quintile, Round 1	20.0%		*	28.6%	
SES, First Quintile, Round 2	20.0%		*	14.4%	
SES, Second Quintile, Round 2	20.0%		*	15.7%	
SES, Third Quintile, Round 2	20.0%			20.3%	
SES, Fourth Quintile, Round 2	20.0%			21.6%	
SES, Fifth Quintile, Round 2	20.0%		*	27.9%	
Maternal age=35 or older	13.6%		*	20.2%	
Marital status=unmarried, Round 1	33.6%		*	25.7%	
Marital status=unmarried, Round 2	32.4%		*	24.6%	
White	53.6%		*	63.6%	
African American	13.6%			13.1%	
Korean, Chinese, Indian or Japanese	1.2%			0.9%	
Other Asian	1.4%		*	0.5%	
Hispanic	25.5%		*	17.8%	
Native American	0.4%			0.2%	
Hawaiian\Pacific Islander	0.2%			0.2%	
Mixed Race	4.0%			3.7%	
Medical Risk Factor(s)	0.18	0.7	*	0.27	1.1
Behavior Risk Factor(s)	0.11	0.6		0.12	0.6
Obstetric procedures	0.58	1.1	*	0.85	1.1

NIH-PA Author Manuscript

NIH-PA Author Manuscript

	Singletons	: (n=7,308)		Non-Singleto	ons (n=1,463)
Labor complications	0.34	1.2	*	0.62	2.3
Very Pre-term 32 weeks	2.0%		*	15.6%	
Moderately Pre-term 33 to 36 weeks	8.1%		*	43.2%	
Very low birthweight 1500 gm	1.0%		*	10.0%	
Moderately low birthweight 1501–2500 gm	4.9%		*	46.5%	
Congenital Anomaly	4.8%		*	9.0%	
Age of Mother	27.2	12.6	*	29.5	18.5
Gestation (weeks)	39.5	10.3	*	36.0	7.6
Birth Weight (grams)	3346	750.9	*	2355	650.6
Bayley Mental Score, Round 1	76.9	25.2	*	71.9	18.5
Bayley Mental Score, Round 2	127.2	21.6	*	123.4	18.5
Low Cognitive Functioning, Round 1	9.5%		*	24.6%	
Low Cognitive Functioning, Round 2	9.8%		*	17.5%	
^a Weighted estimates; see text for details					
*					
Significant at p=0.05					

Table 2

Odds Ratios from Multiple Logistic Regressions Modeling Low Cognitive Functioning^{*a*} at Approximately 9 and 24 Months of Age, Singleton Children (n=7,308)

	9 Months		24 Months	
Child age (months)	0.2*[0.2,0.3]	0.2*[0.1,0.2]	0.6*[0.6,0.7]	0.6*[0.6,0.7]
Child sex=male	1.3*[1.1,1.6]	1.4*[1.2,1.7]	2.1*[1.6,2.5]	2.1*[1.7,2.5]
SES1	1.5 [1.0,2.3]	1.3 [0.8,2.0]	2.7*[1.8,4.1]	2.7*[1.7,4.1]
SES2	1.7*[1.2,2.5]	1.5 [1.0,2.2]	2.3*[1.5,3.3]	2.3*[1.5,3.3]
SES3	1.3 [0.9,1.7]	1.1 [0.8,1.5]	2.5*[1.7,3.8]	2.5*[1.7,3.7]
SES4	1.3 [0.9,1.8]	1.2 [0.8,1.8]	1.9*[1.3,2.9]	1.9*[1.3,2.9]
Maternal age=35 or older	0.9 [0.7,1.1]	0.8 [0.6,1.0]	1.1 [0.8,1.4]	1.1 [0.8,1.4]
Marital status=unmarried	1.1 [0.8,1.4]	1.0 [0.7,1.3]	1.2 [0.9,1.5]	1.2 [0.9,1.6]
African American	1.1 [0.8,1.6]	1.0 [0.7,1.5]	2.0*[1.5,2.7]	1.8*[1.3,2.5]
Korean, Chinese, Indian or Japanese	1.2 [0.8,1.7]	1.2 [0.8,1.9]	1.8*[1.1,2.9]	1.7*[1.1,2.8]
Other Asian	1.0 [0.6,1.6]	1.0 [0.6,1.6]	3.0*[2.0,4.6]	3.0*[2.0,4.5]
Hispanic	0.9 [0.6,1.3]	0.9 [0.6,1.3]	2.4*[1.7,3.3]	2.3*[1.6,3.2]
Native American	1.1 [0.6,2.1]	1.2 [0.6,2.3]	0.9 [0.4,2.0]	0.9 [0.4,2.1]
Hawaiian\Pacific Islander	1.3 [0.2,9.6]	1.4 [0.2,14.0]	1.2 [0.5,3.0]	1.1 [0.4,3.0]
Mixed Race	1.2 [0.8,1.7]	1.0 [0.7,1.5]	1.8*[1.2,3.0]	1.8*[1.1,2.9]
Medical Risk Factor(s)		1.3 [1.0,1.7]		1.0 [0.8,1.2]
Behavior Risk Factor(s)		1.2 [0.8,1.7]		0.8 [0.6,1.1]
Obstetric procedures		0.8*[0.7,0.9]		0.9 [0.8,1.1]
Labor complications		0.9 [0.8,1.1]		1.0 [0.8,1.2]
Very Pre-term 32 weeks		3.6*[2.0,6.7]		1.8*[1.1,2.9]
Moderately Pre-term, 33 to 36 weeks		2.4*[1.7,3.5]		0.9 [0.7,1.3]
Very low birthweight, 1500 gm		55.0*[28.3,107.9]		3.7*[2.3,5.9]
Moderately low birthweight, 1501–2500 gm		3.6*[2.6,5.1]		1.8*[1.4,2.3]
Congenital Anomaly		1.6*[1.0,2.4]		1.6*[1.1,2.3]

^aLowest 10% of Bayley Mental Score

*Significant at p=0.05

Table 3

Odds Ratios from Multiple Logistic Regressions Modeling Low Cognitive Functioning^a at Approximately 9 and 24 Months of Age, Non-Singleton Children (n= 1,463)

	9 M	Ionths	24 Months	
Child age (months)	0.5*[0.4,0.6]	0.3*[0.2,0.4]	0.7*[0.6,0.9]	0.7*[0.6,0.9]
Child sex=male	1.1 [0.8,1.4]	1.2 [0.8,1.7]	2.2*[1.6,2.9]	2.3*[1.7,3.1]
SES1	1.1 [0.6,2.1]	1.6 [0.8,3.1]	3.0*[1.4,6.4]	3.6*[1.7,7.8]
SES2	1.5 [0.7,2.9]	1.8 [1.0,3.5]	3.4*[1.8,6.3]	4.0*[2.1,7.4]
SES3	1.1 [0.6,2.0]	1.4 [0.8,2.4]	1.8*[1.0,3.0]	1.9*[1.1,3.1]
SES4	1.1 [0.7,1.8]	1.2 [0.7,2.1]	1.1 [0.6,1.9]	1.2 [0.7,2.0]
Maternal age=35 or older	1.2 [0.8,1.9]	1.3 [0.8,2.1]	1.4 [0.9,2.3]	1.6*[1.0,2.5]
Marital status=unmarried	1.1 [0.6,1.7]	1.2 [0.7,2.1]	0.8 [0.5,1.2]	0.7 [0.5,1.2]
African American	1.3 [0.7,2.3]	0.8 [0.4,1.6]	3.7*[2.3,6.0]	3.9*[2.5,6.0]
Korean, Chinese, Indian or Japanese	1.4 [0.5,3.7]	1.6 [0.6,4.2]	1.1 [0.2,5.2]	1.3 [0.3,6.5]
Other Asian	0.2 [0.1,1.6]	0.6 [0.1,4.8]	2.8 [0.7,12.0]	4.5 [1.0,20.7]
Hispanic	0.9 [0.6,1.5]	0.7 [0.4,1.1]	1.8 [1.0,3.5]	2.0*[1.0,3.7]
Native American	1.6 [0.7,3.7]	0.8 [0.4,1.8]	5.8*[1.1,31.3]	4.7*[1.0,20.9]
Hawaiian\Pacific Islander	0.9 [0.1,10.5]	0.4 [0.1,2.6]	3.4*[1.8,6.6]	2.7*[1.3,5.8]
Mixed Race	1.0 [0.5,2.1]	0.7 [0.3,1.4]	1.9 [0.7,5.0]	1.9 [0.7,5.3]
Medical Risk Factor(s)		0.9 [0.7,1.2]		1.1 [0.8,1.4]
Behavior Risk Factor(s)		0.5 [0.3,1.0]		1.0 [0.6,1.8]
Obstetric procedures		0.9 [0.7,1.2]		1.2 [0.9,1.6]
Labor complications		1.1 [0.8,1.3]		1.1 [0.8,1.4]
Very Pre-term 32 weeks		4.3*[2.1,9.1]		2.3*[1.1,4.8]
Moderately Pre-term, 33 to 36 weeks		3.6*[2.5,5.2]		1.3 [0.8,2.0]
Very low birthweight, 1500 gm		14.8*[8.8,24.9]		2.2*[1.1,4.5]
Moderately low birthweight, 1501-2500 gm		3.0*[2.0,4.4]		1.5 [1.0,2.4]
Congenital Anomaly		0.9 [0.4,2.0]		1.0 [0.6,1.8]

^aLowest 10% of Bayley Mental Score

* Significant at p=0.05