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Association Between Feeding Difficulties and Language Delay in Preterm Infants Using Bayley III

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Abstract

Objective—To evaluate the relationship between abnormal feeding patterns and language performance on the Bayley Scales of Infant Development-III (BSID-III) at 18–22 months adjusted age among a cohort of extremely premature infants.

Study design—This is a descriptive analysis of 1477 preterm infants born 26 weeks gestation or enrolled in a clinical trial between January 1, 2006 and March 18, 2008 at a NICHD Neonatal Research Network center who completed the 18 month Neurodevelopmental Follow-Up assessment. At 18–22 months adjusted age, a comprehensive neurodevelopmental evaluation was performed by certified examiners including the Receptive and Expressive Language Subscales of the BSID-III and a standardized adjusted age feeding behaviors and nutritional intake. Data were analyzed using bivariate and multilevel linear and logistic regression modeling.

Results—Abnormal feeding behaviors were reported in 193 (13%) of these infants at 18–22 months adjusted age. Abnormal feeding patterns, days of mechanical ventilation, hearing impairment and Gross Motor Functional Classification Score 2 each independently predicted lower composite language scores.

Conclusions—At 18 months adjusted age, premature infants with a history of feeding difficulties are more likely to have language delay. Neuromotor impairment and days of mechanical ventilation are both important risk factors associated with these outcomes.

Advances in neonatal intensive care have increased survival rates for extremely premature infants; however, concern remains for the long term neurodevelopmental (ND) and functional status of surviving infants. (1–6) Researchers have demonstrated a dynamic yet maturation dependent vulnerability in the development, maturation and differentiation of cerebral structures, including areas responsible for feeding and language. These

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*A list of members of the NICHD Neonatal Research Network is available at www.jpeds.com (Appendix).

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neuropathological risks are consistent with clinical data showing that prematurely born infants are more likely than term peers to have language delay and require additional educational resources at school entry, including speech therapy services. (7–9) Similarly, premature infants are more likely to have feeding difficulties compared with term peers and more likely to be evaluated in a feeding disorders clinic.(10)

Given that feeding and sucking behaviors are one of the earliest manifestations of motor control in a newborn which utilize overlapping neural pathways involved in both feeding and language.(11) We hypothesized that feeding difficulties in early infancy in prematurely born infants would be associated with an increased risk of language delay in early childhood. This study explores the relationship between feeding dysfunction and language development at 18 months of age in a cohort of extremely low birth weight infants (ELBW) < 1000 grams, using the Bayley Scales of Infant Development-III (BSID III) with a specific focus on the receptive and expressive language subscales.

Methods

This study is a descriptive analysis of a prospectively followed cohort of extremely premature infants followed in the NICHD Neonatal Research Network (NRN) Follow-Up Study who were born at 26 weeks (N=1100) or who were greater than 26 weeks gestation and enrolled in a NRN clinical trial (N=377). All infants have a comprehensive neurodevelopmental assessment at 18 months adjusted age.

Neonatal and maternal data were systematically collected from birth until hospital discharge, transfer, death or 120 days postnatal age, including demographic and neonatal variables which could potentially affect neurodevelopmental outcome.

Bronchopulmonary dysplasia (BPD) was defined as the need for supplemental oxygen at 36 weeks post-menstrual age. Necrotizing Enterocolitis (NEC) was defined as modified Bells Stage IIA or greater (12, 13). Early onset sepsis (within 72 hours of birth) and late onset sepsis (after 72 hours) were defined by a positive blood culture and antibiotic therapy for 5 days. Cultures positive for organisms generally considered contaminants were not counted as indicative of sepsis.

Cranial sonograms were reviewed by the radiologist at each center based on Papile criteria (14), with Grade 3 and 4 considered severe for this analysis. Periventricular Leukomalacia (PVL) was defined as cystic echolucencies in the periventricular white matter.

Hearing impairment was defined as a permanent hearing loss that interferes with the ability of the child to follow directions and communicate despite amplification.

Adjusted age- and sex-specific percentiles for height, weight, and head circumference were computed using the Centers for Disease Control and Prevention growth charts (15).

As defined in the NICHD Follow-Up Manual of Operations, parents are routinely questioned about feeding behaviors of the child at the time of the 18 month follow-up visit. Dysfunctional feeding behaviors were defined as any of the following: (1) physician order not to ingest feedings by mouth; (2) any need for gastrostomy or tube feedings; (3) gags/ chokes or coughs with oral feeds; (4) documented history of aspiration; (5) excessive drooling during feeds; or (6) difficulty swallowing. The type and consistency of the feedings the child receives was recorded, including table foods, soft foods, and/or thickened liquids.

A comprehensive neurodevelopmental evaluation was performed at 18–22 months adjusted age adjusted age. Certified examiners performed a standardized neurosensory examination

and the Cognitive and Receptive and Expressive Language Subscales of the Bayley Scales of Infant Development-III (BSID-III). Cognitive and language domains are reported as composite scores with a mean of 100 and $SD \pm 15$. Based on their BSID III language composite scores, children were classified into 3 groups: No delay (85+), mild delay (70–84) and moderate-severe delay (< 70). Children judged to be so severely developmentally delayed that they were untestable ($N=39$) were assigned a Cognitive score of 54 and a Composite Language Score of 46, per guidelines from the Harcourt manual. Receptive and Expressive Language subscale scores were generated based on scale of 1–19(16). Neurodevelopmental impairment (NDI), a composite outcome, was defined as one or more of the following: moderate to severe cerebral palsy with a Gross Motor Functional Classification Scale (17)(GMFCS) Level 2, a Bayley III Cognitive Score < 70 , bilateral blindness with vision $< 20-200$ or permanent hearing loss despite amplification which interferes with ability to understand or communicate.

Statistical Analyses

We conducted descriptive bivariate analyses comparing demographic and neonatal characteristics and neurodevelopmental outcomes for children with dysfunctional vs. normal feeding behaviors, using chi-square tests for categorical variables and analyses of variance for continuous variables. Next, we conducted a multilevel logistic regression model to determine which factors predicted dysfunctional feeding behaviors at 18 months when including demographic and neonatal characteristics (sex, birth weight, primary language, race, education, insurance status, multiple gestation, postnatal steroids, ventilator days, severe grade 3–4 IVH and/or PVL, NEC), as well as post-neonatal hearing impairment and GMFCS at 18 months, and accounting for clustering of children within research center. Similarly, multilevel linear regression models were conducted to examine the relationship between feeding behaviors and Bayley III scores, controlling for the variables listed above and clustering within research centers. The multilevel models for categorical outcomes (i.e., dysfunctional feeding) were conducted using the SAS (SAS Institute, Cary, NC) GLIMMIX procedure and the multilevel models for the continuous outcomes were conducted using the SAS MIXED procedure. We then conducted receiver operating characteristic (ROC) curve analyses to further explore the relationship between days on ventilation and dysfunctional feeding behaviors and to determine a cut point for days of ventilation that maximizes sensitivity and specificity for identifying children with dysfunctional feeding behaviors.

Results

Of the 2,678 infants born between January 1, 2006 and March 18, 2008 at one of the 20 participating NICHD Neonatal Research Network sites who did not have congenital infection or congenital anomalies, 1054 died prior to the 18 month follow-up assessment, 147 were lost to follow-up. We evaluated outcomes of the remaining 1,477 (91%) infants whose characteristics are shown in Table I. Comparisons between these 1,477 infants and 147 infants who were lost to follow-up indicated that those lost to follow-up were less likely to be white ($p=.033$), multiple gestation ($p < .001$), have early onset sepsis ($p=0.043$), and have parents with less than a high school education ($p < 0.001$); however, they had similar birth weight, gestational age and other neonatal morbidities.

Of the 1,477 children in the study, 193 (13%) were reported to have dysfunctional feeding behaviors at 18 months adjusted age. Compared with children who reported normal feeding behaviors, those with dysfunctional feeding patterns had significantly lower gestational age and birth weight (Table I). They were also more likely to be black, have IVH/PVL, late onset sepsis, NEC, BPD, have received postnatal steroids and spent more time on the ventilator. The spectrum of feeding abnormalities included 96 infants (6%) who were primarily tube fed at 18 months adjusted age, 110 infants (7%) with dysphagia and 45

infants (3%) who had a history of documented aspiration. Types of foods consumed in the diet varied based on feeding status. Children reported to have abnormal feeding were significantly more likely to receive primarily liquids (eg, Pediasure, KinderKal), thickened liquids, and soft baby food for their nutritional intake compared with those reported to have normal feeding behaviors.

Feeding difficulties were independently associated with days of mechanical ventilation (OR 1.02, 95% CI 1.02, 1.03; $p < .001$), hearing impairment (OR 2.38, 95% CI 1.06, 5.33; $p < .036$) and evidence of neuromotor impairment based on a GMFCS score ≥ 2 (OR 14, 95% CI 7.7, 25.3; $p < .001$; Table II available at www.jpeds.com). The ROC analyses indicated that a cut point of 34.5 days on ventilation results in sensitivity of 0.64 and specificity of 0.74 for identifying children with dysfunctional feeding patterns. Of the 1010 children with fewer than 34.5 days on ventilation, 69 (7%) had dysfunctional feeding patterns compared with 124 (27%) of the 462 children with 34.5 or more days on ventilation.

Infants with dysfunctional feeding behaviors had significantly lower cognitive and language subscale scores on the BSID-III (Table III). Eighty-seven (45%) of the children with dysfunctional feeding behaviors met NICHD criteria for NDI compared with 81 (6%) of those with normal feeding behaviors ($p < .001$). Interestingly, 64% of infants with dysfunctional feeding behaviors did not have any evidence of cerebral palsy (Table III).

Independent predictors of lower BSID language and/or cognitive scales are shown in Table IV. GMFCS ≥ 2 , associated in most cases with moderate to severe cerebral palsy, had the strongest influence on performance on the BSID III cognitive and language scales at 18 months adjusted age resulting in a 21 point decrease in the mean cognitive composite score and a 19 point decrease in the language composite score. Dysfunctional feeding was independently associated with decreased performance in all areas assessed. One month of mechanical ventilation predicted similar decreases in these areas. Non-English speaking infants had significantly lower performance on the cognitive and all language scales compared with those who were primary English speakers. Children with hearing impairment had substantially lower language performance although their cognitive scores were similar to other infants in the cohort. Male sex, black race, lower maternal education, no private insurance and multiple births were also independently associated with lower cognitive and language scores in this cohort.

Gross motor functional classification score ≥ 2 captures a broader group of patients with neurologic injury and is a more sensitive indicator of at risk status. Of those with severe IVH/PVL, 21% (48/220) also had a GMFCS ≥ 2 , and 53% (48/91) with a GMFCS ≥ 2 had a history of severe IVH/PVL. If evaluated independently, each of these variables predicts cognitive and language outcomes; however, after controlling for other variables ($p < 0.5$; not shown) and including both IVH/PVL and GMFCS ≥ 2 in the models, IVH/PVL is no longer a significant predictor of language outcomes (Data not shown).

Compared with those with normal feeding behaviors, children with dysfunctional feeding behaviors, were more likely to have severe language delays (47% vs 11%; $p < .001$) and less likely to have mild delays (20% vs 30%; $p = .002$). In a multinomial logistic regression model, controlling for other factors, those with dysfunctional feeding behaviors were significantly more likely to have severe language delays (OR 2.51, 95% CI 1.55–4.07, $p < .001$). There was no significant difference for mild language delays (OR 0.84, 95% CI 0.52–1.37, $p = .494$).

Finally, we compared growth among those with dysfunctional vs. normal feeding patterns. Those with dysfunctional feeding patterns were in significantly lower percentiles for height, weight, and head circumference (Table III). These differences remained for height

(coefficient (SE) = -6.35 (2.43), $p=.009$) and head circumference percentiles (coefficient (SE) = -6.66 (2.66), $p=.012$), but not weight percentiles (coefficient (SE) = -4.33 (2.45), $p=.078$), after controlling for the other factors listed in Table IV. Tests of possible interactions between sex and dysfunctional feeding were not significant for any of the models, suggesting similar impact of dysfunctional feeding behaviors on growth across sex (not shown).

Discussion

Understanding patterns of brain injury in the prematurely born infants has improved over the past decade. Clinical, pathological and neuroimaging data suggest that these infants are uniquely vulnerable to injury to the developing language centers during this critical period of brain development and organization. The ability to identify infants at greatest risk for language delay would be useful as the numbers of surviving ELBW infants continues to increase. We evaluated language skills in early childhood in this cohort of extremely premature infants using the 2005 revision of the Bayley Scales of Infant Development III which evaluates receptive and expressive language skills separately from overall cognitive functioning. Separation of these two related but independent components of language functioning provides an opportunity to explore specific areas of neurocognitive impairment in this high risk population. Furthermore, we explored the relationship between a history of abnormal feeding patterns and language delay to determine if this could be used as an early predictor of those at risk for language delay.

Neurodevelopmental follow-up data confirm that prematurely born infants are indeed at risk for language delay at school entry. In a national longitudinal cohort of ELBW infants < 25 weeks gestation, Wolke et al noted that prematurely born infants had overall lower cognitive performance and an increased risk for speech and language difficulties, including auditory comprehension, expressive communication and articulation at school age. (18) Children <25 weeks gestation enrolled in a regional cohort study evaluated at 11 years of age were found to have significant cognitive impairment, including difficulties involving language and reading skills compared with term controls.(8)

Understanding the interplay between feeding dysfunction and later acquisition of language is poorly understood. Functional neuroimaging studies have shown overlap between cortical areas known to be involved in both feeding and language.(19) Therefore, we hypothesized that the development pathways responsible for early feeding competence may be linked to pathways involved in language development. Functional MRI studies have shown that language is predominantly a left hemisphere function, primarily involving the left perisylvian regions in the frontal, temporal and parietal lobes which correspond to Broca's and Wernicke's area. (20) At 8 year follow-up, Ment et al reported lower developmental and language scores among preterm infants enrolled in the Indomethacin Trial compared with term controls and differential regional activation in Broca's area among those with language delay (21). Gozza, et al demonstrated different patterns of connectivity in Wernicke's region and fewer cross hemispheric connections among a cohort of preterm infants at 8 years of age who had lower performance on cognitive and reading comprehension tests compared with term controls. (16) The authors postulated that these differences could represent delayed maturation of these neural pathways or a compensatory response due to difficulties with language processing. At present, there are limited data evaluating MRI findings in premature infants with a history of feeding difficulties; however, additional analyses are anticipated due to the increased routine utilization of routine MRI neuroimaging in this population.

Children in our study with gross motor impairment, defined by our diagnosis moderate/severe CP and/or GMFCS ≥ 2 , were most likely to have feeding difficulties at follow-up.

However, the fact that over 50% of those reported to have abnormal feeding behaviors had *no* evidence of motor impairment suggests that other variables are contributing to this outcome. Children with language delay are also more likely to have impaired growth at follow-up. Additional research is needed to understand how these factors may relate to the high overall rate of postnatal growth failure reported in preterm infants (13, 22, 23) and also among children in our cohort with a history of feeding difficulties.

Increased number of ventilator days beyond one month was strongly associated with both abnormal feeding patterns and language delay in our study group. Barlow and others have described the functional linkage in suck-swallow-respiration patterns and how these responses are modified by specific disease processes and experience. (11, 24) Barlow postulates that delayed activation and maturation of critical suckling pathways in infants requiring prolonged mechanical ventilation may contribute to an increased risk of oral motor difficulties by disrupting maturation of ororhythmic pattern needed for the development of normal sucking patterns.(25) Furthermore, Mizuno, et al reported that infants with chronic lung disease had abnormal patterns of sucking and generated weaker sucking pressures.(26) Research is ongoing to identify interventions that target experience dependent mechanisms involved in the suck-swallow reflex which may potentially decrease the risk for feeding difficulties in these patients long term.

A limitation of our study is the reliance on parent report for assessment of feeding and the lack of quantitative data measuring feeding competence during the neonatal period or the follow-up assessment. We also have limited information regarding timing or results of oropharyngeal motility studies. The feeding status of the 7% of children who were lost to follow-up is unknown; it is unclear how this may have affected our results since those infants were more likely to have other sociodemographic characteristics that are known negative predictors of language outcome. In addition, although PsychCorp reports a strong correlation for language items when compared with other instruments, the ability of this instrument to predict school age outcomes in a preterm population has not yet been systematically evaluated. A strength of this study is that it focuses on feeding behaviors at 18 months which limits potential overestimation of impact by including infants with transient feeding difficulties around the time of hospital discharge. An area of future research would be prospective evaluation of feeding competence in an ELBW population at discharge and follow-up.

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Abbreviations

BPD	Bronchopulmonary Dysplasia
BSID-III	Bayley Scales of Infant Development-III
ELBW	Extremely Low Birth Weight
GMFCS	Gross Motor Functional Classification Score
IVH	Intraventricular Hemorrhage

MRI	Magnetic Resonance Imaging
ND	Neurodevelopmental
NDI	Neurodevelopmental Impairment
NEC	Necrotizing Enterocolitis
NICHD	National Institute of Child Health and Human Development
NRN	Neonatal Research Network
PVL	Periventricular Leukomalacia

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Table I**Sample Characteristics by Feeding Status at 18 Months**

Characteristic	All (N=1,477)	Feeding Status		p
		Normal (n=1,284)	Dysfunctional (N=193)	
Male, n (%)	742 (50)	639 (50)	103 (53)	.372
Race/ethnicity, n (%)				
Black	595 (40)	500 (39)	95 (49)	.025
White	808 (55)	718 (56)	90 (47)	
Other	49 (3)	44 (3)	5 (3)	
Non-English, n (%)	230 (16)	205 (16)	25 (13)	.278
Education, n (%)				
< High school	271 (18)	240 (19)	31 (16)	.461
High school graduate	832 (56)	725 (56)	107 (55)	
College	374 (25)	319 (25)	55 (29)	
Private insurance, n (%)	527 (36)	465 (36)	62 (32)	.269
Multiple birth, n (%)	372 (25)	327 (25)	45 (23)	.506
Gestational age, mean $\pm$ SD	26 $\pm$ 2	26 $\pm$ 2	25 $\pm$ 1	<.001
Birth weight, mean $\pm$ SD	805 $\pm$ 168	815 $\pm$ 166	735 $\pm$ 162	<.001
Number of days on ventilation, mean $\pm$ SD	25 $\pm$ 27	22 $\pm$ 23	49 $\pm$ 34	<.001
BPD, n (%)	678 (46)	550 (43)	128 (66)	< .001
Postnatal steroids, n (%)	174 (12)	128 (10)	46 (24)	< .001
Severe IVH/PVL, n (%)	220 (15)	165 (13)	55 (29)	< .001
Early onset sepsis, n (%)	40 (3)	33 (3)	7 (4)	.403
Late onset sepsis, n (%)	560 (38)	461 (36)	99 (51)	< .001
NEC, n (%)	128 (9)	101 (8)	27 (14)	.005

Note: In some cases, percentages do not sum to 100% due to missing values.

Table II

Logistic Regression Model of Dysfunctional Oral Motor Skills at 18 Months

Variable	% Dysfunctional	OR	95% CI	p
Sex				
Male	14	1.14	0.78, 1.66	.501
Female	12	REF		
Birth weight (kg)	--	0.91	0.80, 1.04	.160
Race				
Black	16	1.32	0.87, 2.01	.196
White	11	REF		
Other	10	0.98	0.31, 3.09	.974
Language				
English	13	REF		
Non-English	11	1.24	0.69, 2.25	.474
Education				
College	15	1.79	0.93, 3.47	.083
High school graduate	13	1.36	0.80, 2.31	.256
Less than high school	11	REF		
Private insurance				
Yes	12	0.81	0.50, 1.31	.393
No	14	REF		
Multiple birth				
Yes	12	0.78	0.50, 1.21	.266
No	13	REF		
Postnatal steroids				
Yes	26	1.22	0.72, 2.05	.463
No	11	REF		
Ventilation days	--	1.02	1.02, 1.03	< .001
IVH/PVL				
Yes	25	1.14	0.71, 1.84	.583
No	11	REF		
NEC				
Yes	21	0.96	0.53, 1.74	.903
No	12	REF		
Hearing impairment				
Yes	36	2.38	1.06, 5.33	.036
No	12	REF		
GMFCS Level				
2+	70	14.00	7.74, 25.32	< .001
< 2	9	REF		

Note: N=1,415. Model also controls for research center.

Table III**Neurodevelopmental and Growth Outcomes by Feeding Status**

Outcome	All (N=1,477)	Feeding Status		
		Normal (n=1,284)	Dysfunctional (N=193)	p
Neurodevelopmental				
Hearing impairment, n (%)	47 (3)	30 (2)	17 (9)	< .001
Cerebral palsy, n (%)				
None	1300 (88)	1176 (92)	124 (64)	< .001
Mild	101 (7)	85 (7)	16 (8)	
Moderate/Severe	72 (5)	19 (1)	53 (27)	
GMFCS Level 2 or higher, n (%)	91 (6)	27 (2)	64 (33)	< .001
Cognitive Composite, mean ± SD	90 ± 15	92 ± 13	77 ± 18	< .001
Language Composite, mean ± SD	85 ± 17	87 ± 16	73 ± 20	< .001
Receptive Language [†] , mean ± SD	8 ± 3	8 ± 3	6 ± 3	< .001
Expressive Language [†] , mean ± SD	8 ± 3	8 ± 3	6 ± 3	< .001
Growth				
Height percentile, mean ± SD	36 ± 28	37 ± 28	25 ± 26	< .001
Weight percentile, mean ± SD	28 ± 29	29 ± 29	20 ± 24	< .001
Head circumference percentile, mean ± SD	40 ± 32	43 ± 32	25 ± 28	< .001

[†]Scores represent scale score ranging 1–19

Table IV

Linear Regression Models of Bayley III Scale Scores at 18 Months

Variable	Cognitive Composite [*]		Language Composite [*]		Receptive Language [†]		Expressive Language [†]	
	Coeff (SE)	p	Coeff (SE)	p	Coeff (SE)	p	Coeff (SE)	p
GMFCS Level 2+	-21.06 (1.48)	<.001	-19.15 (1.83)	<.001	-2.11 (0.38)	<.001	-2.30 (0.41)	<.001
Dysfunctional feeding	-5.45 (1.05)	<.001	-4.43 (1.29)	.001	-0.71 (0.24)	.003	-0.64 (0.25)	.011
Postnatal steroids exposure	-0.80 (1.11)	.469	0.55 (1.36)	.685	0.28 (0.25)	.268	-0.02 (0.27)	.927
Ventilation days	-0.09 (0.02)	<.001	-0.13 (0.02)	<.001	-0.02 (0.00)	<.001	-0.02 (0.00)	<.001
Severe IVH/PVL	-2.54 (0.92)	.006	-0.76 (1.12)	.497	-0.19 (0.20)	.363	-0.27 (0.22)	.215
NEC	-2.70 (1.12)	.016	0.84 (1.37)	.538	0.13 (0.25)	.617	0.24 (0.27)	.372
Hearing impairment	-2.52 (1.76)	.152	-10.51 (2.13)	<.001	-1.51 (0.43)	<.001	-1.45 (0.46)	.002
Male	-2.85 (0.64)	<.001	-3.77 (0.78)	<.001	-0.80 (0.14)	<.001	-0.57 (0.15)	<.001
Birth weight (kg)	0.63 (0.21)	.004	0.42 (0.26)	.106	0.08 (0.05)	.085	0.11 (0.05)	.038
Race								
Black	-2.43 (0.74)	.001	-3.71 (0.90)	<.001	-0.72 (0.16)	<.001	-0.35 (0.17)	.048
Other	-1.48 (1.81)	.413	-2.61 (2.24)	.244	-0.46 (0.40)	.248	-0.51 (0.43)	.237
Non-English	-2.11 (1.02)	.038	-6.34 (1.25)	<.001	-1.14 (0.23)	<.001	-1.00 (0.24)	<.001
Education								
College	4.35 (1.11)	<.001	5.79 (1.36)	<.001	1.12 (0.25)	<.001	0.97 (0.26)	<.001
High school graduate	1.70 (0.87)	.051	3.41 (1.07)	.001	0.54 (0.19)	.005	0.58 (0.21)	.005
Private insurance	2.28 (0.80)	.005	4.53 (0.97)	<.001	0.87 (0.18)	<.001	0.62 (0.19)	.001
Multiple birth	-2.80 (0.73)	<.001	-3.11 (0.89)	<.001	-0.41 (0.16)	.011	-0.64 (0.17)	<.001

* Scores based on mean 100 ± 15 SD;

† Scores based scale of 1–19

Note: Coeff=regression coefficient; SE=standard error; Models also control for research center. R² by model are: cognitive composite (R²=0.41), language composite (R²=0.35), receptive language (R²=0.26), and expressive language (R²=0.21).