Late Language Emergence at 24 Months: An Epidemiological Study of Prevalence, Predictors, and Covariates

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Purpose: The primary objectives of this study were to determine the prevalence of late language emergence (LLE) and to investigate the predictive status of maternal, family, and child variables.
Method: This is a prospective cohort study of 1,766 epidemiologically ascertained 24-month-old singleton children. The framework was an ecological model of child development encompassing a wide range of maternal, family, and child variables. Data were obtained using a postal questionnaire. Item analyses of the 6-item Communication scale of the Ages and Stages Questionnaire (ASQ; D. Bricker & J. Squires, 1999; J. Squires & D. Bricker, 1993; J. Squires, D. Bricker, & L. Potter, 1997; J. Squires, L. Potter, & D. Bricker, 1999) yielded a composite score encompassing comprehension as well as production items. One *SD* below the mean yielded good separation of affected from unaffected children. Analyses of bivariate relationships with maternal, family, and child variables were carried out, followed by multivariate logistic regression to predict LLE group membership.
Results: 13.4% of the sample showed LLE via the ASQ criterion, with 19.1% using the single item of "combining words." Risk for LLE at 24 months was not associated with particular strata of parental educational levels, socioeconomic resources, parental mental health, parenting practices, or family functioning. Significant predictors included familial history of LLE, male gender, and early neurobiological growth. Covariates included psychosocial indicators.
Conclusion: Results are congruent with models of language emergence and impairment that posit a strong role for neurobiological and genetic mechanisms of onset that operate across a wide variation in maternal and family characteristics.

KEY WORDS: late language emergence (LLE), late talkers

Children's language comprehension and production emerge between 12 and 24 months of age. Some otherwise healthy children require more time to begin talking, a condition described here as *late language emergence (LLE).* The reasons for such variation at the toddler stage of development are relatively unexplored. Variations in family or maternal resources are thought to play a role, although actual outcomes are mixed. More recently, genetic studies have focused on possible inherited risk for LLE (cf. Dale et al., 1998; Spinath, Price, Dale, & Plomin, 2004). LLE is widely assumed to be the first diagnostic symptom of children with language impairments. Tager-Flusberg and Cooper (1999) called for studies of early identification of specific language impairment (SLI) "with particular emphasis on predicting which late talkers develop SLI" (p. 1277).

A handful of studies have documented the phenomenon (Fenson, Reznick, Bates, Thal, & Pethick, 1994; Paul, 1996; Rescorla, 1989; Thal & Katich, 1996; Whitehurst & Fischel, 1994) and provided valuable descriptive and interpretive information. At the same time, with few exceptions, the studies were limited by small sample sizes and/or convenience sampling procedures and a small number of independent variables. In addition, much of the literature relies on relatively extensive parental questionnaires to document children's lexical development. These instruments are often infeasible for large-scale investigations of a wide range of possible predictors and covariates of LLE. A few alternatives have been developed, although they have not yet been used to evaluate predictors of LLE in single-born children in a study with a large number of participants and independent variables. The following factors remain unknown: (a) the prevalence of LLE in the general population of 24-month-old children and (b) the extent to which a wide range of maternal, family, and child variables are predictive of late talking. These issues are addressed in this investigation of an epidemiologically ascertained sample of 1,766 24-month-old singleton children who were participants in a large-scale investigation of health outcomes. Participants provided information on a wide range of targeted maternal, family, and child variables.

Ecological Model of Child Development

The participants in this study were recruited at birth (1995–1996) into an ongoing longitudinal study of children's health and developmental outcomes known as Randomly Ascertained Sample of Children born in Australia's Largest State (RASCALS), based in Perth, Australia. According to current census data, Western Australia is demographically similar to some states in the midwestern United States. For example, the population of Kansas is 2.7 million, and the population of Western Australia is 1.8 million. Also, in each state, most members of the population live in urban areas. The states are predominately Caucasian (86% for Kansas; 96% for Western Australia) and are native speakers of English, are well educated (86% high school completion in each state), and are family oriented (in Kansas, 55% of all families are couple families with children and 9% are sole-parent families; in Western Australia, 49% and 15%, respectively). On a wide variety of behavioral and biological assessments of children and adults, distributional outcomes conform to normative expectations for instruments normed in the United States or the United Kingdom.

This health outcomes study was guided by an ecological model of child development (Bronfenbrenner, 1979). This model views child development as a complex interplay between a child's biogenetic endowment and the proximal (i.e., maternal and family) and distal (i.e., societal) resources available to the child. The model recognizes that the proximal and distal resources available to the child will vary over the life course due to changes in circumstances for better or worse. Although this framework has not been used in previous studies of late talkers, the independent variables linked to late talking can all be placed in this model and categorized as relating to the child (neurobiological and genetic mechanisms) or the maternal or family environment. Consistent with the ecological framework, information was collected on a wide range of variables to describe maternal and family attributes and socioeconomic factors, concurrent with extensive documentation of children's perinatal status and developmental and health outcomes. Findings are summarized in a series of reports commissioned for public policy application (Silburn et al., 1996; Zubrick et al., 1995, 1997).

Candidate predictors of the emergence of language. Maternal and family variables—in particular, socioeconomic indictors—have been linked with the onset of language in young children. Mother's education level and family socioeconomic status (SES) are thought to be proxy measures of environmental support for language learning. Mother's education is reported to be associated with the amount of talking to children (cf. Hart & Risley, 1995; Hoff-Ginsberg, 1994; Wells, 1985), which in turn is predictive of vocabulary development in singletons (Dollaghan et al., 1999; Huttenlocher, Haight, Bryk, Seltzer, & Lyons, 1991) and twins (Lytton, Conway, & Sauve, 1977) and is positively associated with a number of language indices in the first 3 years, including verb tenses (Hart & Risley, 1995) and utterance length (Dollaghan et al., 1999). Furthermore, maternal and paternal education is reported to be a predictor of language impairment (Tomblin, 1996). LaBenz and LaBenz (1980) document language outcomes of a national sample of 20,137 children, followed from birth to 8 years of age, and report that mother's education predicted failure at age 8 years on language comprehension testing. The outcomes of these studies and the conclusions of Entwisle and Astone (1994)—that mother's education is the preferred index of "human capital" in the home when considering environmental contributions to young children's development—support consideration of levels of mother's education as a general risk index for children's language acquisition.

Recent studies, however, yield mixed evidence with regard to the chain of predictive effects sketched above. In an investigation of 108 low-income toddlers, Pan, Rowe, Singer, and Snow (2005) found that maternal talkativeness was not related to growth in children's vocabulary production in the 24- to 36-month period observed. Instead, maternal language and literacy (which was collinear with maternal education) was a significant predictor of growth; mothers with lower vocabularies/lower reading

levels had children with lower levels of vocabulary production in spontaneous samples. Pan et al. (2005) noted that the outcomes are compatible with either a genetic view of shared linguistic aptitude between mothers and children or an environmental input view that mothers with higher language and literature skills interact with their children differently than do mothers with lower language and literacy skills. They conclude that the maternal language and literacy effect, in their data, is not entirely mediated by maternal input, suggesting a need to consider influences beyond input. Pan et al. (2005) also investigated maternal depression as a predictor of growth and found it to have direct effects that increased with time. In their article, they note earlier reports that depressed mothers talk less to their children than do healthy mothers (Breznitz & Sherman, 1987) and caution that in their study, the outcome measure was children's vocabulary use in interactions with their mothers. Further investigation of maternal depression as a possible predictor is warranted.

Family SES levels are also implicated as risk factors. Although SES and maternal education are highly associated in the general population, there is strong reason to consider them as separate variables when evaluating children's development (cf. Entwisle & Astone, 1994). For example, in the Western Australia Child Health Survey (Zubrick et al., 1997), there was a stronger association between the education and employment status of caregivers and children's academic competence than income and family structure and academic outcomes. This finding would have been obscured if a composite measure had been used. With regard to SES, in general, as noted by Hoff-Ginsberg (1998), high SES mothers are reported to have higher levels of child-related adjustments that are thought to enhance children's language acquisition. However, as she notes in her study of 63 high- and mid-level SES children ages 18–29 months (Hoff-Ginsberg, 1998), the effects of maternal differences and child differences may be less detectable in the mid to high levels of SES. Total vocabulary size in young children is significantly related to SES, although at very low levels of association and/or localized at the low end of the distribution (Fenson et al., 1994; Rescorla & Alley, 2001; Rice, Spitz, & O'Brien, 1999; Wells, 1985). Pan et al. (2005) reported no effect for family income in their study of low-income toddlers. Overall, there is reason to regard mother's education and SES as risk factors for language emergence in toddlers, although the associations may not be strong, and the available evidence yields mixed outcomes.

LLE: Measurement, Prevalence, Candidate Predictors, and Covariates

Parent report measures of children's vocabulary and early word combinations are used to determine LLE status, described as *late talking,* which is typically benchmarked to the 24-month age level. Two widely used criteria are 50 words in reported vocabulary and the presence of 2–3 word combinations. Rescorla (1989) developed the Language Development Survey (LDS) as a parental report measure, which comprises a 310-word vocabulary checklist and a question about the presence and frequency of children's early word combinations. Prevalence estimates from this instrument, using a criterion of fewer than 50 words or no word combinations, yield estimates of 10–20% of children as late talkers (Klee et al., 1998; Rescorla, 1989; Rescorla & Achenbach, 2002; Rescorla & Alley, 2001; Rescorla, Hadick-Wiley, & Escarce, 1993). Paul (1996) also used this criterion to identify a group of late talkers for longitudinal assessment. The MacArthur Communicative Development Inventories: Words and Sentences (CDI/WS; Fenson et al., 1993) is a widely used parent report measure that uses a 680-word checklist as well as questions about early word combinations. According to the normative data provided by Fenson et al. (1993), at 24 months the bottom 5% of the distribution varies according to gender: For boys, it is an expressive vocabulary of 70 words; for girls, it is 48 words. Estimates of word combinations are available for children 16–30 months of age. Within this age range, for children who were reported to produce 50 words or less, 21% were reported to combine words "sometimes" and 1.3% were reported to do so "often." Children with an expressive vocabulary of fewer than 50 words or not combining words by 24 months represented the bottom 10% of the CDI norming sample (Fenson et al., 1993). Thal, Tobias, and Morrison (1991) used the CDI/WS criterion at or below the 10th percentile to identify late talkers in a follow-up study. Dale et al. (2003) used the 10th percentile; Feldman et al. (2005) reported five levels to define delays (i.e., 5th, 10th, 15th, 20th, and 25th percentiles).

Candidate predictors for LLE in children younger than 3 years have been identified from studies that have differentiated late talkers and controls and compared them on a select number of child, maternal, and family variables that have been linked theoretically and/or empirically to language development and language impairment. The child characteristics that have been examined include gender, gestational age, perinatal and obstetric risks, child behaviour, and motor development. The maternal characteristics include mother's education and mother–child interaction. The family influences include SES, birth order, family size, and family history of late talking (cf. Olswang, Rodriguez, & Timler, 1998; Whitehurst & Fischel, 1994, for reviews).

Among the candidate predictors, there is a strong, replicated gender risk for late talking. In prevalence studies, the proportion of boys who are late to talk is much higher than girls (Horwitz et al., 2003; Klee et al., 1998; Rescorla, 1989; Rescorla & Achenbach, 2002; Rescorla &

Alley, 2001; Rescorla et al., 1993), as well as in late talker cohort studies (Ellis Weismer, Murray Branch, & Miller, 1994; Paul, 2000; Rescorla, 2002; Whitehurst, Fischel, Arnold, & Lonigan, 1992). This strong gender effect seems to be a phenomenon of the lower tail of the distribution of children; it is not apparent across the full distribution where the gender effects in favor of girls are significant but small (Feldman et al., 2000; Fenson et al., 1994; Huttenlocher et al., 1991; Rescorla & Achenbach, 2002) or not evident at all (Bornstein, Tamis-LeMonda, & Maurice Haynes, 1999; Pan et al., 2005; Wells, 1985). Normal distributions that differ only modestly in their means can have very large relative differences at the extremes.

Children's birth history and perinatal status do not appear to be viable risk indicators for LLE. Late talkers do not have elevated rates of fetal and birth complications compared with controls (Paul, 1991; Rescorla et al., 1993; Whitehurst et al., 1992). In the most recent epidemiological study of SLI in kindergarten-aged children, adverse intrauterine and birth events were not risk exposures for SLI (Tomblin, Smith, & Zhang, 1997). Similarly, in a recent twin study, prenatal, perinatal, and obstetric risks were not associated with lower levels of language performance in twins compared with singletons at 20 and 36 months (Rutter, Thorpe, Greenwood, Northstone, & Golding, 2003). Marschik et al. (in press) reported that children (N = 15) who scored below the 10th percentile on an Austrian adaptation of the CDI/WS at 18 months had lower Apgar scores than did controls and that 5 late talkers (and none of the controls) required neonatal intensive care. Interestingly, 8 of the 15 late talkers scored within the normal range on the CDI/WS at 24 months.

Delayed motor development has been reported in several studies of late talkers. Rescorla and Alley (2001), Klee et al. (1998), and Carson et al. (1998) conducted direct assessment of motor abilities using standardized tests and reported that late talkers had lower levels of motor development than did controls. None of the children in these studies had developmental disorders or syndromes associated with delayed motor development.

Information about the influence of SES, parental education, and occupation on late talkers is very limited, in part because of the predominately convenience sampling methods that draw heavily from middle-class families (Rescorla, 2002). Using the CDI, Thal, Bates, Goodman, & Jahn-Samilo (1997) reported at 16–25 months a slight SES advantage for early talkers and a slight disadvantage for late talkers, although this trend was not confirmed by post hoc testing and was not present when the children were 21–31 months. Using the MacArthur Communicative Development Inventory (MCDI)–Short Form (Fenson et al., 1993), Horwitz et al. (2003) found SES and maternal education effects at 24 months, although their study also reported that living in a bilingual household was a strong predictor, thereby confounding the interpretation of LLE risk with bilingualism.

There is evidence that LLE influences the dynamics of parent–child interaction. Whitehurst and colleagues (1988) compared parental interactions for groups of late talkers, age-matched controls, and language-matched controls. They reported differences for late talkers compared with age-matched controls and similarities between late talkers and language-matched controls. They concluded that the differences in parent interaction between late talkers and age-matched normally developing children reflected parental adaptation to the language abilities of the children. Paul and Elwood (1991) reported similar results.

Feldman et al. (2005) call for investigation of the role of a positive family history of language disorders or delays as a potential predictor of outcomes. Hadley and Holt (2006) investigated maternal education and positive family history as predictors of growth in tense marking abilities in 2-year-old children with low levels of language development. Positive family history was related to differences in tense marking growth trajectories, whereas maternal education was not a predictor. The finding contrasted with Hart and Risley's (1995) finding that maternal education was associated with children's production of verb tenses. Hadley and Holt studied children in the low range of language abilities, whereas Hart and Risley studied children across the full range of the distribution of language abilities. This suggests that the influence of maternal education on performance is modulated by child characteristics. Hadley and Holt's study was the first to carry out growth curve analyses including positive familial history as a predictive variable for children's late talking. This extends the findings from previous studies that have reported higher levels of familial risk in late talkers compared with controls (Ellis Weismer et al., 1994; Paul, 1991; Rescorla & Schwartz, 1990).

Psychosocial development has been linked to late talking. The temperament and behavior characteristics of small numbers of late talkers have been investigated in several studies. Caulfield, Fischel, DeBaryshe, and Whitehurst (1989) studied 34 late talkers and 34 controls (24–32 months); Carson et al. (1998) studied 11 late talkers and 53 controls (24–26 months); Irwin, Carter, and Briggs-Gowan (2002) studied 14 late talkers and 14 controls (21–32 months); Paul and James (1990) studied 34 late talkers and 33 controls (24 months). Higher rates of problems were reported for late talkers compared with controls in these studies. In contrast to these studies, Rescorla and Achenbach (2002) did not find an association between language delay and behavior problems in a general population sample of 278 children 18–35 months of age.

Interpretation of the relation between psychosocial development and LLE is not straightforward. There are measurement confounds, in that many parent report measures of psychosocial ability include items such as "talks with other children" that are confounded with language ability. Further, psychosocial differences could be consequences of limited language ability (cf. Redmond & Rice, 1998, 2002). Thus, psychosocial development does not carry the straightforward predictor status carried by other variables, such as gender and maternal education.

The outcome of LLE is of considerable interest. Late onset of language is a hallmark characteristic of children with language impairments. In the case of children with SLI, who do not demonstrate other developmental limitations, late talking can be the first diagnostic symptom. Available studies report that 17% (Rescorla, 2002) to 26% (Paul, 1996) of the late talkers have persistent SLI at 4–6 years, although the actual estimates are complicated by the criteria used for diagnosis. These outcomes should be considered preliminary, given very small sample sizes. Rescorla (2002) followed 34 children; Paul (1996) followed 31 children; Whitehurst and Fischel (1994) followed 37 children; and Thal et al. (1991) followed 10 children. Not surprisingly, outcomes of late talking include social and academic risk (i.e., reading ability), in tandem with the likelihood of immature language competencies (Paul, 1996; Rescorla, 2002; Whitehurst & Fischel, 1994).

Limitations of current evidence. The samples of children studied have been small in size (with the exception of the twin study of Dale et al., 2003) and drawn from predominately middle- and upper-middle-class families. Furthermore, some of the samples have included an admixture of monolingual and bilingual children or households in which multiple languages are spoken (Fenson et al., 1994; Klee et al., 1998; Rescorla, 1989; Rescorla et al., 1993), providing a possible confounding effect of undetermined significance. Under such sampling constraints, it is not possible to estimate the extent to which the outcomes generalize to the general population of 24-month-old children or to interpret risk for LLE independent of risk associated with English acquired simultaneously with one or more other languages. The examination of potential predictors of late talking has been limited to a few variables and has been further constrained by the small sample sizes and sampling confounds.

Multivariate models of risk have also been constrained by the available empirical evidence. Our review of the literature yielded a single model (Olswang, et al. 1998). The model posits the child's speech, language, and social development prior to age 3 years as predictors of subsequent language growth, combined with the risk factors of positive family history, prolonged periods of otitis media, and the family's SES levels. The proposed risk factors were not subjected to formal analyses.

What is needed is information on a relatively wide range of maternal, family, and child variables as possible predictors of late talking in the same population of late talkers and controls, with formal multivariate analyses for risk assessment. The questions addressed in this study are: (a) What is the prevalence of LLE in an epidemiologically ascertained sample of 24-month-old children? and (b) Which maternal, family, and child variables are predictive of LLE?

Method

Participants

Data for this analysis came from the RASCALS study in Western Australia. RASCALS is an ongoing longitudinal postal study of a sample of children born in Western Australia in 1995 and 1996. The sample design is an epidemiological prospective observational study of infants randomly ascertained from a total population frame of birth notifications for the state of Western Australia and followed annually from birth. These designs are sometimes called *cohort studies*. In cohort studies, the relationship between exposure and the incidence of an outcome is examined by following the entire cohort and measuring the rate of occurrence of new cases in different exposure groups. The prospective follow-up allows the investigator to identify participants with and without the outcomes of interest. In a case-control study, the individuals who develop the outcome condition (the *cases*) are identified by some other mechanism than follow-up, and a group of participants (the *controls*) is used to represent the participants who do not develop the outcome condition. As findings in this report focus on the 24-month follow-up of the RASCALS cohort only, the data analyses has been approached from the standpoint of a case-control study nested in a cohort study (i.e., nested case control study; Clayton & Hills, 1996).

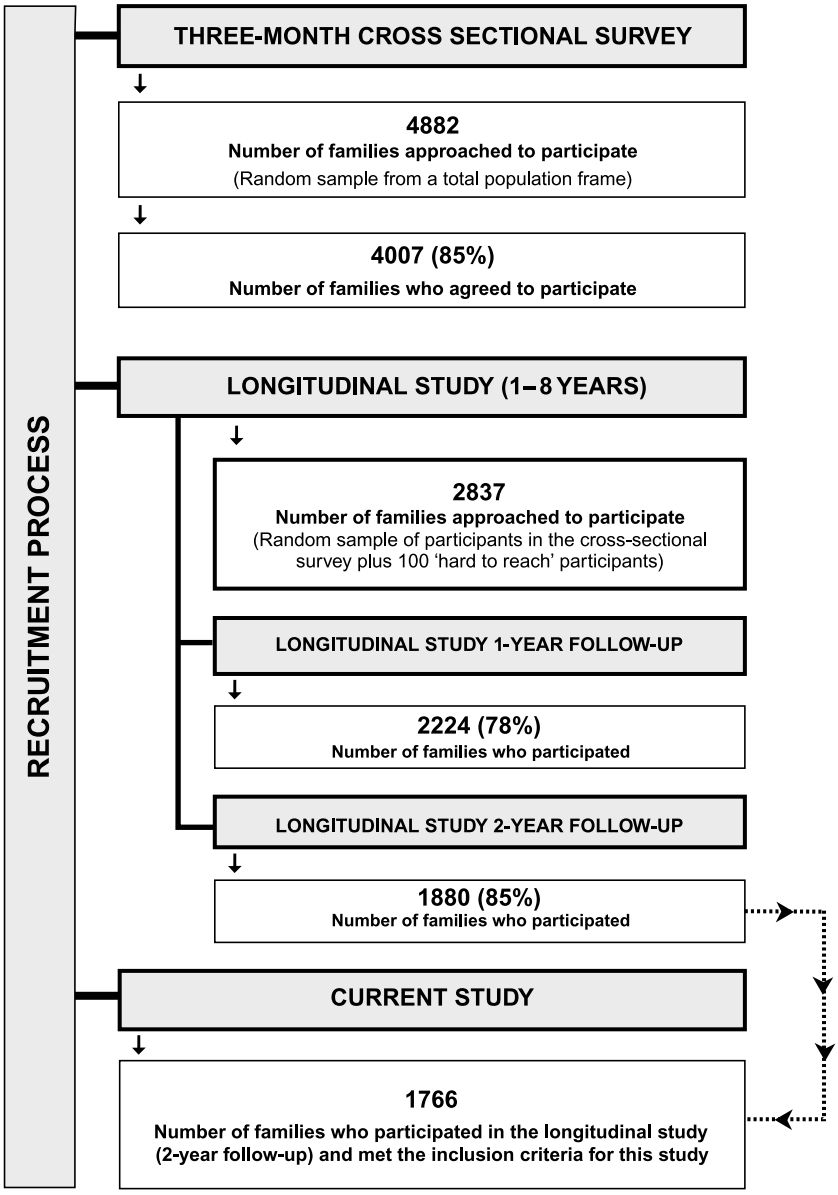
The original sample was randomly drawn from a total population sample frame comprising statutory notifications of birth (Stanley, Read, Kurinczuk, Croft, & Bower, 1997). The RASCALS study was designed to survey health-related behaviors (Kurinczuk, Parsons, Dawes, & Burton, 1999) as a way of identifying and evaluating health promotion opportunities from birth to 8 years and as a way of investigating early causal pathways of mental health problems in childhood. Four thousand and seven mothers responded to the initial questionnaire sent at 3 months postpartum. A comparison with data available about all births in Western Australia (Stanley et al., 1997) showed that the 4,007 respondents were representative of all women with live births in that period, with the exception of a slight underrepresentation

of women with low birthweight babies (5.3% overall vs. 4.7% in the sample) and mothers aged younger than 20 years (6% overall vs. 3.6% in the sample). Because metropolitan Aboriginal mothers were participating in a similar but more culturally appropriate study, they were not recruited into the RASCALS study.

Following the 3-month postpartum response, the study was converted to a longitudinal study and, for resource reasons, just less than a 70% random sample of mothers of singletons was drawn from the initial 4,007 respondents. However, to ensure that "hard-to-reach" groups remained in the RASCALS study in sufficiently informative proportions, we also included all mothers who were either unmarried or not cohabitating, those women who had an annual household income of $A16,000 or less, and those women whose partner was absent from the household; as a result, 100 mothers were added to the sample. Thus, a total sample of 2,837 mothers and their singleton infants were selected for longitudinal follow-up, of whom 2,224 (78%) agreed, when their infant was 1 year old, to participate. Of the 2,224 women who agreed to participate, 1,880 (85%) returned a completed questionnaire when their child was 2 years old. These children are the focus of this report (see Figure 1).

Of potential concern is the representativeness of the study sample to the population from which it was drawn.

Figure 1. The study sample.



Our assessment of this suggests that the 2-year-old follow-up sample is a reasonably representative sample of 2-year-old non-Aboriginal Western Australian singleton children. The potential effects of sample attrition were examined by comparing a range of early life characteristics present at 3 months of age for the respondents at 2 years of age compared with the respondents at 3 months. Small but significant variations were noted. Participants at 2 years of age were more likely to be from families earning more than $A25,000 per year (74.5% vs. 70.7%), $\chi^2(2) = 10.4$, $p < .01$, and from married households (79.3% vs. 75.6%), $\chi^2(4, p < .03) = 11.7$, with a higher level of maternal education (27.8% vs. 23.3%), $\chi^2(4) = 23.6$, $p < .001$. Aside from this, no significant differences were observed for mother's place of birth, number of children or adults in the household, father absence, or receipt of government benefits.

Exclusions

As the principal focus of this study is on the phenomenology of late talking in children where English is their only language, prior to undertaking analysis, the 1,880 participants were assessed for eligibility and possible exclusion. Thirty-three participants were excluded because they did not speak English, another 66 children who spoke both English and another language were excluded, and 7 children with known medical conditions or syndromes were excluded. An additional 8 participants lacked sufficient data from which to determine their possible eligibility and, hence, they were excluded. In total, 114 participants were excluded, leaving a final participant pool of 1,766 (see Figure 1).

Data Collection

Data from the RASCALS participants were collected by postal questionnaire. On or within 1 month of the study child's first and subsequent birthdays up to age 8 years, the parents were mailed a questionnaire for self-completion with a reply paid envelope. Mothers (who represented the majority of the respondents) provided all details on themselves, their partners, and the study child. Nonrespondents received a reminder letter and were subsequently contacted directly by the study research assistant to confirm receipt of the questionnaire and ascertain reasons for nonresponse. Only data collected at ages 1 and 2 were used in the analysis reported here.

Measures

In line with our ecological model, the variable space is drawn from three broad domains of potential developmental influence: Characteristics related to the mother, to the family, and to the child. The measures have been used successfully in other population-based child health surveys in Western Australia (Garton, Zubrick, & Silburn, 1995; Silburn et al., 1996; Zubrick et al., 1995; Zubrick et al., 1997). The high completion rate (85%) and low level of missing data (2.2%) in this study provide further support for the suitability of the measures for diverse population samples.

Maternal characteristics. Each respondent was asked her age in years; current educational level; place of birth; whether she was currently employed and, if so, the numbers of hours per week that she was in paid employment; whether she smoked during her pregnancy with the index child; and whether she was a current smoker.

Each mother completed the 42-item Depression Anxiety Stress Scale (DASS; Lovibond & Lovibond, 1995a, 1995b). The DASS assesses symptoms of depression, anxiety, and stress in adults. Each item (i.e., "I feel sad and depressed") is rated on a four-point Likert scale. Items are summed to generate a score for each of the three domains. The scale has high reliability for the Depression ($\alpha = .91$), Anxiety ($\alpha = .81$), and Stress ($\alpha = .89$) scales and has good discriminant and concurrent validity. Higher scores are associated with higher levels of distress.

Mothers were also asked to complete the Parenting Scale (PS; Arnold, O'Leary, Wolff, & Acher, 1993). This 30-item questionnaire measures three dysfunctional discipline styles: laxness (permissive discipline); overreactivity (authoritarian discipline; displays of anger, meanness, and irritability); and verbosity (overly long reprimands or reliance on talking). The PS Total score (range = 1–7) increases with increasingly dysfunctional parenting, has good internal consistency ($\alpha = .84$), good test–retest reliability ($r = .84$), and reliably discriminates between parents of clinical and nonclinical children where scores in excess of 3.1 denote "clinical" levels of dysfunctional parenting (Arnold et al., 1993). It has been used extensively in research and been shown to be responsive to parenting interventions.

Family characteristics. Respondents were also asked to provide details on the number of individuals who usually reside with the child and their biological and nonbiological relationship to one another. This allowed a basic description of family structure (i.e., original, step/blended, sole parent, or other). Although information on birth order was not gathered, the number of children in the household under the age of 18 was recorded. Total family income before taxes was gathered, as well as receipt of government benefits. The residential address was linked to census track data permitting each child's record to be populated with three small-area indices—the Socioeconomic Indicators for Areas (SEIFA; Australian Bureau of Statistics, 1998).

The SEIFA indicators used in this report measure disadvantage, resources, and occupation/education within the census collection district of the index household. These indexes were developed by the Australian Bureau of Statistics (1998). Each index summarizes a different aspect of the socioeconomic conditions of the Australian population using a combination of variables—in this case, from the 1996 Population and Housing Census. The Index of Relative Socioeconomic Disadvantage (Australian Bureau of Statistics, 1998) is derived from variables that reflect or measure relative disadvantage. Variables used to calculate the index of relative socioeconomic disadvantage include low income, low educational attainment, high unemployment, and low-skill occupations. Lower scores are associated with greater disadvantage. The Index of Economic Resources (Australian Bureau of Statistics, 1998) summarizes the income and expenditure of families, such as income and rent living in the census district. Additionally, variables that reflect wealth, such as dwelling size, are also included. Lower scores reflect lower area economic resources. The Index of Education and Occupation (Australian Bureau of Statistics, 1998) is designed to reflect the educational and occupational structure of communities. The education variables in this index show either the level of qualification achieved or whether further education is being undertaken. The occupation variables classify the workforce into the major groups of the Australian Standard Classification of Occupations (ASCO) and the unemployed. This index does not include any income variables. Lower scores are associated with lower levels of education and lower levels of job skill. Each index is standardized to have a mean of 1000 and a standard deviation of 100.

Mothers completed the 12-item General Factor scale from the McMaster Family Assessment Device (FAD; Miller, Epstein, Bishop, & Keitner, 1985). The 12-item General Factor scale measures overall family functioning across six areas of family functioning: problem-solving, communication, affective involvement, affective responsiveness, roles, and behavior control. It has adequate test–retest reliability, has low correlations with social desirability, and shows evidence of both concurrent and discriminative validity (Miller, Epstein, Bishop, & Keitner, 1985). Cronbach's alphas on the general factor scale are on the order of .86 (Epstein et al., 1993; Miller et al., 1985; Zubrick et al., 1997). Higher scores are associated with higher levels of dysfunction.

Finally, mothers were asked if there was a family history of late talking (i.e. "Has anyone in your family been slow in learning to talk?"). Although this is a minimal estimate of family risk, there is evidence to support validity. Rice, Haney, and Wexler (1998) investigated 19 families who were ascertained because of a child with SLI versus 41 control families. This question yielded 39% of the SLI families with a positive history versus 10% of the control families, a statistically significant difference.

Characteristics of the child: Birth status. The population database from which the RASCALS sample was drawn contains each child's gender, birth date, race (Caucasian, Aboriginal, and Other), birthweight in grams, low birthweight status (<2,500 grams), time to spontaneous respiration in minutes, and gestational age in weeks (Stanley et al., 1997). These data are collected by statute on all live births, stillbirths, and neonatal deaths in the state of Western Australia. An additional measure, the Proportion of Optimal Birth Weight (POBW), is also derived from these data.

POBW is a measure of the appropriateness of intrauterine growth and is routinely calculated from the birth records of all children born in Western Australia. Because birthweight is the end result of growth over the period of gestation, it is therefore determined both by the length of gestation and the rate of intrauterine growth. The rate of intrauterine growth is determined by many factors that are both pathological (maternal, fetal, or environmental) and nonpathological (genetic endowment [particularly fetal gender] and maternal environment). Thus, it is appropriate that fetal growth rate should vary between individuals, as the nonpathological factors determining growth rate varies between individuals: Female newborns appropriately weigh less than male newborns of the same gestation, babies of small women weigh less than babies of tall women, and a woman's first birth tends to weigh less than her subsequent births. We define the *optimal fetal growth rate* for any particular fetus as the median birthweight achieved by fetuses with the same values for the nonpathological determinants of fetal growth and duration of gestation[1], in the absence of any pathological determinants of fetal growth. This median is expressed as the *optimal birthweight* once the values of the nonpathological determinants of growth have been specified.

The nonpathological determinants considered in our statistical models were fetal gender, maternal age, height, and parity. Exclusion of pathological factors was achieved by limiting the sample from which optimal birthweights were identified to singleton, live births without congenital abnormalities born to nonsmoking mothers following pregnancies without any complications known to affect intrauterine growth (Blair, 1996). The median value of POBW is 100; values less than this signify infants that are undergrown, whereas values greater than this represent growth in excess of optimal growth.

[1]Duration of gestation may be curtailed or prolonged, and this is usually the result of pathological factors; hence, abnormal duration of gestation may be considered to reflect pathological factors. However, because delivery must follow the period of intrauterine growth, duration of gestation is not a determinant of growth and hence cannot be a pathological determinant of growth, although it is the primary determinant of birthweight.

Infants whose POBW is less than 85% are classified as being growth restricted at birth.

POBW is an important index of the child's developmental status and is associated with increased risks for developmental and academic failure (Zubrick et al., 2000). The advantage of this measure of appropriateness of growth over birthweight is that it is both individualized and takes into account the duration of gestation. The advantage over the commonly used percentile measures (sometimes termed *small for gestational age*) is that it is more accurate and generalizable at the extremes and, being a parametric ratio quantity, is more amenable to statistical manipulation. Where POBW can be calculated, it is generally preferable to more traditional measures such as gestational age and birthweight (Blair, Liu, de Klerk, & Lawrence, 2005).

Characteristics of the child: Developmental status. Mothers were asked to complete the Infant/Child Monitoring Questionnaires (now called the Ages and Stages Questionnaire [ASQ]; Bricker & Squires, 1999; Squires & Bricker, 1993; Squires, Bricker, & Potter, 1997; Squires et al., 1999) for children aged 24 months. The ASQ requires the mother to observe her child and answer six questions about her child's development in each of five principal areas (which are also the names of the subscales)—Communication, Gross Motor Skills, Fine Motor Skills, Adaptive Problem Solving, and Personal–Social Skills—for a total of 30 items. A final set of questions inquire about the child's overall development and are not used here. The authors of the ASQ have undertaken revision of the instrument and, as a result, there is some slight variation between the original items used in the RASCALS cohort, which are those in the original Infant/Child Monitoring Questionnaires, and those now currently used in the ASQ (see Squires, Potter, & Bricker, 1999).

The manual reports that at 24 months of age, the internal reliability for each of the ASQ scales is 0.75 for Communication, 0.80 for Gross Motor Skills, 0.58 for Fine Motor Skills, 0.57 for Adaptive Problem Solving, and 0.58 for Personal–Social Skills. Two week test–retest reliability, measured as a percentage of agreement between parent-completed questionnaires, was 94%, and interobserver reliability is similarly high. Extensive analyses of the validity of the ASQ, including receiver operator characteristics (ROC) analysis and assessments of concurrent validity using the Stanford–Binet Intelligence Scale (Thorndike, Hagen, & Sattler, 1985), the Bayley Scales of Infant Development (Bayley, 1969), and the Revised Gesell and Armatruda Developmental and Neurological Examination (Knobloch, Stevens, & Malone, 1980), show the ASQ to be valid for the identification of children at risk for developmental difficulties and in need of additional examination (Squires et al., 1999). Using the RASCALS data, composite scores for each of the five areas were calculated using the method outlined in the administration manual, and cutoffs were applied to differentiate normal and abnormal performances (Squires & Bricker, 1993). Further details about the Communication scale are provided below.

Each mother completed a Child Behavior Checklist (CBCL) on the child (Achenbach & Edelbrock, 1991). The CBCL is a checklist of 99 specific behavior problems (e.g., "nightmares," "too shy or timid," "wakes up often at night," "aches or pains (without medical cause)," "gets in many fights," "destroys his/her own things"). Respondents are asked to identify if the item is *not true* (0), *somewhat true* (1), or *very true* (2). The number of problems are converted to normalized *T* scores ($M = 50$, $SD = 10$). The higher the *T* score, the higher the problem behaviors. *T* scores can be computed for Total Problems, Internalizing and Externalizing Problems, and seven syndrome scales (Anxiety/Depression, Withdrawal, Sleep Problems, Somatic Problems, Aggressive Behavior, Destructive Behavior, and Other Behavioral Problems). We report *T* scores and Abnormal *T* scores for Total Problems and Internalizing and Externalizing problems. The CBCL was chosen on the basis of its extensive use in Australia and in numerous other settings and cultures (Hensley, 1988; Sawyer, Arney, et al., 2000; Sawyer et al., 2001; Sawyer, Clark, & Baghurst, 1993; Sawyer, Kosky, et al., 2000; Sawyer, Sarris, Baghurst, Cornish, & Kalucy, 1990; Verhulst et al., 2003; Zubrick et al., 1995). Test–retest reliability is reported to be on the order of 0.87 over 1 week and 0.75 over periods of 12 months (Achenbach & Edelbrock, 1991). It has been demonstrated that the CBCL is acceptable, relatively quick to administer, and supplies adequate coverage of the phenomenology of child behavior problems. Australian studies have found test–retest reliability of the parent CBCL to be 0.87 at 8 weeks and 0.75 at 6 months (Garton et al., 1995; Zubrick et al., 1997). The CBCL has also been used in previous studies of late talkers (Carson et al., 1998; Rescorla & Achenbach, 2002).

The Revised Dimensions of Temperament Survey (DOTS-R; Windle, 1992; Windle & Lerner, 1986) was completed by the mother of each study child. In preschool children, this 54-item scale measures nine characteristics of temperament: (a) activity level–general (high scores signify high levels of energy and motor activity), (b) activity level–sleep (high scores signify high motor activity during sleep; e.g., tossing and turning), (c) approach–withdrawal (low scorers tend to withdraw or move away from persons, objects, and situations), (d) flexibility–rigidity (low scorers respond inflexibly to changes in the environment), (e) mood (low scores are characterized by negative affect), (f) rhythmicity–sleep (low scorers signify an irregular timing of daily sleep–wake cycle), (g) rhythmicity–eating (low scores characterize irregularity of eating habits pertinent to appetite and quantity consumed), (h) rhythmicity–daily habits

(low scores characterize irregularity of diurnal activities such as toileting, peak periods of vigor, and taking a rest), and (i) task orientation (low scorers lack concentration and lack perceptual focus in the presence of extraneous stimuli and do not tend to stay with or continue with an activity for relatively long periods of time). Alpha coefficients of internal consistency for each of the characteristics range from .62 to .89, and 6-week test–retest correlations range from .59 to .75 (Windle, 1992). We conducted a preliminary factor analysis to assess the suitability of Windle's model for use with Australian children. Using Pearson product–moment correlations as input, a principal components analysis using the RASCALS data revealed good factorability (KMO = .88) and communalities ranging from .306 to .839, with nine factors accounting for 52% of the common factor variance. The number of nontrivial factors was determined by using Cattel's scree plot in association with those eigen values greater than or equal to 1.0. Following a varimax rotation, the final factor structure revealed a satisfactory correspondence with only four of the 54 variables loading on factors different from those reported by Windle (Windle, 1992; Windle & Lerner, 1986). In keeping with Windle's recommendations, each of the dimension scores was coded into a dichotomous variable with a score of 1 indicative of dimension scores below the 30th percentile for all but activity-level sleep and general activity level—these being coded 1 if above the 70th percentile (Windle, 1992).

Finally, the mother was asked the child's day care status at the time of the interview and the hours per week that the child attended or received daycare.

Results

All data were screened and distributions inspected for outliers and incorrect values. Missing data were present, to some degree, in all modeled variables. The average amount of missing data among the 1,766 subjects was 2.2% and ranged from zero (mother's place of birth, child gender, and age) to 7.6% (dimensions of temperament, rhythmicity–sleep). To address this problem, we carried out data imputation via a multiple imputation procedure using SAS PROC MI (SAS Institute, 2004). Five complete data sets were generated; each subsequent analysis was performed on each of the data sets, and results were then combined. This imputation approach is preferable to single imputation, which substitutes a single number for each missing value in that the multiple imputation approach accounts for the variability in plausible replacement values (Rubin, 1987). Using a Markov Chain Monte Carlo procedure, all data were imputed at the item level before computing the scale values.

Following imputation, characteristics of the mother, the family, and the child were summarized (see Table 1). Mothers were predominately between the ages of 24 and 34 years at the time of the child's birth. Australia mandates 10 years of compulsory education. Years (i.e., Grades) 11 and 12 are principally used for college entry preparation. The majority of mothers completed 10 years of education, and the distribution of maternal education was bimodal, with about one quarter of mothers having fewer than 12 years of education, 19.3% having completed 12 years of schooling, 13.1% having completed a trade certificate, 13.7% having done some study toward a postschool qualification, and another 29.2% having completed a postschool technical qualification or university degree. Three quarters of the mothers were born in Australia, and 40% of them were in paid employment, working an average of 22 hours per week. Mean maternal DASS scores for depression, anxiety, and stress are comparable to those of the normative sample (Lovibond & Lovibond, 1995b). The mean PS score reported by the RASCALS mothers was also commensurate with the means reported originally by Arnold et al. (1993) for their clinical and nonclinical groups and is comparable to population means reported by Zubrick et al. (2005).

Families were predominately two-parent original families, with 13% of the remaining families being either step/blended or sole-parent families. The average number of children per family was two. Assessment of family income revealed a small proportion of families (5.5%) earning $A16,000 or less per annum. Area SEIFA indicators for disadvantage, resources, and occupation/education were well within population averages for these measures. About 9% of families were classified as having abnormal family function using the FAD. This compares well to the population proportion of Western Australian families reporting abnormal family function (10%; Silburn et al., 1996). A family history of late talking was reported in 13.5% of the families.

Children in the study were an average age of 2.1 (SD = 0.13) years and were nearly all Caucasian (96.6%). With respect to neonatal characteristics, fewer low birthweight infants were in the study sample (3.7%) relative to the Western Australian population proportion (6.4%), but otherwise, the neonatal characteristics of the study sample were unremarkable, with mean birthweight, mean gestational age, and time to spontaneous respiration being comparable to Western Australian population averages (Gee, 1996).

With respect to normative development, study sample mean CBCL T scores were at the approximate 50th percentile, and about 10% of the study children had a CBCL Total T score in the clinical range. These are the first Australian data to be gathered on children as young as 2 years; however, the proportion of children scoring in the clinical range is comparable to Western Australian

Table 1. Maternal, family, and child variables for total sample (N = 1,766[a]).

Variable	Total sample	
	M (SD)	%
Maternal characteristics		
Age at child's birth (years)	29.5 (4.9)	
≤19 years		2.5
20–34 years		82.1
35+ years		15.4
Education		
<12 years		24.6
12 years		19.3
Trade certificate		13.1
Postschool study		13.7
Completed postschool qualification		29.2
Mother's place of birth		
Australia		74.7
United Kingdom		14.8
New Zealand		3.9
Asia and India		2.4
Europe		1.8
North America		1.5
Africa		1.0
Employment		
Currently in paid employment		40.1
Hours in paid employment per week	22.0 (12.7)	
Cigarette use		
Smoked before or during pregnancy		28.2
Current smoker		20.0
DASS scores[b]		
Depression	3.7 (5.3)	
Anxiety	1.9 (3.3)	
Stress	7.49 (6.8)	
Clinical ranges		
Depression		
Normal		90.1
Mild		4.1
Moderate to severe		5.7
Anxiety		
Normal		94.3
Mild		1.9
Moderate to severe		3.8
Stress		
Normal		86.7
Mild		7.1
Moderate to severe		6.1
Parenting score[c]	2.8 (0.57)	
Parenting score in clinical range		28.8
Family characteristics		
Family structure		
Original		84.6
Step/blended		5.1

(Continued on the following page)

Table 1 *Continued.*

Variable	Total sample	
	M (SD)	%
Family characteristics		
Family structure		
Sole parent		7.9
Other		2.4
Family size		
No. children	2.06 (0.95)	
1		29.8
2 or more		70.2
Income		
$1–$154 wk / $1–$8,000 yr		0.7
$155–$308 wk / $8,001–$16,000 yr		5.5
$309–$481 wk / $16,001–$25,000 yr		11.5
$482–$769 wk / $25,001–$40,000 yr		25.9
$770–$961 wk / $40,001–$50,000 yr		17.0
$962 or more wk / $50,001 or more yr		35.4
Not stated		3.8
Area SES indicators		
Disadvantage	1007.9 (59.6)	
Resources	1028.6 (63.3)	
Occupation/education	987.3 (76.2)	
Disadvantage (–1.0 *SD*)		3.4
Resources (–1.0 *SD*)		1.7
Occupation/education (–1.0 *SD*)		7.0
Family function		
Abnormal family function		9.4
Child characteristics		
Gender (male)		50.7
Age (years)	2.1 (0.13)	
Race[e]		
Caucasian		96.6
Aboriginal		0.8
Other		2.4
Neonatal period		
Gestational age < 37 weeks		6.2
<85% POBW		9.1
Low birth weight (<2500 gms)		3.7
Mean percent expected birth weight	100.4(12.3)	
Mean birth weight - grams	339.7 (515.7)	
Time to spontaneous respiration (mins)	1.26 (0.9)	
Gestational age (weeks)	38.9 (1.7)	
Family history of late talking		
Yes		13.5
Ages and Stages Questionnaire (% abnormal)		
Communication score		2.3
Gross motor score		2.0
Fine motor score		3.7
Adaptive score		8.3
Personal–social score		1.6

(Continued on the following page)

Table 1 *Continued.*

Variable	Total sample	
	M (SD)	%
Child characteristics		
Child Behavior Checklist		
CBCL Total *T* score	48.1 (8.4)	
CBCL Internalizing *T*	46.3 (8.4)	
CBCL Externalizing *T*	49.5 (8.3)	
Total *T* score Abnormal		10.4
Total Internalizing *T* score Abnormal		7.2
Total Externalizing *T* score Abnormal		16.3
Child characteristics		
Dimensions of temperament		
Higher general activity level		37.1
Higher sleep activity level		38.9
Withdrawal orientation		23.8
Inflexible behavioral style		26.6
Negative mood quality		24.7
Irregularity in sleeping pattern		26.3
Irregularity in eating behavior		23.1
Irregular daily habits		25.5
Low persistence and high distractibility		26.3
Daycare status at interview		
In day care		37.4
Hours per week in day care	15.5 (12.9)	

[a]Missing data were present in 44 of the 47 variables in this table.The average amount of missing data was 2.2% and ranged from 0% to 7.6%. Data imputation methods have been used (see Method section). [b]Depression Anxiety Stress Scales (Lovibond & Lovibond, 1995a, 1995b). [c]Parenting scale (Arnold et al., 1993). [d]*$p < .05$. **$p < .01$. ***$p < .001$. [e]Racial status recorded on Midwive's Notification Form.

population studies of 4- to 11-year-old children using the appropriate-for-age CBCL parent-reported measure (Zubrick et al., 1995). The ASQ developmental measures ranged from 1.6% (Personal–Social score) to 8.3% (Adaptive score) in the abnormal range, with 2.3% of the sample having ASQ Communication scores in the abnormal range. A little over one third of the children were receiving daycare, with a mean number of 16 hours per week.

Determination of LLE and Prevalence

The scale of the study required an assessment of LLE with minimal effort loading on the part of the parent respondents. The instrument used is the ASQ Communication scale, which comprises a short list of language milestones drawn from the normative literature by Bricker and Squires (1999). The Communication scale is part of an instrument developed as a parent-report measure to screen for developmental impairments. Recently, Luinge, Post, Wit, and Goorhuis-Brouwer (2006) followed the same milestone method to develop a brief language screening instrument intended for public health assessments.

The ASQ Communication scale uses six items to assess aspects of the child's developing skills in speech production and comprehension. Mothers were asked to report whether their child could (a) point to pictures on request, (b) use two- or three-word phrases, (c) carry out simple directions on request, (d) name simple objects, (e) point to body parts on request, and (f) use personal pronouns such as "me", "I," and "you." The response categories for each item were (a) "Not Yet," (b) "Sometimes," and (c) "Yes."

In our sample, the Communication Scale had a Cronbach's alpha of .71, essentially replicating the estimate provided in the test manual. Because the manual does not report validity estimates for the ASQ Communication subscale (only for the full instrument), we carried out analyses of criterion and concurrent validity for our ASQ outcome measure based on *item response theory (ASQ IRT*; described further below). Criterion validity is hampered by the lack of an external "gold

standard" measure of LLE (McCardle, Cooper, & Freund, 2005; Tager-Flusberg & Cooper, 1999). We were, however, in a position to assess some aspects of concurrent validity against another measure of speech and language collected via parent report at the time of the survey. For approximately half of the cohort (N = 902), the LDS had been sought from the parent at the time the questionnaire was completed. Of the children with LDS data, 888 also had ASQ data. This permitted estimating concurrent validity for our measure against the LDS. Both the ASQ IRT score and the LDS score are continuous variables and were moderately correlated (.675; $p < .001$). Additionally, for those children for whom we had a parent-completed LDS, we were able to calculate mean LDS scores for children differentiated by LLE status on the ASQ. Children defined with LLE on the ASQ measure had significantly lower mean LDS scores than those children classified in the normal range on the ASQ measure (M_{LLE} = 62.5, SD = 52.5 vs. M_{Normal} = 196.2, SD = 70.7; df = 198.1, t = 24.7, $p < .001$).

We also assessed the correspondence between the LDS item, "Does your child combine 2 or more words into phrases…?" and the ASQ item, "Does your child say 2 or 3 words together…?". Complete data were available on both of these items for 896 of the children. Frequency distributions were obtained on both the LDS and ASQ items. Ninety percent of children were reported, on the LDS, to be combining two or more words into phrases, and 89% of children were reported, on the ASQ, to be saying two or three words together. Crosstabulation of these items indicated complete correspondence of these items for 860 of these cases, $\chi^2(1)$ = 547.9, $p < .001$; κ = .78. As initial reports of the validity of the ASQ Communication scale, these findings suggest an acceptable level of concurrent validity with another measure frequently used to assess early language emergence.

The Graded Response Model

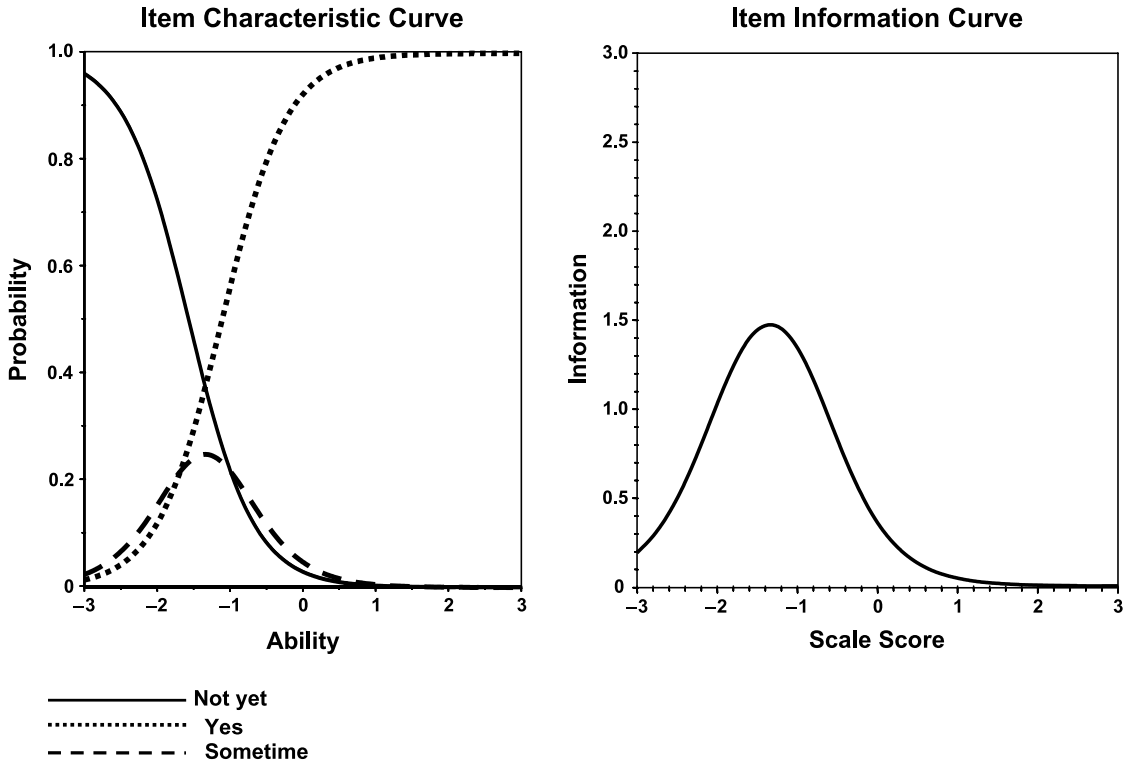
To assess the suitability of the ASQ Communication scale to identify children with LLE, we undertook an item response analysis using a type of polytomous item response theory (IRT) model known as the *graded response model (GRM*; Samejima, 1969). The GRM models each of the three response categories simultaneously, creating a scaled value representing a person's overall ability on the test. In general, Likert-type scales with fewer than five response choices and a small number of items are difficult to summarize with a single "scale" score that has a quantifiable standard error of measurement. The GRM is well suited to the ASQ analyses because it generates an ordering of persons on the ability scale where the responses for the scale are essentially ordered categorical responses. The GRM assumes (a) that the relationship between ability level and the probability of endorsing a particular item response category (or a higher category) is monotonic, (b) that the items are unidimensional and have only one common factor, and (c) that ability is distributed normally with a mean of zero and a standard deviation of 1, even if the items do not measure the entire range of the distribution. This third assumption is not a necessary assumption of the model but is merely an identification condition to set the scale of ability and may be modified if desired. The major advantages of the IRT approach over other methods of scaling include (a) use of all items rather than a reliance on a single item; (b) differential adjustment for item difficulty; (c) provisions for appropriate handling of missing data by determining estimates of ability that are based on all of the items answered and that do not impute the individual's mean score for missing items; and (d) use of a continuous estimate of (in this case) communication/verbal ability, which is on a scale that is not sample dependent.

We commenced our assessment of the ASQ Communication scale by testing the tenability of the dimensionality assumption. To do this, we used a principal components analysis. In addition to this traditional analysis, we also used the DETECT algorithm (Stout, 1987) which is confirmatory in nature. The results of both of these analyses indicated that the six items represent only one dimension.

Item characteristic curves (ICCs; also known as *item response functions*) for each of the six items were then evaluated. For economy of space, an example of one of the items is shown in Figure 2. The lines show the probability of endorsing a certain response at a given level of ability. These figures show that with increasing ability, the probability of a "not yet" response decreases, whereas the probability of a "yes" response increases. At the upper end of the ability scale, there is very little difference in the probability of a "yes" response. Thus, for Item 2, measuring the use of two- or three-word phrases (see Figure 2), a child with an ability of 1 SD above the mean would have about the same probability of a "yes" response as an individual with an ability of 3 SDs above the mean. The graph in the right panel, the item information curve, represents how well the item can distinguish or discriminate between different levels of ability. We can see that Item 2 is best at discriminating individuals with ability near –1.5 SDs. This is where the item is most informative and where measurement error is the lowest. Our assessment of each of the ICCs showed that the ASQ Communication scale measured the low end of ability quite well.

Having determined the item parameters from the child's response on each of the six items, these parameters

Figure 2. The item characteristic curve and information curve for Communication Item 2 ("Does your child say two or three words together that are different ideas, such as 'see dog,' 'mommy come home' or 'cat gone'?").



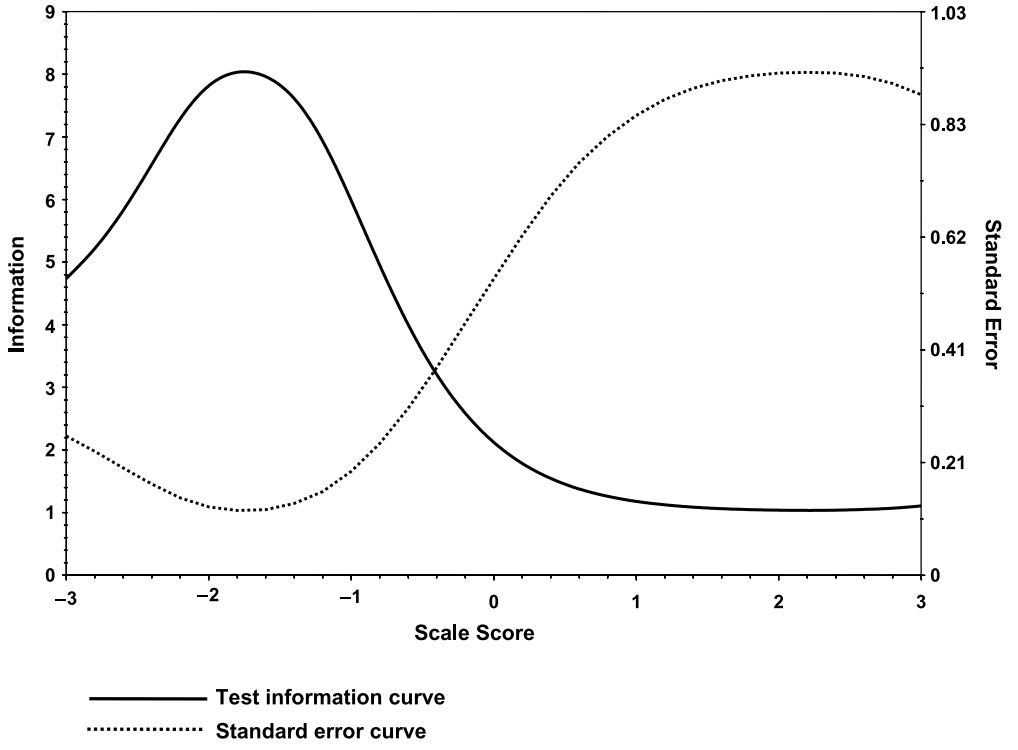
were then used to create an estimate of each child's ability. This estimate gives the child's most "likely" ability level that explains the child's responses. As shown in the test information curve in Figure 3, we can see that the six-item scale provides increasing discrimination and lower measurement error in the range from –1.0 to –1.5 *SD*s below the mean. The IRT/GRM models do not generate an exact cutoff point for creating a dichotomous variable for LLE, but the choice of the cutoff point is guided, in part, by the range of scores within which the scale is more precise in discriminating between different ability levels and also by the researcher's judgment based on previous research and clinical factors.

For reasons of clinical benchmarking and to avoid missing children with LLE, we chose –1.0 *SD* as the cutoff to demark those children with and without LLE (cf. Feldman et al., 2005). Of the 1,766 children, 238 (13.4%) were classified as having LLE (see Table 2). The 13.4% estimate from the IRT composite can be compared to an alternative estimate. Following precedents in the literature, the ability to combine words at 24 months was used as a criterion for grouping children. Of the sample, 10.7% of the children were reported to not combine words, 8.4% were reported as "sometimes," and 80.9% were reported as "yes," yielding an overall estimate of 19.1% of the sample who were not routinely combining words in utterances.

LLE—Bivariate Relationships With Maternal, Family, and Child Characteristics

Comparisons of maternal, family and child characteristics were made for children differentiated by LLE (see Table 2). Alpha levels were not adjusted for family-wise or study-wise error in order to detect any possible differences among the groups. When differences were evident, almost all of them were at conventional levels of adjustment—that is, <.01 or .001. With respect to maternal characteristics, no significant differences for children with and without LLE were observed with regard to maternal age at the birth of the child, levels of maternal education, mother's place of birth, maternal uptake of paid employment, and cigarette use. There were no significant differences among these groups in their mean maternal DASS scores nor in the proportions of mothers reporting varying levels of clinical depression, anxiety, and stress. The only statistically significant difference observed with regard to maternal characteristics was in the Parenting score—the mothers of children with LLE reported higher mean PS scores

Figure 3. Communication composite information curve.



(M = 2.9, SD = 0.6 vs. M = 2.8, SD = 0.6, ps < .01), with a correspondingly higher proportion falling within the clinical range (36.9% vs. 27.6%), $\chi^2(1) = 8.67$, $p < .01$, denoting a higher level of dysfunctional parenting.

Within families, LLE was associated with a family history of late talking (22.2% vs. 12.1%), $\chi^2(1) = 18.2$, $p < .001$, and with larger family size as measured by the number of children in the family. When compared with children who did not have LLE, children with LLE were less likely to be the only child (20.1% vs. 31.4%), $\chi^2(3) = 16.6$, $p < .001$. Otherwise, there were no significant differences in the family characteristics of children with and without LLE in terms of family type (i.e., two-parent, sole-parent), income, area level indicators of SES, and family function.

With respect to characteristics of the child, there were several significant differences between children with and without LLE. Children with LLE were significantly more likely to be male (70.8% vs. 47.6%), $\chi^2(1) = 44.3$, $p < .001$. Although comparisons of their mean ages showed children with LLE to be significantly younger (M = 2.08 years, SD = 0.104 vs. M = 2.11, SD = 0.135, $p < .001$), this equates to a mean difference of 10 days in age between these groups. In practical terms, 99.8% of the children were between the ages of 23 and 24 months of age.

There was no significant difference between LLE groups on neonatal measures of birthweight, low birthweight status, and time to spontaneous respiration. However, children with LLE were significantly more likely to be born weighing less than 85% of their optimal birthweight (14.7% vs. 8.2%), $\chi^2(1) = 10.5$, $p < .01$, and at less than 37 weeks' gestation. Because gestational age—specifically, prematurity—is frequently cited as a confounding factor for LLE, separate investigation of this as a possible threat to the validity of the findings is reported below.

With regard to development, significantly higher proportions of children with LLE were in the abnormal range on the ASQ Gross Motor, Fine Motor, Adaptive, and Personal–Social scores. Results on the ASQ Communication score, which is calculated from the six variables used to define LLE status, revealed all children with LLE to fall in the abnormal range of the Communication score. In terms of behavioral and emotional adjustment, significantly higher proportions of children with LLE were in the abnormal range on the parent-reported CBCL Total Score (15.6% vs. 9.6%), $\chi^2(1) = 7.94$, $p < .001$, with corresponding and statistically significant elevations in CBCL Internalizing problems (11.0% vs. 6.7%), $\chi^2(1) = 5.64$, $p < .001$, and Externalizing problems (23.8% vs. 15.1%), $\chi^2(1) = 11.4$, $p < .01$.

Table 2. Maternal, family, and child variables for control and LLE children (N = 1,766[a]).

Variable	Language emergence: Normal (N = 1,528)		Language emergence: Late (N = 238)			
	M (SD)	%	*M (SD)*	%	χ^2	(*t*)[b]
Maternal characteristic						
Age at child's birth (years)	29.5 (5.0)		29.2 (4.7)			(0.79)
≤19 years		2.5		2.6	.99	
20–34 years		81.8		84.2		
35+ years		15.7		13.2		
Education						
<12 years		23.9		29.6	6.82	
12 years		19.3		18.5		
Trade certificate		13.3		12.4		
Postschool study		13.4		15.9		
Completed postschool qualification		30.1		23.6		
Mother's place of birth						
Australia		74.5		76.5	4.26	
United Kingdom		15.0		13.2		
New Zealand		4.0		3.4		
Asia and India		2.2		3.8		
Europe		1.7		1.3		
North America		1.6		1.3		
Africa		1.1		0.4		
Currently in paid employment						
Yes		40.7		36.7	1.37	
Hours per week in paid employment	22.0 (12.7)		21.7 (12.9)			(0.37)
Cigarette use						
Smoked before/during pregnancy		28.0		30.0	0.41	
Current smoker		19.6		23.0	1.48	
Depression	3.6 (5.3)		4.0 (5.5)			(–1.22)
Anxiety	1.9 (3.3)		2.1 (3.6)			(–0.99)
Stress	7.4 (6.8)		7.6 (6.5)			(–0.34)
Clinical ranges						
Depression					0.47	
Normal		90.3		89.1		
Mild		4.1		4.2		
Moderate to severe		5.6		6.7		
Anxiety					0.51	
Normal		94.5		93.3		
Mild		1.9		2.1		
Moderate to severe		3.7		4.6		
Stress					2.98	
Normal		87.1		84.2		
Mild		6.7		9.8		
Moderate to severe		6.2		6.1		
Parenting score	2.8 (0.6)		2.9 (0.6)			(–3.04)**
In clinical range		27.6		36.9	8.67**	
Family characteristics						
Family structure						
Original		84.9		82.8	0.73	
Step/blended		5.0		5.6		

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Table 2 *Continued.*

Variable	Language emergence: Normal (*N* = 1,528) *M (SD)*	Normal (*N* = 1,528) %	Late (*N* = 238) *M (SD)*	Late (*N* = 238) %	χ^2	(*t*)[b]
Family characteristics						
Family structure						
Sole parent		7.7		9.0		
Other		2.4		2.6		
Family size						
No. children	2.0 (0.9)		2.2 (1.0)			(−2.85)**
1		31.4		20.1	16.6***	
2		68.6		79.9		
Income						
$1–$154 wk / $1–$8,000 yr		0.6		0.9	9.41	
$155–$308 wk / $8,001–$16,000 yr		5.5		5.6		
$309–$481 wk / $1,6001–$25,000 yr		10.4		16.7		
$482–$769 wk / $25,001–$40,000 yr		26.0		26.1		
$770–$961 wk / $40,001–$50,000 yr		17.3		16.7		
$962 or more wk / $50,001 or more yr		36.4		31.2		
Not stated		3.7		3.0		
Area SES indicators						
Disadvantage	1008.6 (59.6)		1004.0 (59.3)			(1.11)
Resources	1028.6 (63.3)		1028.4 (63.3)			(0.05)
Occupation/education	988.3 (76.2)		981.0 (75.8)			(1.36)
Disadvantage (−1.0 *SD*)		3.4		3.3	0.01	
Resources (−1.0 *SD*)		1.7		1.4	0.11	
Occupation/education (−1.0 *SD*)		6.8		8.5	0.91	
Family function[c]						
Abnormal family function		9.1		11.8	1.75	
Family history of late talking						
Yes		12.1		22.2	18.2***	
Child characteristics						
Gender (male)		47.6		70.8	44.3***	
Age (years)	2.11 (0.135)		2.08 (0.104)			(3.64)***
Race						
Caucasian		96.6		96.4	0.34	
Aboriginal		0.8		0.7		
Other		2.3		2.9		
Neonatal period						
<85% POBW		8.2		14.7	10.5**	
Low birthweight (<2,500 g)		3.4		5.9	3.56	
<37 weeks' gestation		5.5		10.7	9.6**	
Mean POBW	100.6 (12.0)		99.5 (14.0)			(1.28)
Mean birthweight (g)	3446.4 (498.0)		3396.9 (615.7)			(1.35)
Time to spontaneous respiration (min)	1.3 (0.9)		1.3	1.0		(−0.83)
Gestational age (weeks)	39.0 (1.6)		38.8 (2.1)			(1.86)
Ages and Stages Questionnaire (% abnormal)						
Communication Score		0.0		16.9	264.3***	
Gross Motor Score		1.4		5.9	21.2***	
Fine Motor Score		2.7		8.1	18.1***	

(Continued on the following page)

Table 2 *Continued.*

Variable	Language emergence: Normal (*N* = 1,528) *M (SD)*	Normal (*N* = 1,528) %	Late (*N* = 238) *M (SD)*	Late (*N* = 238) %	χ^2	(*t*)[b]
Child characteristics						
Ages and Stages Questionnaire (% abnormal)						
Adaptive Score		6.3		21.1	59.3***	
Personal–Social Score		0.6		8.1	73.1***	
Child Behavior Checklist						
Total *T* score	47.8 (8.3)		49.8 (8.8)			(−3.42)***
Internalizing *T* score	46.0 (8.3)		48.0 (8.8)			(−3.46)***
Externalizing *T* score	49.2 (8.2)		51.1 (9.0)			(−3.16)**
Total *T* score Abnormal		9.6		15.6	7.94**	
Total Internalizing *T* score Abnormal		6.7		11.0	5.64*	
Total Externalizing *T* Score Abnormal		15.1		23.8	11.43***	
Dimension of temperament						
Higher general activity level		36.9		38.2	0.15	
Higher sleep activity level		38.0		44.1	3.22	
Withdrawal orientation		23.6		25.0	0.22	
Inflexible behavioral style		26.3		28.4	0.46	
Negative mood quality		23.7		31.3	6.39*	
Irregularity in sleeping pattern		25.6		31.3	3.44	
Irregularity in eating behavior		23.2		22.6	0.04	
Irregular daily habits		24.9		29.2	2.00	
Low persistence and high distractibility		25.7		30.3	2.25	
Daycare status at interview						
In daycare		37.8		34.6	0.90	
Mean hours in daycare	15.2 (17.3)		14.4 (−1.38)			

Note. LLE = late language emergence; SES = socioeconomic status; POBW = Proportion of Optimal Birthweight.

[a]A total of 49 participants had missing data on the outcome variable ("late talker" status). [b]*$p < .05$. **$p < .01$. ***$p < .001$. [c]General Factor of the McMaster Family Assessment Device (Byles, Byrne, Boyle, & Offord, 1988).

The only temperament difference between those children with and without LLE was in negative mood quality. Relative to children without LLE, a significantly greater proportion of children with LLE were reported by their mothers to have negative mood quality (31.3% vs. 23.7%), $\chi^2(1) = 3.44$. $p < .05$. Finally, there was no difference in the proportion of children with and without LLE who were enrolled in daycare nor in the amount of daycare they received as measured by mean number of hours.

LLE—Multivariate Relationships With Maternal, Family, and Child Characteristics

The numerous relationships of maternal, family, and child characteristics with LLE (see Table 2) were further investigated using multivariate logistic regression. Logistic regression allows the prediction of a discrete, binary outcome (in this case, LLE) from a set of predictor variables (Hosmer & Lemeshow, 1989). The predictor variables may be continuous, dichotomous, discrete, or a mix of these types. Estimated effects of the predictor variables are multivariately adjusted for the effects of the other predictors. In this study, the associations between the outcome variable (LLE) and the candidate predictor variables were expressed as odds ratios. An *odds ratio* is the ratio of the probability of an event's occurrence to the probability of the event's nonoccurrence. In this study, the "event" is LLE, and because LLE is an adverse outcome, the predictor variables are "risk" variables. Where predictors are categorical, these odds ratios are calculated with reference to a specific base or "reference" category.

The candidate predictor variables were selected from Table 2. In fitting the logistic model, virtually all variables were used and, following Hosmer and Lemeshow (1989), most were coded to be categorical, rather than continuous. Two exceptions were made. First, the mother's

country of birth was not entered in the model. The distribution of this variable reflects differential bias in the exclusion of cases owing to English language requirements. Second, the child's age in months at the time of the interview was entered as a continuous variable. All other variables were coded as categorical variables (see Table 2).

To account for data imputation procedures (described in the first paragraph of the Results section), we undertook logistic regression using SAS 9.1 (PROC LOGISTIC and PROC MIANALYZE; SAS Institute, 2004). Instead of filling in a single value for each missing value, these procedures combine the results of the analyses of imputations and generate valid statistical inferences by replacing each missing value with a set of plausible values that represent the uncertainty about the correct value to impute (Rubin, 1976, 1987).

All variables were entered into the model in a single step with LLE as the response variable. For each of the predictor variables, parameter estimates (betas), their standard errors, 95% confidence intervals (CIs), degrees of freedom, *t* values, and their probabilities, along with the odds ratios and their 95% CIs, are shown in Table 3.

There were no statistically significant associations between the various maternal characteristics and LLE. No significant associations between LLE and maternal education, age, smoking, psychological state, or parenting style were observed.

In the variables characterizing the family, LLE was significantly associated with the number of children in the family. Relative to singleton children, those children with LLE were significantly more likely to have one or more siblings (odds ratio [OR] = 2.07, 95% CI = 1.39–3.09). Relative to families without a history of late talking, children with LLE were significantly more likely to be born to families in which a parent has a history of late talking (OR = 2.11, CI = 1.39–3.19). All other statistical associations between LLE and the set of family variables were nonsignificant. This included family type, income, local area disadvantage, low economic resources, and low education and occupational status, family function, and daycare use.

Several characteristics of the child were associated with LLE status. Relative to female children, male children were significantly more likely to have LLE (OR = 2.74, 95% CI = 1.96–3.83). LLE children were more likely to be born at 32 weeks' or less gestation (OR = 1.84, 95% CI = 1.04–3.25) and weigh 85% or less of their optimal birthweight (OR = 1.89, 95% CI = 1.18–3.01). All ASQ variables were significantly associated with LLE. Relative to children in each of the respective normal categories, children with LLE were more likely to fall in the abnormal range of the ASQ on measures of Gross Motor score (OR = 3.12, 95% CI = 1.29–7.51), Fine Motor score (OR = 2.39, 95% CI = 1.19–4.77), Adaptive score (OR = 2.64, 95% CI = .66–4.21), and Personal–Social Score (OR = 5.52, 95% CI = 2.05–14.86).

Potential Threats to Validity

These findings are based upon a well-defined and well-described sample of children aged 2 years. Exclusions from this sample included non-English background and medical conditions or syndromes known at the time of the 2-year observation. To what extent might "covert" disability—that is, conditions not known at the time of the 2-year assessment but associated with LLE—impart bias to these findings? Although the focus of these findings is on the phenomenology of LLE at 2 years, the study children were followed until 8 years of age.

Subsequent examination revealed that 19 additional children developed syndromal conditions that potentially were related to LLE. These children were assessed on the ASQ Communication scale at age 2 years, and 37% were in the normal range, whereas 63% were classified as having LLE. Of the 19 children, 10 were subsequently found to have intellectual disabilities, 4 were diagnosed with autism spectrum disorders, and the remaining 5 were diagnosed with developmental syndromal conditions. The multivariate analysis (see Table 3) was repeated without these children. Only one change occurred in the estimates: Prematurity was no longer a significant predictor of LLE status.

Further inspection of the data revealed that an additional 7 children had been born at less than 31 weeks' of gestation. Six of these children had ASQ Communication scale scores. Fifty percent of these children were measured at age 2 years to have LLE. All 7 of these children were subsequently removed from the multivariate analysis, along with the 19 children found later to have syndromal conditions. Aside from the nonsignificance of gestational age, results revealed no substantive changes to those reported in Table 3.

Discussion

In this study of a large number (N = 1,766) of epidemiologically ascertained 24-month-old children, early language acquisition was assessed via a six-item parent report scale that combined comprehension and production benchmarks. Item response analyses found the composite to measure the low end of ability quite well, providing acceptable levels of discrimination and measurement error. With a criterion of –1 *SD* from the mean, 13.4% of the sample was identified as showing LLE. Using the criterion of "no or only occasional word combinations," 19.1% of the sample was identified.

Table 3. Multivariate logistic regression: Prediction of LLE status by maternal, family, and child variables (bolded entries are significant).

Parameter	Estimate	*SE*	95% CI min	95% CI max	*df*	*t*	*p*	Odds ratio	95% CI min	95% CI max
Intercept	3.7298	2.0435	−0.2755	7.7351	26164	1.83	0.068			
Maternal factors										
Educational level										
<12 years	−0.1064	0.2371	−0.5713	0.3586	2147.5	−0.45	0.654	0.899	0.565	1.431
12 years	0.0258	0.2539	−0.4722	0.5238	1876.4	0.1	0.919	1.026	0.624	1.688
Trade certificate	−0.3990	0.2321	−0.8543	0.0563	1358.5	−1.72	0.086	0.671	0.426	1.058
Postschool study	−0.1079	0.2769	−0.6510	0.4353	1274.9	−0.39	0.697	0.898	0.522	1.545
Completed postschool qualification	ref							1.000		
Age at birth of child										
≤19 years	−0.1813	0.5941	−1.3482	0.9855	580.43	−0.31	0.760	0.834	0.260	2.679
20–34 years	−0.4144	0.6277	−1.6463	0.8176	865.16	−0.66	0.509	0.661	0.193	2.265
35+ years	ref							1.000		
Employment status										
No paid employment	0.1608	0.1780	−0.1882	0.5098	5108.8	0.9	0.366	1.174	0.828	1.665
In paid employment	ref							1.000		
Cigarette use										
Nonsmoker during pregnancy	ref							1.000		
Smoked during pregnancy	−0.2403	0.2707	−0.7725	0.2920	372.93	−0.89	0.375	0.786	0.462	1.339
Current nonsmoker	ref							1.000		
Current smoker	−0.3242	0.2970	−0.9084	0.2600	343.89	−1.09	0.276	0.723	0.403	1.297
Depression Anxiety Stress Scale (DASS)										
Depress – Normal	ref							1.000		
Depress – Mild	−0.2274	0.4140	−1.0394	0.5846	1970.3	−0.55	0.583	0.797	0.354	1.794
Depress – Severe	−0.1514	0.4810	−1.0946	0.7918	2569.6	−0.31	0.753	0.859	0.335	2.207
Anxious – Normal	ref							1.000		
Anxious – Mild	0.2318	0.6171	−0.9777	1.4414	9287.8	0.38	0.707	1.261	0.376	4.227
Anxious – Severe	−0.2373	0.5379	−1.2918	0.8171	5106.2	−0.44	0.659	0.789	0.275	2.264
Stress – Normal	ref							1.000		
Stress – Mild	0.2232	0.3000	−0.3650	0.8115	2055.9	0.74	0.457	1.250	0.694	2.251
Stress – Severe	−0.2487	0.4774	−1.1847	0.6874	3775	−0.52	0.603	0.780	0.306	1.988
Parenting Scale										
Nonclinical	ref							1.000		
Clinical	0.3284	0.2010	−0.0758	0.7326	47.432	1.63	0.109	1.389	0.927	2.081

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Table 3 *Continued.*

Parameter	Estimate	*SE*	95% CI min	95% CI max	*df*	*t*	*p*	Odds ratio	95% CI min	95% CI max
Family factors										
Family type										
Original	ref							1.000		
Step/blended	−0.0459	0.3585	−0.7487	0.6569	11048	−0.13	0.898	0.955	0.473	1.929
Sole parent	0.1704	0.3552	−0.5260	0.8667	5459.5	0.48	0.632	1.186	0.591	2.379
Other	−0.3894	0.5275	−1.4239	0.6451	2027.4	−0.74	0.461	0.677	0.241	1.906
Number of children in family										
Number of children – 1	ref							1.000		
Number of children – 2 or more	**0.7315**	**0.2037**	**0.3322**	**1.1309**	**5836.8**	**3.59**	**0.000**	**2.078**	**1.394**	**3.099**
Family income										
<8K	0.1298	1.0090	−1.8481	2.1077	10423	0.13	0.898	1.139	0.158	8.229
$8–$16K	0.5797	0.9896	−1.3600	2.5193	23772	0.59	0.558	1.785	0.257	12.420
$16–$25K	−0.0232	0.9946	−1.9727	1.9262	282892	−0.02	0.981	0.977	0.139	6.864
$25–$40K	0.1267	1.0046	−1.8423	2.0958	94872	0.13	0.900	1.135	0.158	8.132
$40–$50K	0.0980	1.0015	−1.8650	2.0609	72746	0.1	0.922	1.103	0.155	7.853
$50K or more	ref							1.000		
Socioeconomic indicators for area (SEIFA)										
SEIFA disadvantage – No	ref							1.000		
SEIFA disadvantage – Yes	−1.2546	0.6877	−2.6158	0.1066	123.52	−1.82	0.071	0.285	0.073	1.113
SEIFA low econ resource – No	ref							1.000		
SEIFA low econ resource – Yes	0.5416	0.7679	−0.9694	2.0526	303.35	0.71	0.481	1.719	0.379	7.788
SEIFA low educ occ – No	ref							1.000		
SEIFA low educ occ – Yes	0.4475	0.3568	−0.2541	1.1492	377.35	1.25	0.211	1.564	0.776	3.156
Family function										
Normal	ref							1.000		
Poor	0.2721	0.2657	−0.2491	0.7932	2162.1	1.02	0.306	1.313	0.780	2.210
Family history of late talking										
No	ref							1.000		
Family history late talking – Yes	**0.7480**	**0.2103**	**0.3348**	**1.1612**	**511.21**	**3.56**	**0.000**	**2.113**	**1.398**	**3.194**
Currently in daycare										
No	ref							1.000		
Yes	−0.1239	0.1790	−0.4748	0.2270	19077	−0.69	0.489	0.883	0.622	1.255

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Table 3 *Continued.*

Parameter	Estimate	*SE*	95% CI min	95% CI max	*df*	*t*	*p*	Odds ratio	95% CI min	95% CI max
Child factors										
Gender										
Female	ref							1.000		
Male	**1.0087**	**0.1714**	**0.6726**	**1.3449**	**2754.1**	**5.88**	**<.0001**	**2.742**	**1.959**	**3.838**
Proportion of optimal birth weight										
POBW >85%	ref							1.000		
POBW <85%	**0.6370**	**0.2383**	**0.1699**	**1.1040**	**58026**	**2.67**	**0.008**	**1.891**	**1.185**	**3.016**
Ages and Stages Questionnaire results										
Gross Motor – normal	ref							1.000		
Gross Motor – abnormal	**1.1381**	**0.4484**	**0.2590**	**2.0171**	**5400.3**	**2.54**	**0.011**	**3.121**	**1.296**	**7.517**
Fine Motor – normal	ref							1.000		
Fine Motor – abnormal	**0.8717**	**0.3529**	**0.1794**	**1.5641**	**1081.3**	**2.47**	**0.014**	**2.391**	**1.196**	**4.778**
Adaptive score – normal	ref							1.000		
Adaptive score – abnormal	**0.9730**	**0.2372**	**0.5074**	**1.4386**	**769.19**	**4.1**	**<.0001**	**2.646**	**1.661**	**4.215**
Personal–social – normal	ref							1.000		
Personal–social – abnormal	**1.7099**	**0.5036**	**0.7208**	**2.6991**	**575.61**	**3.4**	**0.001**	**5.529**	**2.056**	**14.867**
Child Behavior Checklist										
Total *T* Score – Normal	ref							1.000		
Total *T* Score – Abnormal	0.1111	0.3407	−0.5567	0.7790	34510	0.33	0.744	1.118	0.573	2.179
Internalizing – Normal	ref							1.000		
Internalizing – Abnormal	0.5158	0.3395	−0.1524	1.1839	288.85	1.52	0.130	1.675	0.859	3.267
Externalizing – Normal	ref							1.000		
Externalizing – Abnormal	0.2529	0.2730	−0.2847	0.7904	270.79	0.93	0.355	1.288	0.752	2.204
Dimension of Temperament scale										
High general activity – no	ref							1.000		
High general activity – yes	−0.0704	0.1819	−0.4275	0.2868	583.11	−0.39	0.699	0.932	0.652	1.332
High sleep activity – no	ref							1.000		
High sleep activity – yes	0.2410	0.1735	−0.1004	0.5825	283.57	1.39	0.166	1.273	0.904	1.790
Withdrawal orientation – no	ref							1.000		
Withdrawal orientation – yes	−0.0695	0.2070	−0.4774	0.3384	224.95	−0.34	0.737	0.933	0.620	1.403
Inflexible style – no	ref							1.000		
Inflexible style – yes	−0.0442	0.1960	−0.4298	0.3414	323.49	−0.23	0.822	0.957	0.651	1.407
Negative mood quality – no	ref							1.000		
Negative mood quality – yes	0.2970	0.1879	−0.0718	0.6658	940.91	1.58	0.114	1.346	0.931	1.946
Irregular sleep pattern – no	ref							1.000		

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Table 3 *Continued.*

Parameter	Estimate	*SE*	95% CI min	95% CI max	*df*	*t*	*p*	Odds ratio	95% CI min	95% CI max
Child factors										
Dimension of Temperament scale										
Irregular sleep pattern – yes	0.1396	0.2214	−0.3058	0.5849	47.243	0.63	0.532	1.150	0.737	1.795
Irregular eat pattern – no	ref							1.000		
Irregular eat pattern – yes	−0.2039	0.2114	−0.6193	0.2114	516.33	−0.96	0.335	0.816	0.538	1.235
Irregular daily habits – no	ref							1.000	1.000	1.000
Irregular daily habits – yes	0.1341	0.1859	−0.2303	0.4984	52886	0.72	0.471	1.143	0.794	1.646
Low persist high distract – no	ref							1.000		
Low persist high distract – yes	0.2992	0.1856	−0.0650	0.6633	1006.1	1.61	0.107	1.349	0.937	1.941
Time to spontaneous respiration										
<2 min	ref							1.000		
>2 min	0.0939	0.3028	−0.4996	0.6874	91758	0.31	0.756	1.098	0.607	1.989
Premature birth										
No >36 weeks	ref							1.000		
Yes ≤ 36 weeks	**0.6107**	**0.2898**	**0.0426**	**1.1788**	**4349.3**	**2.11**	**0.035**	**1.842**	**1.044**	**3.250**
Age months	**−0.0094**	**0.0023**	**−0.0140**	**−0.0049**	**6988.8**	**−4.06**	**<.0001**	**0.991**	**0.986**	**0.995**

Note. CI = confidence interval; econ = economic; educ = educational; occ = occupation; qual = quality; persist = persistence; distract = distraction

Bivariate relationships with maternal, family, and child characteristics found the following maternal variables to be nonsignificant: age at child's birth, education, birthplace, paid employment, cigarette use, depression levels, anxiety, and stress. A parenting instrument found that mothers of LLE children were more likely to use dysfunctional parenting practices. Children with LLE were more likely to have a positive family history of late talking and were less likely to be only children. Nonpredictors included family type (e.g., two-parent vs. sole-parent), income, SES, family function, daycare enrolment, or amount of time in daycare. At the child level, children with LLE were more likely to be male and younger (by a mean of 10 days' difference). Neonatal nonpredictors included birthweight, low birthweight status, and time to spontaneous respiration; significant predictors were percentage of optimal birthweight and less than 37 weeks' gestation. Concurrent predictors at 24 months included gross and fine motor development, adaptive scores, personal–social scores, psychosocial development, and temperament (i.e., negative mood quality).

Multivariate analyses yielded the following significant predictors, listed in order of odds ratio, from highest to lowest: Personal–social levels, gross motor skills, gender, adaptive motor skills, fine motor skills, family history, number of children, proportion of optimal birthweight, prematurity, and age.

Prevalence

Our estimate of 13.4% LLE in the general population falls in the same range as previous estimates that have varied between 10% and 20% (Fenson et al., 1994; Horwitz et al., 2003; Klee et al., 1998; Rescorla, 1989; Rescorla & Achenbach, 2002; Rescorla et al., 1993). In this sample, the prevalence estimate of 13.4% using a composite index of Receptive and Expressive Language was more conservative than 19.1% using the Expressive Language criterion, "combining words." Our overall estimate of 19.1% of the sample who were not routinely combining words at 24 months was comparable to the 19% estimate for the CDI sample at 25 months (Bates, Dale, & Thal, 1996) and the ALSPAC sample at 25 months (Roulstone, Loader, Northstone, Beveridge, & the ALSPAC team, 2002). With specific regards to family history, 23% of those children in families reporting a family history of late talking were found to have LLE as measured in this study, versus 12.0% of those children in families who reported no such history.

Predictors

The comprehensive framework provided by the Bronfenbrenner (1979) model established a set of potential predictors unprecedented in the literature for multivariate evaluation of the child's biogenetic endowment as well as the proximal (maternal and family) and distal (societal) resources available to the child. The large number of null findings is a noteworthy outcome. Although the literature suggests positive predictor status for maternal education, maternal depression, family SES, and parental occupation, none of these variables predicted LLE, either in bivariate comparisons or in multivariate analyses. Simply put, in this large and diverse sample of children and families, risk for LLE at 24 months was not associated with particular strata of parental educational levels, socioeconomic resources, parental mental health, parenting practices, or family functioning. Put another way, children with lower levels of proximal and distal resources are as likely as children with higher levels of these resources to be beyond the LLE category at 24 months.

The only environmental risk for LLE that we identified was the presence of siblings. There was a twofold increase in the risk for LLE for children with siblings, relative to only children. Although we did not examine birth order effects directly, firstborn children are temporary "only" children, so our outcome is consistent with studies that report advantages in language development for firstborn children that are attributed to the quantity and quality of maternal speech (Fenson et al., 1994; Hoff-Ginsberg, 1998). According to the resource dilution model, the addition of even one sibling would halve home resources for language acquisition (Downey, 2001). In this study, the risk conferred by siblings was independent of other home resources. It is possible that the number of children in the family may be a more sensitive proxy measure of home resources for language acquisition in the low performance range than measures such as maternal education and SES.

Alternatively, evidence of possible neurobiological and genetic contribution to LLE was more abundant. The first of these risk indicators was present at birth and was related to male gender and suboptimal fetal growth. A disproportionate number of male children had delayed language development, a finding that aligns strongly with previous studies. Male children were at almost three times the risk for LLE compared with female children. In contrast to the strong disadvantage for male children in the low performance range, there is only a modest advantage for female children across the full range of performance (Fenson et al., 1994; Huttenlocher et al., 1991; Wells, 1985). Children who were less than 85% of their optimum birthweight or who were born earlier than 37 weeks' gestation were at almost twice the risk for LLE. As developed by Blair et al. (2005), the proportion of optimal birthweight is a population-based estimate of fetal growth that is a more differentiated measure of fetal growth than absolute birthweight. Our findings suggest that it is more sensitive to LLE than birthweight that is

not associated with risk for language delay (Paul, 1991; Rescorla et al., 1993; Whitehurst et al., 1992) or SLI (Tomblin et al., 1997). At the same time, there was no difference in the physical condition of the children at birth, referenced to the time it took for the children to breathe independently. Prenatal, perinatal, and obstetric risks have not been implicated previously in the etiology of LLE, although empirical data are scarce (Paul, 1991; Rescorla et al., 1993; Whitehurst et al., 1992). The results of this large epidemiological study that had access to medical information collected at the time of delivery showed that some risks for LLE were present at the moment of birth.

Our finding that lower levels of motor, adaptive, and personal–social performance were predictors of LLE extends the findings of previous smaller-scale studies that compared late talkers and controls and reported lower levels of performance on concurrent measures of general development at 24 months (Carson et al., 1998; Klee et al., 1998; Rescorla & Alley, 2001). The toddlers in previous studies—and the toddlers in our sample—did not have developmental conditions that might account for the group differences reported in previous studies or the significant prediction in our study. Our study cannot address, for example, the extent to which lower levels of motor performance or personal–social development are etiological or phenotypic.

A further complication is that although we treated personal–social skills as a predictor in the analyses, the domain of personal–social skills is difficult to interpret. Measurement confounds are an issue. The item that was most discriminating was a linguistic item—that is, "Does your child call himself/herself 'I' or 'me' more often than his/her own name?" Confounding of language emergence and personal–social skills in this age range is a difficult one to avoid in early assessments thus requires caution in interpretation. Further, children with LLE may find it more difficult to establish social interactions because of their language limitations. It will be difficult to sort out predictive status for this variable.

Yet, in general, our results indicate that children with LLE lag behind control children in multiple dimensions of development, and this maturational lag features in either the etiology or the phenotype of LLE. Although temperament was not a risk factor for LLE, negative mood quality is biologically regulated and provides additional support for the role of maturational lag in the etiology of late language onset.

As suggested by Feldman et al. (2005), a positive family history of late talking is predictive of LLE. Children with positive histories had double the risk for LLE compared with children in families with no family history, suggesting a genetic liability for LLE. Family aggregation data have been reported for four late talker cohorts (Ellis Weismer et al., 1994; Paul, 1991; Rescorla & Schwartz, 1990; Whitehurst et al., 1991), and all but Whitehurst and colleagues (1991) reported an elevated rate of affectedness for family members of late talkers.

Negative mood quality, abnormal child behavior, and dysfunctional parenting did not contribute to the risk for late language onset but were more frequent in children with LLE compared with control children. Differences in temperament, behavior, and parenting of children with and without early language delay at 24 months have been reported previously (Carson et al., 1998; Carson, Perry, Diefenderfer, & Klee, 1999; Irwin et al., 2002; Paul, Looney, & Dahm, 1991). Plomin and colleagues (2002) reported modest genetic associations between behavior problems and verbal and nonverbal abilities in 2-year-old twins. This finding provides provisional support for the view that common biogenetic mechanisms influence problematic temperament, abnormal behavior, and language delay in children. An alternative view is that language delay mediates child temperament and behavior. The conclusion we can draw from our data is that problematic child temperament, abnormal child behavior, and dysfunctional parenting are more likely to be part of the psychosocial profile of late talkers than children with normal language development. The caveat is that the results here are inherently ambiguous; the direction of influence is undetermined, and the full interpretation is likely to be quite complex.

Overall, the results of this study are congruent with models of language emergence and impairment that posit a strong role for neurobiological and genetic mechanisms of onset that operate across a wide variation in maternal and family characteristics. This study points toward familial history of LLE, male gender, and early neurobiological growth as concomitant indicators of risk at 24 months.

Directions for Future Research

The import of LLE can be viewed in terms of recent growth model studies of children with SLI (cf. Hadley & Holt, 2006; Rice, Redmond, & Hoffman, 2006; Rice, Wexler, & Hershberger, 1998; Rice, Wexler, Marquis, & Hershberger, 2000; Rice, Tomblin, Hoffman, Richman, & Marquis, 2004). A consistent finding is that the affected group differs from unaffected children in the intercept but not the trajectory of change over time, pointing to delayed onset of language as an important part of the phenotype of language impairment (cf. Rice, in press; Rice, 2007; Rice & Smolik, 2007; Rice, Warren, & Betz, 2005, for more complete discussion). There is an important empirical gap, however, in fleshing out the connection between LLE and later SLI. LLE status at 24 months is a limited predictor of later language impairment (cf.

Dale et al., 2003) because an indeterminate number of children "outgrow" their delayed onset of language. Two kinds of evidence are needed to help sort this out: The first is an accurate estimate of the proportion of children with LLE who at later ages are identified as having SLI; the second is identification of predictors of subsequent language impairment.

It would be premature to use the predictors evaluated here for screening purposes (i.e., identifying 2-year-olds at risk for subsequent SLI) because this use requires measures and data collected at time points beyond the 24-month period of the current panel of data on which this article is based. Longitudinal data are needed to investigate whether predictors of LLE at 24 months also predict language impairment later or whether the early predictors are modulated or supplanted later by other variables that did not predict LLE. That is, it is conceivable that predictive relationships change over time. For example, maternal or family variables may come into play as a child's vocabulary accelerates or as clausal structures emerge. It will be important to evaluate the extent to which predictive relationships are the same across all language dimensions. Further, long-term outcome data can help sort out possible differences in predictive relationships between children in the low performance range versus children in the normative range. Evidence of this sort will provide a foundation for the development of comprehensive etiological models of LLE and SLI.

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