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FAMILIAL AGGREGATION IN SPECIFIC LANGUAGE IMPAIRMENT

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Self-report data from the families of children participating in the San Diego Longitudinal Study of specific developmental language impairment were used to assess familial aggregation in the disorder. Families of impaired children reported higher rates of affected first-degree relatives than did families of matched controls. Significantly higher incidence of maternal and paternal childhood language and/or learning disabilities, as well as sibling disability rates, were reported. The extent to which familial aggregation reflects genetic or environmental influences in specific language disorders remains to be determined.

The development of language is a complex process. Language must be received either auditorily or visually, processed centrally (this usually being subdivided into receptive and expressive components), and then expressed with appropriate motor (articulation) skills. Developmental language disorders can, therefore, result from peripheral sensory (hearing loss) or motor (speech apparatus) dysfunction. In addition, language deficits can occur secondarily to a more global disorder, such as mental retardation, psychiatric problems, or neurological disorders (epilepsy, autism, etc.). Language disorders can also be acquired secondarily to postnatal brain injury to areas of the brain that subserve language (acquired childhood aphasia). However, even if all the above causes for language delay are excluded, there still exists a group of children with developmental language disorders, and they have come to be known as "developmentally dysphasic" or "specifically language impaired" (Benton, 1964).

Currently, exclusionary criteria are generally the main criteria used for both clinical diagnosis (see DSM III, 1980) and subject selection of specifically language-impaired children for research studies. This adherence to diagnosis by exclusion reflects a lack of information pertaining to etiology, as well as symptomatology, of developmental language disorder. Prevalence studies estimate 8%–15% of all preschool children are affected with some form of speech or language disorder (Beitchman, Nair, Ferguson, & Patel, 1986; Silva, 1980).

Case histories of families with several members having language disorders have been reported (Arnold, 1961; Borges-Osorio & Salzano, 1985; McReady, 1926; Samples & Lane, 1985), suggesting a possible genetic etiology for some language disorders. However, few group studies of children with specific developmental language impairment have focused on family history data. Ingram (1959) obtained family histories on 75 language-impaired children (hereafter, "probands"). Results demonstrated that 18 probands (24%) had at least one parent with a history of language difficulty. Thirty (30) of the 131 siblings of probands (23%) carried a diagnosis of language disorder, this being confirmed by direct observation in 23. However, Ingram noted that the frequency of sibling disability reported must be considered as an underestimate, as the sibship contained several young children not yet old enough to display symptomatology. Although these results are supportive of familial aggregation of language disorder, they are difficult to interpret because proband diagnosis of language impairment was made based on clinical judgment without objective test support, and no inclusionary or exclusionary criteria were specified for this diagnosis. Although some language-impaired children were excluded because of hearing difficulties, the sample included children both with general cognitive deficit (IQ < 90) and emotional problems. It is also not possible to determine the significance of the results as no control group was included. Despite these methodological problems, Ingram's report provided the first group evidence of a possible familial contribution to some types of language impairment.

Two additional reports of familial aggregation of specific developmental language impairment appeared quite recently (Bishop & Edmundson, 1986; Robinson, 1987). Bishop published parental report data that demonstrated a significantly increased frequency of affected primary and secondary relatives in language-impaired as compared to control children. Unfortunately, only brief mention was made of these familial data in the context of a larger study. Robinson (1987) reports family history results from three separate data sets. He reports that 28% of language-impaired probands had a first-degree relative (parent, sibling) with a reported history of speech delay,

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whereas 20% report a history of learning problems. Unfortunately, no control group was included to allow the significance of these results to be determined.

Despite the dearth of empirical, controlled, group studies related to familial aggregation in specific developmental language impairments, there is growing interest in a possible genetic basis for specific communication disorders (see Ludlow & Cooper 1983, for review). As part of a large, multidisciplinary, longitudinal study evaluating the outcomes of preschool impairments in language, specific questions relating to a possible familial etiology for specific language impairment were addressed. This study assessed whether children with specific developmental language impairment come from families with positive histories of developmental communication disorders significantly more often than do normally developing, matched control children.

METHOD

Subjects

Two groups of subjects—those with specific developmental language impairment and their age-, race-, socioeconomic status (SES)-, and IQ-matched normal controls—participated in this study. These subjects were participants in the San Diego Longitudinal Study, which included evaluation of speech, language, neuropsychological, academic achievement, intellectual, social, and emotional development over a 5-year period.

Ninety (90) well-defined, specifically language-impaired, 4-year-old subjects and 60 matched controls were selected over a 2 1/2-year period. First, the language-impaired subjects were selected. In an attempt to include a broadly representative sample, school boards, clinics, and private professionals serving language-impaired children in San Diego County were asked to refer all children who might meet specified study criteria. Approximately 200 language-impaired children were referred and tested by a team of professionals, including a speech-language pathologist, clinical psychologist, audiologist, and learning disability specialist. In addition, each child was assessed for autism and neurological disorder by a pediatric neurologist.

Each child had to meet all the following criteria to be included in the study as a specifically language-impaired subject: (a) age 4:0–4:11 at time of induction; (b) a nonverbal performance IQ of 85 or better on the Leiter International Performance Scale (Leiter, 1940); (c) a mean language age (when computed from standardized expressive and receptive scores) at least 1 year below both performance mental age and chronological age; (d) normal hearing acuity (no more than 20-db loss in either ear at frequencies of 250–6000 Hz), no motor handicaps, and no oral, structural, or motor impairments affecting nonspeech movement of the articulators; (e) an English language background only, without significant nonstandard dialectal usage or other languages spoken in the home; (f) not autistic (as defined by DSM III, 1980); and (g) no known neurological disorders (seizure disorder, hemiparesis, etc.).

Previous reports on putative genetic influences on learning disabilities (e.g., the Colorado Family Reading Study; e.g., Foch, DeFries, McClear, & Singer, 1977 and DeFries, Singer, Foch, & Lewitter, 1978) have concentrated on families for which information was available on both biological parents. Because putative genetic influences may come from either maternal or paternal influences, this study included only those families from whom data could be collected directly from both biological parents. Data on both biological parents were obtained for 76 of the original 90 probands, and this comprised the data set for this study. Excluded cases included subjects living in one-parent families where data could not be collected directly from the other biological parent and subjects that were adopted or lived with secondary relatives.

The normal children were selected after the language-impaired group was completed, so that matching for age, race, IQ, and SES could be accomplished. As more variance was expected to occur in the language-impaired group than in the normal group, a larger sample size of impaired (90) than normal subjects (60) was included, based on statistical design.

Control subjects were sought from the same geographical locations, school districts, and medical groups that had referred the language-impaired subjects. Over 100 normally developing 4-year-olds were referred and tested by professionals in order to select the 60 that both met all the criteria listed below and were matched on age, race, SES, and IQ to the language-impaired group.

In order to be included as a matched, normal control subject, each child had to meet each of the following criteria: (a) age of 4:0–4:11 at time of induction; (b) a nonverbal performance IQ of 85 or better (and not greater than the highest IQ demonstrated by a language-impaired subject) on the Leiter International Performance Scale (Leiter, 1940); (c) a mean language age (when computed from standardized expressive and receptive test scores) not more than 6 months below performance mental age and chronological age; (d) speech articulation age not more than 6 months below chronological age; (e) normal hearing acuity (no more than 20-db loss in either ear at frequencies of 250–6000 Hz), no motor handicaps, and no oral, structural, or motor impairments affecting nonspeech movements of the articulators; (f) no emotional or neurological problems; and (g) an English language background only, without significant nonstandard dialectal usage or other languages spoken in the home.

Of the 60 normal children participating in the longitudinal study, family history data on both biological parents were obtained on 54, and these comprised the control data set for this study.

The means and standard deviations for age, IQ, SES, and language scores of the 76 language-impaired and 54 normal children who form the data set for this study are presented in Table 1. The normal group included 27 boys and 27 girls. The language-impaired group included 54

TABLE 1. Demographic and test performance data for normal and language-impaired groups.

	Normal	*Language impaired*	*Significance*
Total number of subjects	54	76	
Number of boys	27	54	
Number of girls	27	22	
	M *SD*	*M* *SD*	
Leither IQ (Leither, 1940)	112 ± 8	109 ± 12	N.S.
Age	4.4 ± 0.3	4.4 ± 0.3	N.S.
Socioeconomic status [based on low, medium, high Hollingshead & Redlich (1958) ranges]	1.8 ± 0.46	2.0 ± 0.46	N.S.
Receptive language measures			
Sequenced Inventory of Communicative Development (years) (Hendrick, Prather, & Tobin, 1979)	3.9 ± 0.2	3.2 ± 0.5	*
Northwestern Syntax Screening Test (years) (Lee, 1968)	4.8 ± 1.1	3.3 ± 0.6	*
Token Test (DeRenzi & Vignolo, 1962)	4.9 ± 1.4	3.0 ± 0.7	*
Expressive Language Tests			
Sequenced Inventory of Communicative Development (years) (Hendrick et al., 1979)	3.9 ± 0.2	2.8 ± 0.4	*
Northwestern Syntax Screening Test (years) (Lee, 1968)	4.9 ± 1.1	3.0 ± 0.1	*
Carrow Elicited Language Inventory (years) (Carrow, 1973)	5.4 ± 1.4	3.1 ± 0.3	*
Arizona Articulation Proficiency Scale (years) (Fundala, 1980)	>5.0	4.1 ± 1.7	*

*Significance level $p < .01$ or greater (paired t test).

boys and 22 girls, which represents the expected higher incidence (2–3:1 ratio; Ludlow & Cooper, 1983) of language impairment in boys. The children in the language-impaired group showed a range of profiles, both in degree and pattern of deficit. The majority of subjects had both severe expressive and receptive language delays. However, some of the children demonstrated receptive language delays that were greater than their expressive deficit, whereas others showed the opposite profile. On every standardized language test used, the language-impaired group was one and a half or more standard errors below the mean for the age-matched normal group. Over 60% of the language-impaired sample also had a speech articulation deficit. However, children with speech articulation deficits alone, without language disorders, were not included in the study.

Family History Data

At the time of induction into the longitudinal study, the biological mother and father of each subject were each requested to fill out a separate questionnaire relating to family history of language, reading, writing, and academic achievement. This questionnaire was comprised of 35 detailed questions adapted from questionnaires used previously by Childs and Finucci (1979) and Kidd (1980) to investigate familial aggregation in other communicative disorders. Because of the lack of good diagnostic criteria when the parents were children and because of the relationship between language disorders and subsequent academic achievement, particularly reading and writing, *parents* were classified as "affected" if any of the following were reported: (a) a history of language problems; (b) a history of below average school achievement, to the eighth grade, in reading, writing, or both; or (c) a history of ever having been kept back a grade in school, through the eighth grade. Considerable help was given to parents filling out these questionnaires, including written descriptions, explanations, and examples of technical terms such as "expressive language," and so forth to aid them in making accurate judgments. Questionnaires were only accepted if filled out directly by each biological parent. Questionnaires were filled out initially by the parent and then reviewed together with a research assistant so that questions could be resolved. Every effort was made to assure that questionnaires were completed and returned and were accurate.

Sibling classification was somewhat more problematic. In the original questionnaire, we inadvertently failed to either (a) clarify whether proband-sibling relationships were on full- or half-sibs or (b) specify sex of siblings. The latter information is of interest, and the former information is imperative to accurately assess familial impairment frequencies. Parents were recontacted by telephone or mail to obtain these additional sibling data. Combining questionnaire and telephone data, information was available on all primary relatives (both biological parents and all full-sibs) on 62 impaired probands and on 50 control probands. Siblings were diagnosed as "affected" if parents reported for them a positive history for difficulties in reading, writing, language, or other learning disabilities. This reduced data set was used for analyses pertaining to sibling and family impairment frequency.

Due to the young age of the subjects in the study, siblings also tended to be young. Because of this, subclassification of siblings by type of impairment was impossible (e.g., many sibs had not yet reached school age when reading and writing deficiencies might first become apparent). Therefore, sibs were classified as either "affected" or "not affected" without further differentiation. It must be kept in mind that, because of the young age of many of the siblings, results may underrepresent the actual number of eventually affected sibs.

Analysis

Control and language-impaired probands were compared using χ^2 analysis on aggregated data (aggregated across all probands within a group) on rate of maternal impairment (number of affected mothers/total number of mothers), rate of paternal impairment (number of affected fathers/total number of fathers), and rate of offspring impairment (number of affected sibs/total number of sibs).

Sibling impairment rate was also examined by number of affected parents. If familial genetic contribution is an important part of language impairment, increased number of affected parents should positively correlate with increased frequency of sibling impairment.

Whole family data were also analyzed. First, using χ^2 analysis, control and impaired probands were compared on the frequency of positive family history, defined as having at least one first-degree relative affected. Second, for each proband, a familial impairment rate (number of affected first-degree relatives/total number of first-degree relatives) was computed. Control and impaired groups were then compared, using a student's t test. For the purpose of this study, a first-degree relative was defined as including proband's mother, father, and all full-sibs (sibs that had common mother and common father).

RESULTS

Parental Data

Tables 2 and 3 summarize the results pertaining to the parental data. Table 2 shows that mothers of impaired children significantly differed from mothers of controls on all but one of the individual items used for classification as "affected." Mothers of impaired children were more likely to report a history of language problems [$\chi^2(1) = 5.6$, $p < .02$] and also more likely to report having a history of being kept back in school [$\chi^2(1) = 4.7$, $p < .03$] than were mothers of controls. Mothers of impaired children were also more likely than were mothers of controls to report poorer school performance to Grade 8 in reading [$\chi^2(2) = 19.0$, $p < .0001$] and writing [$\chi^2(2) = 20.7$, $p < .0001$] but not in math, [$\chi^2(2) = 2.9$, n.s.]. Maternal data showed that 29.2% of all mothers met the criteria for "affected"; 36.8% of mothers of impaired children, and 18.5% of mothers of controls [$\chi^2(1) = 4.2$, $p < .05$] met the criteria.

Table 3 shows that of the criteria used for classification, fathers of impaired children were significantly different from fathers of controls on only one variable: Fathers of impaired children were more likely to report a history of being kept back in school than were fathers of controls, $\chi^2(1) = 5.1$, $p < .03$). Nonetheless, combining all variables, fathers of impaired children met the criteria for "affected" significantly more often than did fathers of controls [43.4% vs. 20.4%; $\chi^2(1) = 6.4$, $p < .02$], and more fathers than mothers met the criteria for "affected."

Sibling Data

Table 4 summarizes the sibling data. These data demonstrate that the siblings of the controls have a significantly lower affected-sibling frequency (11 of 57 siblings; 19.3%) than do the sibs of the impaired children [37 of 99; 37.4%; $\chi^2(1) = 4.7$, $p < .05$].

Table 5 reorganizes sibling data by parental impair-

TABLE 2. History of language/learning problems—mothers.

	Below average (in %)	*Average (in %)*	*Above average (in %)*	*Significance level*
Math				
Controls	3.7	61.1	35.2	
Impaired	12.3	57.5	30.1	
Writing				
Controls	3.7	24.1	72.2	$p < .0001$
Impaired	6.8	61.6	31.5	
Reading				
Controls	1.9	25.9	72.2	$p < .0001$
Impaired	13.7	52.1	34.2	
	Yes (in %)	*No (in %)*		
Kept back in school				
Controls	1.9	98.1		$p < .03$
Impaired	14.9	85.1		
History of language problems				
Controls	13.0	87.0		$p < .02$
Impaired	32.9	67.1		
Met criteria for "affected"				
Controls	18.5	81.5		$p < .05$
Impaired	36.8	63.5		

TABLE 3. History of language/learning problems—fathers.

	Below average (in %)	*Average (in %)*	*Above average (in %)*	*Significance level*
Math				
Controls	5.6	46.3	48.1	
Impaired	4.1	53.4	42.5	
Writing				
Controls	7.4	40.7	51.9	
Impaired	9.5	56.8	33.9	
Reading				
Controls	7.4	42.6	50.0	
Impaired	8.1	55.4	36.5	
	Yes (in %)	*No (in %)*		
Kept back in school				
Controls	7.4	92.6		$p < .03$
Impaired	24.3	75.7		
History of language problems				
Controls	7.5	92.5		
Impaired	17.8	82.2		
Met criteria for "affected"				
Controls	20.4	79.6		$p < .02$
Impaired	43.4	56.6		

TABLE 4. History of language/learning problems—siblings.

	Number probands	*Affected male sibs*	*Affected female sibs*	*Total affected*	χ^2	*Significance level*
Controls	50	4/28 (14.3%)	7/29 (24.1%)	11/57 (19.3%)		
Impaired	62	27/65 (41.5%)	10/34 (29.4%)	37/99 (37.4%)	4.7	$p < .05$

ment. The frequency of sibling impairment does not differ significantly, based upon number of parents affected, for either the control probands [$\chi^2(1) = 0.09$, n.s.] or language-impaired probands [$\chi^2(2) = 3.8$, n.s.] although trends are in the expected direction, with a higher number of affected parents yielding a higher frequency of offspring impairment. However, combining both groups, there was a significant effect of the number of affected parents on the number of affected offspring (not including proband). Whereas only 20 out of the 80 siblings (25%) of probands with nonaffected parents were affected, 18 out of 57 siblings (31.6%) were affected in families with one affected parent and 10/19 siblings (52.6%) were affected in families with both parents affected, $\chi^2(2) = 7.3$, $p < .05$.

Family Data

When compared to controls, impaired children are significantly more likely to have a positive family history, defined as at least one first-degree relative affected [77.0% vs. 46.0%; $\chi^2(1) = 10.0$, $p < .002$], and report a higher average frequency of impairment in first-degree relatives (41.5 ± 3.1 vs. 18.5 ± 2.4), $t = 4.3$, $p < .001$. Even looking just at those families from both groups that report a positive family history, there was a significant increase in reported family impairment frequencies between the impaired and control groups (53.6 ± 2.4 vs. 40.1 ± 2.0), $t = 2.3$, $p < .03$.

Figure 1 shows the distribution of affected family member (mothers, fathers, siblings) frequencies for the language-impaired and control children. Further analysis demonstrated that there is a similar bimodal distribution in the data for both impaired and control groups. Most families have impairment rates (numbers of affected family members/total number of family members) of 0 or >.32. Few families in either group reported positive histories but with low impairment rates (.01–.32). Thus, for families in both groups, most families reported either several positive nonproband cases in the same family or no nonproband cases at all. That is, isolated impaired cases in families appear rare.

DISCUSSION

The results of this study indicate that families of language-impaired children are significantly more likely to report a history of impairment in other family members than are families of controls, supporting a hypothesis of familial aggregation in specific developmental language impairment. Specifically, families of impaired children are more likely than families of controls to report: (a) a positive family history—at least one first-degree relative affected, (b) a higher average frequency of impairment in first-degree relatives, (c) a higher rate of affected siblings, (d) a higher rate of affected fathers, and (e) a higher rate of affected mothers. Further support for the hypothesis of familial aggregation in specific language impairment is shown by the positive correlation between number of affected parents and rate of sibling impairment.

It can be argued that the higher impairment rate found in families of impaired probands could be biased by self-report data. That is, parents with a diagnosed language-impaired child may be more aware of what language/learning difficulties are, may identify with their

TABLE 5. Affected siblings by number of affected parents.

Number of affected parents	*Number probands*	*Affected male sibs*	*Affected female sibs*	*Total affected*	χ^2	*Significance level*
Control probands						
0	33	2/18 (11.1%)	4/18 (22.2%)	6/36 (16.7%)		
1 or 2[a]	17	2/10 (20.0%)	3/11 (27.3%)	5/21 (23.8%)	0.09	N.S.
Language-impaired probands						
0	24	9/30 (30.0%)	5/14 (35.7%)	14/44 (31.8%)		
1	26	11/23 (47.8%)	3/14 (21.4%)	14/37 (37.8%)		
2	12	7/12 (58.3%)	2/06 (33.3%)	9/18 (50.0%)	3.8	N.S.
All probands						
0	57	11/48 (22.9%)	9/32 (28.1%)	20/80 (25.0%)		
1	42	12/32 (37.5%)	6/25 (24.0%)	18/57 (31.6%)		
2	13	8/13 (61.5%)	2/06 (33.3%)	10/19 (52.6%)	7.3	$p < .05$

[a]Due to the small number of control probands with two impaired parents ($n = 1$), probands with one and with two impaired parents were combined.

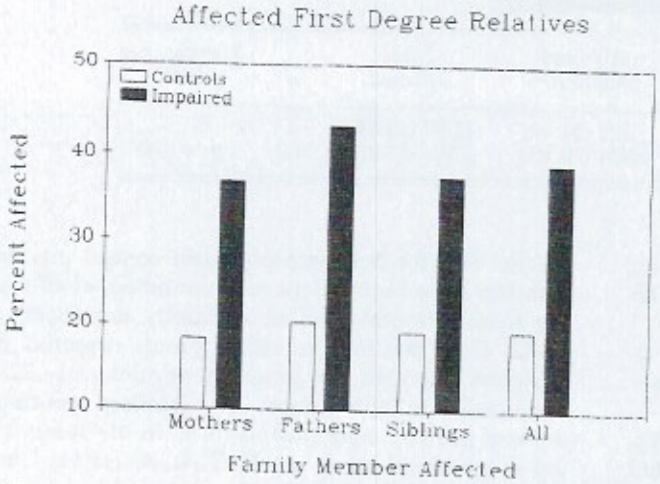

FIGURE 1. Percentage of impaired mothers, fathers, and siblings as well as combined familial impairment frequency is shown for the language-impaired and control probands.

impaired child, and, therefore, may be more likely to report a history of problems for themselves and their other children than would parents of controls. Several factors mitigate against this as the basis for interpretation of these data. Perhaps due to advancement in our understanding of learning disabilities, scholastic and, when indicated, diagnostic testing is now performed regularly on all school-aged children, and the results are made available to parents. This greater awareness of learning disability may be reflected in these data with parents in *both* groups reporting relatively high incidence of language/learning problems in their children and themselves.

Secondly, examination of Figure 1 supports the conclusion that there are primarily two types of probands: those with a strong positive family history and those with no family history (note the low frequency of families with a positive family history but low impairment rates). This pattern was similar in both the language-impaired and control groups.

Thirdly, although fathers of impaired children meet the criteria for "affected" more often, and mothers of impaired children report poorer histories in language, reading, and writing than do their control counterparts, reported history of math skills do *not* differ between groups for either mothers or fathers. This information was obtained at the time of induction into the study when probands were all 4 years old, before parents would have been aware of probands' reading, writing, or math abilities. It seems unlikely that parents would, in identifying with their child's *language* impairment, report poorer histories in their own reading and writing development (which their child had not yet reached an age to acquire) and yet not extend that history to include math problems. On the other hand, the lack of histories of math impairment correlates well with previous family history studies for another similar developmental communicative disorder, dyslexia (e.g., Decker & DeFries, 1980; Owen, Adams, Forrest, Stolz, & Fisher, 1971).

As the family history data for this study were obtained from parental report, it is important to question the reliability of parental report data for language/learning abilities. In a recent study, Bates, Bretherton, and Snyder (1987) have assessed directly the reliability of parental questionnaire data pertaining to their child's early language development. They compared parent report of language development to actual language test performance. The study demonstrates extremely high concordance between parental report of language skills and the actual language performance of young children, demonstrating that parents are very reliable reporters of their children's language/learning development.

In conclusion, this is the first controlled group study to investigate empirically whether there is evidence of familial aggregation for specific developmental language disorders. The study took advantage of a large, carefully selected group of children with specific developmental language disorders and well matched controls, who were participating in a major longitudinal study evaluating the outcomes of preschool language impairments. Although the results demonstrate highly significant differences in the incidence of reports of positive family histories for first-degree relatives between the families of language-impaired and control children, they must be interpreted with caution. Because of the limitations of questionnaire data, and the lack of accurate diagnostic criteria when the study parents were children, the criteria used for classifying relatives as "affected" are, by necessity, extremely broad. They need to be validated in future studies by direct examination of the relatives. Similarly, sibling data in both groups may underrepresent the frequency of eventually affected siblings due to the young age of the probands in this study. Studies with older probands may give a more accurate account of affected sibling frequency in language-impaired and control families. It was also found that the language-impaired children had disproportionately more siblings (99 siblings for 62 probands) than the controls (57 siblings for 50 controls). Data analyses were designed to account for varying proportions in the data set when determining "affected" rates (i.e., rate = number of affected sibs/total number of sibs). Nonetheless, this difference may have biased the results in that language-impaired children had more siblings and, thus, may be more likely to be related to someone who was affected. However, this would not account for the differences between groups observed for both mothers and fathers where proportional differences between groups did not occur.

There have been a dearth of studies focusing on potential familial etiology of developmental language disorders. Though these results should be considered as preliminary, the robustness of the results, based on questionnaire data, indicate a fruitful area for future study. Such studies might focus on evaluation of genetic and/or environmental factors that might be contributing to family aggregation. Potential phenotypic differences between language-impaired children, with or without a positive family history, would also be important to document. Finally, actual testing of family members to confirm

a diagnosis of language/learning impairment and to specify type and degree of impairment are also needed to validate the questionnaire data reported in this study.

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REFERENCES

ARNOLD, G. E. (1961). The genetic background of developmental language disorders. *Folia Phoniatrica, 13*, 246–254.

BATES, E., BRETHERTON, I., & SNYDER, L. (1987). *From first words to grammar: Individual differences and dissociable mechanisms*. New York: Cambridge University Press.

BEITCHMAN, J. H., NAIR, R., FERGUSON, M., & PATEL, P. G. (1986). Prevalence of psychiatric disorders in children with speech and language disorders. *Journal of the American Academy of Child Psychiatry, 25*, 528–535.

BENTON, A. L. (1964). Developmental aphasia and brain damage. *Cortex, 1*, 40–52.

BISHOP, D. V. M., & EDMUNDSON, A. (1986). Is otitis media a major cause of specific developmental language disorders? *British Journal of Disorders of Communication 21*, 321–338.

BORGES-OSORIO, M. R., & SALZANO, F. M. (1985). Language disabilities in 3 twin pairs and their relatives. *Acta Geneticae Medicae et Gemellologiae (Roma), 34*, 95–100.

CARROW, E. (1973). *Test for Auditory Comprehension of Language*. Austin, TX: Urban Research Group.

CHILDS, B., & FINUCCI, J. M. (1979). The genetics of learning disabilities. In R. Porter, & M. O'Connor (Eds.), *Human genetics: Possibilities and realities* (pp. 46–53). New York: Excerpta Medica.

DECKER, S. N., & DEFRIES, J. C. (1980). Cognitive ability profiles in families with reading disabled children. *Journal of Learning Disabilities, 13*, 517–522.

DEFRIES, J. C., SINGER, S. M., FOCH, T. T., & LEWITTER, F. I. (1978). Familial nature of reading disability. *British Journal of Psychiatry, 132*, 361–367.

DERENZI, E., & VIGNOLO, L. A. (1962). The Token Test: A sensitive test to detect receptive disturbances in aphasics. *Brain, 85*, 665–678.

FOCH, T. T., DEFRIES, J. C., MCCLEAR, G. E., & SINGER, S. M. (1977). Familial patterns of impairment in reading disability. *Journal of Educational Psychology, 69*, 316–329.

FUNDALA, J. B. (1980). *The Arizona Articulation Proficiency Scale (AAPS), Revised*. Los Angeles: Western Psychological Services.

HENDRICK, D. E., PRATHER, E. M., & TOBIN, A. R. (1979). *Sequenced Inventory of Communication Development*. Seattle: University of Washington Press.

HOLLINGSHEAD, A. B., & REDLICH, F. C. (1958). *Social class and mental illness*. New York: John Wiley.

INGRAM; T. T. S. (1959). Specific developmental disorders of speech in childhood. *Brain, 82*, 450–467.

KIDD, K. K. (1980). Genetic models of stuttering. *Journal of Fluency Disorders, 5*, 187–201.

LEE, L. L. (1968). *Northwestern Syntax Screening Test*. Evanston, IL: Northwestern Press.

LEITER, R. (1940). *The Leiter International Performance Scale*. Santa Barbara, CA: State College Press.

LUDLOW, C. L., & COOPER, J. A. (Eds.). (1983). *Genetic aspects of speech and language disorders*. New York: Academic Press.

MCREADY, E. B. (1926). Defects in the zone of language (word-deafness and word-blindness) and their influence in education and behavior. *American Journal of Psychiatry, 6*, 267.

OWEN, F. W., ADAMS, P. A., FORREST, T. S., STOLZ, L. M., & FISHER, S. (1971). Learning disorders in children: Sibling studies. *Monographs of the Society for Research in Child Development, 36*.

ROBINSON, R. J. (1987). The causes of language disorders. In *Proceedings of the 1st international symposium on specific speech and language disorders in children* (pp. 1–19). Reading, England: Association for all Speech Impaired Children.

SAMPLES, J. M., & LANE, V. W. (1985). Genetic possibilities in six siblings with specific language learning disorders. *Asha, 27*(12), 27–32.

SILVA, P. A. (1980). The prevalence, stability, and significance of developmental language delay in preschool children. *Developmental Medicine and Child Neurology, 22*, 768–777.

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