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Identification of Early Risk Factors for Learning Disabilities

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The authors investigated birth risk factors for school-identified learning disabilities (LD) using a sample of 244,619 six- to eight-year-old public school children (6,715 LD) born in Florida between 1989 and 1990. Epidemiological measures of effect were used to investigate both individual- and population-level risk. Very low birth weight (VLBW), low 5-minute Apgar score, and low maternal education were associated with the highest individual-level risk. Low maternal education, late or no prenatal care, and tobacco use were associated with the highest population-level risk. Birth risk factors can be used to target screening and early intervention services for these high-risk children, which might be the most effective approach to reducing the incidence of school-identified LD.

Early identification and intervention on behalf of children who are at risk for subsequent developmental disability are essential in reducing the social, medical, and economic impact of these risks on both the individual and society (Brooks-Gunn, Klebanov, Liaw, & Duncan, 1995). The identification of children with LD requires the development of sophisticated surveillance systems that can be quite costly. However, using existing information collected by various public agencies can greatly reduce this expense.

Although the numerous existing definitions of LD can make this disability difficult to study, these various definitions are more similar than different. Basically, each definition reflects the philosophy of the organization that defined it. Hammill (1990, 1993) noted that the most influential definitions include many of the same major components (neurological, discrepancy, academic, and exclusion) in defining LD. The central criterion for identifying a child with LD, in all of the definitions, is a discrepancy between ability and achievement (Mercer, 1992). Ability is measured by intelligence tests, whereas achievement is indicated by performance on tests of standardized achievement (Hollomon, 1998).

The prevalence of LD among school-age children is subject to much dispute because of the lack of an agreed-upon definition of LD and objective diagnostic criteria (Lyon, 1996). Prevalence estimates reported in research studies vary from 2% to over 20% (Silver, 1988). The larger estimates reflect a liberal definition of the federal identification guidelines by Child Study teams and researchers, whereas the smaller estimates come from school districts that serve only children with severe learning disabilities because of the high expenses that would occur if more children were identified (Luick & Senf, 1979).

In 1978, 2.3% of the nation's school children were served under the LD category (U.S. Department of Education, 1980). In the 1998–1999 school year, the U.S. Department of Education (2000) reported that 4.5% of children ages 6 through 21 were served under the In-

dividuals with Disabilities Education Act (IDEA). This was a 36.6% increase compared to the percentage of children with LD served in this age group in the 1989–90 school year. The prevalence of LD varied substantially by state in 1998–1999, ranging from 2.4% (Kentucky) to 7.4% (Massachusetts). These numbers, however, only give an idea of how many children are identified with LD, but do not state the stringency of the diagnostic criteria used.

The substantial increase in the identification of children with LD has led many researchers to question the disability as a handicapping condition (Lyon, 1996). Some researchers have argued that the increase in the percentage of children identified with LD is a result of the misidentification of children with borderline mental retardation (MR) as children with LD (Gottlieb, Gottlieb, & Wishner, 1994). Others have argued that the prevalence rates of children with LD are high because changes or problems in society, such as pollution and abuse of drugs and alcohol, are leading to biomedical and psychological stress that result in a mild disability (Hallahan, 1992). Regardless of the reasoning behind higher prevalence rates, there are no set formulas to identify children with LD. This leads to a variety of characteristics of children identified in this category and variable prevalence estimates.

Because the leading definitions show agreement on the major components involved in defining LD, many of the current issues involve wording differences. The wording of the definition the National Joint Committee on Learning Disabilities (NJCLD) uses, appears to be the most adequate one in addressing the major components (Mercer, 1992). This definition states that "LD is intrinsic to the individual and presumed to be due to central nervous system (CNS) dysfunction. It might occur concomitantly with other handicapping conditions or environmental influences, but it is not the direct result of those conditions or influences" (Hammill, Leigh, McNutt, & Larsen, 1981, p. 336). Although Myers and Hammill (1990) limited the etiology of LD to central nervous system (CNS) irregularities, they also listed a number of environmental factors that might contribute to the severity of LD, such as insufficient early learning experience, behavior problems, cultural and linguistic differences, malnutrition, poor teaching, and lack of educational opportunity. When discussing which children are identified with LD, Lovitt (1989) included children who are environmentally impoverished, and Mercer (1992) listed exposure to lead and the influence of diet as environmental factors related to LD.

The medically related etiologies of LD are acquired trauma to the CNS, genetic or hereditary influences, environmental influences, and biochemical abnormalities (Mercer, 1992). Medical risk factors for acquired trauma to the CNS include maternal drug consumption, maternal diabetes, exposure to x-rays, maternal cigarette smoking, maternal infections such as measles or rubella, shortage of oxygen during labor, prematurity, prolonged labor, head trauma to the newborn, low birth weight (LBW), and low Apgar score (Andrews, Goldberg, Wellen, Pittman, & Struening, 1995; Mercer, 1992; Smith, 1994). The evidence for genetic influences on LD is substantial in that "different brain structures, patterns of brain maturation, biochemical irregularities, or susceptibility to diseases that impair brain functioning may be genetically transmitted" (Smith, 1994, p. 93). Also, boys are more often identified as having LD than girls (Lyon, 1996).

Environmental influences have been previously discussed, but they are often characterized by a disruption of learning as a result of exposure to environmental hazards such as drugs, alcohol, or pollution. Several biochemical imbalances in the production of neurotransmitters might lead to LD in children who otherwise would have had a good potential to learn. Some of these imbalances cause severe brain injury whereas others create a hyperactive or hypoactive state that makes it difficult for the child to remain focused and on-task (Smith, 1994). All of these medically related etiologies explain possible causes of LD, but the relative importance of these factors for individuals and the population is unknown.

The current study is a population-based investigation of the association of biological and

environmental risk factors present on birth certificates of children with school-identified LD at ages 6 to 8. The results described in this study are based on birth records obtained from the Florida Department of Health and school records obtained from the Florida Department of Education (FDOE).

METHOD

The data for these analyses were obtained from the Children's Services and Policy Research Center (CSPR) at the University of Miami, which operates demonstration projects and treatment programs that collect information about risk, early detection, and the cost effectiveness of community-based intervention and prevention programs. In the current study, electronic data-linkage methodology was used to investigate how various risk factors listed on birth certificate records could predict school-identified LD at ages 6 to 8 in a population of public school children in Florida.

Procedure

Data were derived from an electronic linkage of birth certificate records of all children born in Florida between January 1, 1989 and December 31, 1990 (n = 394,223) and FDOE public school records from the 1996–1997 school year of children born in 1989 or 1990 (n = 397,670). The method of matching was deterministic, based on an exact match of a child's first name, last name, sex, and date of birth. If any of the matching variables differed, the pair was considered a non-match and not included in the linked sample. Differential coding schemes in the birth and school records prevented exact matching based on race, but it was possible to clean the linked data set by removing any improbable discrepancies. For example, a linked record on which the child's race was listed as "white" on the birth record and "black" on the school records was considered an incorrect match and deleted from the data set. Birth records that were not linked were likely from children who moved out of Florida or attended a private school in 1996–1997. School records that were not linked were likely from children born outside Florida.

The final linked sample in this study refers to children born in Florida in 1989 or 1990 who were attending a Florida public school 6 to 8 years later. The choice of birth certificate years was constrained by the fact that many key variables such as maternal tobacco and alcohol use during pregnancy were not available on birth certificate records prior to 1989. The loss of children who attended private schools was not expected to have a substantial impact on the results of the current study because of the tendency for parents of children with disabilities, including those with higher socioeconomic status, to use the special education services the public schools provided. In Florida, many private centers also receive funding from the state, so children at these facilities appeared in the state database and were included in our sample. Thus, if bias were present, we believe that it would be in the direction of underestimating the effect of the risk factors. Interested readers are directed to Boussy and Scott (1993) for a more comprehensive review of the linkage procedures and the validation of this linkage methodology.

Sample

The sample in the current study consisted of 244,619 children who were born in Florida between 1989 and 1990 *and* who were in the Florida public school system during the 1996–1997 school year. The Florida public school system classified 6,715 of these children as LD. For most analyses, children with LD were compared to a control group of 208,390 children who had no exceptionality or were classified as gifted. Children with other exceptionalities (e.g., MR or emotional handicaps) were excluded from all analyses (n = 29,514).

Variables

The independent variables were obtained from birth certificate records, reflecting the child or family status information collected at birth. Maternal variables abstracted from the birth certificate records were: medical history factors for this pregnancy (e.g., anemia, diabetes, eclampsia), age, education, marital status,

month prenatal care began, and tobacco and alcohol use during pregnancy. Child variables related to labor/delivery consisted of labor/delivery complications (e.g., placenta previa, breech presentation, fetal distress), gestational age, birth weight, 5-minute Apgar score, abnormal conditions of the newborn (e.g., anemia, meconium aspiration syndrome, assisted ventilation), and congenital abnormalities (e.g., spina bifida, Down syndrome, heart malformations).

It should be noted that all of the birth variables except sex, gestational age, and birth weight contained missing or invalid values. The percentage of missing values was very small (less than 1% for most variables) and was more likely to occur in relation to detailed medical information, which had to be abstracted from the medical record, rather than demographic variables. Variables with more than 1% of the values missing were newborn complications (1.7%), labor/delivery complications (1.2%), and congenital abnormalities (1.1%). For each rate calculation, only records with valid entries for the relevant variable(s) were included.

The outcome variable was primary special education placement at the end of the 1996–1997 school year, which was identified from FDOE public school records. In Florida, placement decisions for children with LD are made based on a discrepancy between IQ and achievement. Specifically, LD is defined as "a heterogeneous group of psychological processing disorders manifested by significant difficulties in the acquisition and use of language, reading, writing, or mathematics. These disorders are intrinsic to the individual and might occur across the life span. Although LD might occur concomitantly with other handicapping conditions or with extrinsic influence, the disabilities are not primarily the result of those conditions or influences" (Florida Department of Education, 2001).

Epidemiological Framework

Whereas traditional regression or ANOVA models focus on means, slopes, and variances, epidemiological measures of effect focus on proportions and provide an inherently different measure of effect which can provide unique insight into the study of risk (Mason, Scott, Chapman, & Tu, 2000). An epidemiological perspective was preferred in the current study for three reasons. First, with many uncommon outcomes, such as LBW, it might be mathematically impossible to obtain a large correlation or account for a large proportion of the variance. This is particularly true in cases when dealing with common risk factors such as poverty or low maternal education.

Second, epidemiological methods are particularly suited for public policy decisions that are concerned with a small percentage of the population. Because epidemiological measures of effect focus on rates, ratios, and proportions, they map well onto typical concerns of policymakers, such as the reduction in the number of cases of a disorder or the specific cost savings associated with successful prevention of a given risk factor (Scott, Mason, & Chapman, 1999). In addition, those outside the field who have not had any statistical training are more likely to readily understand rates and proportions than correlations and variance.

Third, epidemiological measures of effect can address the effect of a risk factor on overall rates of a disorder in the community. The distinction between community- and individual-level risk is important because relatively rare risk factors, such as LBW, might have a large effect on individuals who experience it, but have a small impact on the overall number of cases in the community because so few are exposed to that risk factor. On the other hand, a more prevalent risk factor, such as poverty, might have a more modest effect on individuals but might have a large effect on the rates of disorder in the population because it is so common (Mason et al., 2000).

Risk ratio (RR). The RR is the relative increase in the probability of a given outcome when one rather than another condition is true. The RR is typically the increased probability of the occurrence of some adverse outcome, given exposure to a particular risk factor, relative to a comparison or referent group. In the current study, each risk category was compared to the lowest risk group, which was as-

signed a RR of 1.0. As an example, the RR for VLBW among boys using normal birth weight (NBW) as a reference was calculated as follows:

$$RR_{VLBW} = \frac{\text{Rate of LD for males born VLBW}}{\text{Rate of LD for males born NBW}} = 2.4$$

The obtained RR indicates that the rate of LD among boys born VLBW is 2.4 times greater than the rate of LD among boys born NBW. For each RR, 95% confidence intervals were also calculated. These intervals indicate the lower and upper limit of the RR which contains the true parameter 95% of the time over unlimited repetitions of the study, assuming there was no bias. Thus, confidence intervals with a lower limit less than or equal to 1.0 were not considered meaningful effects because one cannot be confident that the rate of LD among the risk group was truly different from the rate found in the referent group. Confidence intervals with large discrepancies between the lower and upper bounds are usually indicative of a small sample size in the risk group, referent group, or both.

Population attributable fraction percent (PAF%). The PAF% is an important estimate of risk to the *population* that weighs the RR based on the prevalence of a given risk factor in the population (Mason et al., 2000). The RR only considers the relative difference between two rates, and not the overall magnitude. The same RR (3.0) is obtained whether the rates of a given outcome are 99% and 33%, 30% and 10%, or .0075% and .0025%. Consideration of the number of individuals in the population exposed to a given risk factor is critical for intervention planning. A risk factor such as VLBW, which occurs in approximately 2% of children with LD, cannot have a large effect on overall rates of LD in the community. Even if an intervention prevented LD placements in *all* children with VLBW, it would fail to address the 98% of children with LD who did *not* have VLBW.

Assuming a causal relationship, the PAF% estimates the effect of a risk factor on the population as a whole. It is the proportion by which the rate of a given outcome (e.g., LD) would be reduced *in the population* if the rate associated with a given risk factor (e.g., VLBW) was reduced to that of the referent group (e.g., NBW). Even if a causal relationship cannot be established, the PAF% will still serve to identify the group which is having the largest impact on the overall rate or number of cases in the population (Scott et al., 1999). This high-risk group can be targeted for services or programs aimed at reducing their rates of disorder. A more complete description of the calculation of these epidemiological statistics and their derivation can be found in Mason et al. (2000).

RESULTS

Preliminary Analyses

Data transformations. Prior to conducting the analyses, continuous independent variables were coded into categorical variables because calculation of the risk ratio (RR) and population attributable fraction percent (PAF%) requires categorization (Mason et al., 2000). Although categorization of continuous variables typically reduces statistical power (Mason, Tu, & Cause, 1996), given the very large sample, a decrease in power was not a concern in this study.

The distribution of study characteristics is presented in Table 1. The largest differences between children with LD and the comparison children were found for sex, maternal education level, and prenatal care. Overall, the administrative prevalence rate for LD at 6 to 8 years of age was 27 per 1,000 or 2.7% of the total sample population. The rate of LD for boys was 38 per 1,000 whereas the rate for girls was 17 per 1,000. Because identification rates for LD were widely discrepant between sexes, separate analyses were conducted for boys and girls.

Table 2 displays the unadjusted RR and PAF% for LD associated with each of the 11 study variables, stratified by sex. Despite the large differences in identification rates of LD found between boys and girls, risk patterns within each sex were strikingly similar. For both boys and girls, the variables associated

Table 1.
The Distribution of Study Characteristics Among 6- to 8-Year-Old Children With and Without Learning Disabilities, State of Florida, 1996–1997.

	Children With LD (n = 6,715)		Comparison Group (n = 208,390)	
	n	%	n	%
Child risk factors				
Sex				
Boys	4,750	71	101,963	49
Girls	1,965	29	106,427	51
Gestational age				
<37 weeks	764	11	15,834	8
37–42 weeks	5,660	84	185,573	89
>42 weeks	291	4	6,983	3
Birth weight				
VLBW (<1500g)	133	2	1,629	1
MLBW (1500–2499g)	579	9	12,319	6
NBW (≥2500g)	6,003	89	194,442	93
5-Minute Apgar				
≤3	20	0.3	272	0.1
4 to 6	105	2	1,534	1
≥7	6,552	98	205,671	99
Newborn conditions				
Yes	392	6	10,288	5
No	6,133	94	194,558	95
Congenital abnormalities				
Yes	146	2	2,882	1
No	6,441	98	203,256	99
Maternal risk factors				
Maternal education				
<12 years	2,336	35	54,892	27
=12 years	2,770	41	85,553	41
>12 years	1,571	24	66,700	32
Maternal age				
<18 years	472	7	12,423	6
18–35 years	5,892	88	183,774	88
>35 years	351	5	12,191	6
Mother married				
Yes	4,251	63	141,640	68
No	2,463	37	66,736	32
Prenatal care began				
1st trimester	4,360	65	147,426	71
2nd trimester	1,658	25	44,457	22
3rd trimester/none	648	10	14,741	7
Tobacco use				
Yes	1,695	25	38,767	19
No	4,996	75	169,043	81

l Children With and Without

Comparison Group (n = 208,390)	
n	%
101,963	49
106,427	51
15,834	8
185,573	89
6,983	3
1,629	1
12,319	6
194,442	93
272	0.1
1,534	1
205,671	99
10,288	5
194,558	95
2,882	1
203,256	99
54,892	27
85,553	41
66,700	32
12,423	6
183,774	88
12,191	6
141,640	68
66,736	32
147,426	71
44,457	22
14,741	7
38,767	19
169,043	81

Table 1.
Continued

	Children With LD (n = 6,715)		Comparison Group (n = 208,390)	
	n	%	n	%
Alcohol use				
Yes	306	5	6,738	3
No	6,380	95	201,018	97
Medical history factors				
Yes	1,572	24	43,024	21
No	5,008	76	162,682	79
Labor complications				
Yes	2,098	32	63,842	31
No	4,476	68	142,012	69

with the highest individual-level risk were VLBW, low 5-minute Apgar score and low maternal education. The RR for boys and RR for girls associated with all variables were comparable with the exception of VLBW and low Apgar score; girls were at greater risk than boys. However, for all variables, the RR confidence intervals overlapped between boys and girls. It should be noted that some risk factors, such as Apgar score ≤ 3 and VLBW have a very low prevalence, especially in a sample such as this one, which excluded cases of infant mortality. Thus, the reader is cautioned to note the sample size when interpreting the data in Table 2.

Table 3 presents the unadjusted RR and PAF% associated with each risk factor for the whole sample. Because the two components of the PAF%, the RR and exposure prevalence, were nearly identical for boys and girls, PAF% calculations were not stratified by sex. In addition, the PAF% was not computed for RRs whose lower 95% confidence limit was ≤ 1.0. As expected, the whole-sample RRs reflected the pattern of risk observed in the stratified analysis. VLBW, low Apgar score, and low maternal education were associated with the highest individual-level risk. The increased sample size resulted in much tighter confidence intervals surrounding the RR calculations, especially for the lower prevalence risk factors. Population-level risk was greatest for maternal education ≤ 12 years (combined PAF% = 25), late prenatal care (combined PAF% = 8) and tobacco use (PAF% = 8.0).

DISCUSSION

The current study investigated the association of risk factors present at birth with school-identified learning disabilities at ages 6 to 8. The goal was to identify those risk factors present at birth that could place an individual at risk for being identified with LD in the future.

Learning disabilities were far more prevalent among boys than girls in this sample. Similar to previous studies (see Kavale & Reese, 1992; Shaywitz, Shaywitz, Fletcher, & Escobar, 1990) this study reports a 2.4:1 ratio of boys to girls with school-identified LD. The pattern of risk factors for boys and girls was nearly identical, with the exception of a low 5-minute Apgar score and VLBW, where girls had higher rates of LD than boys did. It should be noted that these are two of the least common risk factors in the current study. In addition, being a boy, low Apgar score, and VLBW have been identified as strong predictors of infant mortality and mental retardation (Andrews et al., 1995; Brohman, Nichols, Shaughnessy, & Kennedy, 1987; Camp, Brohman, Nichols, & Leff, 1998; Mervis, Decoufle, Murphy, & Yeargin-Allsopp, 1995)—two outcomes not included in the current study.

Table 2.
Unadjusted Risk Ratio Associated With Various Risk Factors Present at Birth on Rates of School-Identified Learning Disabilities at Age 6 to 8, Stratified by Sex.

	Boys		Girls	
	n	RR (95% CI)	*n*	RR (95% CI)
Child risk factors				
Gestational age				
<37 weeks	10,196	1.5 (1.4–1.6)	9,683	1.7 (1.5–2.0)
37–42 weeks	111,500	1.0	104,913	1.0
>42 weeks	4,331	1.3 (1.2–1.5)	3,996	1.4 (1.1–1.7)
Birth weight				
VLBW (<1500g)	1,161	2.4 (2.0–3.0)	1,304	3.2 (2.5–4.2)
MLBW (1500–2499g)	7,070	1.6 (1.4–1.7)	8,319	1.6 (1.4–1.9)
NBW (≥2500g)	117,796	1.0	108,969	1.0
5-Minute Apgar				
≤3	193	1.8 (1.0–3.2)	201	3.3 (1.7–6.2)
4 to 6	1,193	1.9 (1.5–2.5)	970	2.3 (1.6–3.3)
≥7	124,118	1.0	116,822	1.0
Newborn conditions				
Yes	6,884	1.2 (1.0–1.3)	5,871	1.2 (1.0–1.5)
No	116,916	1.0	110,685	1.0
Congenital abnormalities				
Yes	2,153	1.5 (1.2–1.8)	1,729	1.7 (1.2–2.3)
No	122,431	1.0	115,562	1.0
Maternal risk factors				
Maternal education				
<12 years	34,049	1.8 (1.6–1.9)	32,655	2.0 (1.7–2.2)
=12 years	51,585	1.3 (1.3–1.4)	48,694	1.5 (1.3–1.6)
>12 years	39,629	1.0	36,555	1.0
Maternal age				
<18 years	7,554	1.4 (1.2–1.6)	7,281	1.2 (1.0–1.6)
18–35 years	111,143	1.2 (1.0–1.3)	104,398	1.0 (0.8–1.3)
>35 years	7,330	1.0	6,911	1.0
Mother married				
Yes	85,390	1.0	79,305	1.0
No	40,630	1.2 (1.1–1.3)	39,276	1.3 (1.2–1.4)
Prenatal care began				
1st trimester	88,275	1.0	83,074	1.0
2nd trimester	27,471	1.2 (1.1–1.4)	25,666	1.2 (1.1–1.4)
3rd trimester/none	9,165	1.5 (1.4–1.7)	8,836	1.4 (1.2–1.7)
Tobacco use				
Yes	24,296	1.5 (1.5–1.7)	23,132	1.4 (1.2–1.5)
No	101,363	1.0	95,078	1.0
Alcohol use				
Yes	4,226	1.4 (1.2–1.6)	4,008	1.5 (1.2–1.9)
No	121,389	1.0	114,176	1.0

Girls	
n	RR (95% CI)
9,683	1.7 (1.5–2.0)
104,913	1.0
3,996	1.4 (1.1–1.7)
1,304	3.2 (2.5–4.2)
8,319	1.6 (1.4–1.9)
108,969	1.0
201	3.3 (1.7–6.2)
970	2.3 (1.6–3.3)
116,822	1.0
5,871	1.2 (1.0–1.5)
110,685	1.0
1,729	1.7 (1.2–2.3)
115,562	1.0
32,655	2.0 (1.7–2.2)
48,694	1.5 (1.3–1.6)
36,555	1.0
7,281	1.2 (1.0–1.6)
104,398	1.0 (0.8–1.3)
6,911	1.0
79,305	1.0
39,276	1.3 (1.2–1.4)
83,074	1.0
25,666	1.2 (1.1–1.4)
8,836	1.4 (1.2–1.7)
23,132	1.4 (1.2–1.5)
95,078	1.0
4,008	1.5 (1.2–1.9)
114,176	1.0

Table 2.
Continued

	Boys		Girls	
	n	RR (95% CI)	*n*	RR (95% CI)
Medical history factors				
Yes	26,775	1.2 (1.1–1.3)	24,905	1.1 (1.0–1.3)
No	97,608	1.0	92,145	1.0
Labor complications				
Yes	39,861	1.0 (1.0–1.1)	35,996	1.0 (1.0–1.1)
No	84,627	1.0	81,129	1.0

Note. RR = Risk Ratio, CI = 95% Confidence Interval.

The prevalence rate of LD in the current study was 2.7%, whereas the state of Florida reported serving 4.8% of children ages 6–21 during the 1996–97 school year under the same LD category (U.S. Department of Education, 1998). The lower rate of LD in this sample was expected given that even using the most recent available dataset, children in our sample were 6 to 8 years old. Often LD is not identified until later ages. Thus the results of the current study might actually underestimate the effects of risk factors.

Overall, in terms of individual-level risk, VLBW, a low 5-minute Apgar score, and low maternal education were the most salient predictors of school-identified LD. Other risk factors associated with an increase in risk were late or no prenatal care, maternal tobacco use, maternal alcohol use, preterm births, and the presence of a congenital anomaly. This suggests that LD is affected by a combination of perinatal and sociodemographic factors–a finding consistent with previous studies. The emerging research on maternal education has shown that children born to mothers without a high school diploma are at risk for both cognitive and behavioral problems, partially because of deficits in the mother's knowledge of child development and parenting skills (Furstenberg, Brooks-Gunn, & Chase-Landale, 1989; Kochanek, Kabacoff, & Lipsitt, 1990; Miller & Moore, 1990). Gestational age, especially for those born before 37 weeks, has also been associated with later learning disabilities (Cherkes-Julkowski, 1998; Rossetti, 1986; Shaywitz, Shaywitz, McGraw, & Groll, 1984; Touwen & Huisjes, 1984). Other research indicates that minor congenital abnormalities that have their origins prior to birth often are associated with later LD (Bell & Waldrop, 1989; Von Hilsheimer & Kurko, 1979). Finally, LBW has been associated with learning disabilities (Bendersky & Lewis, 1994; Breslau et al., 1994; Resnick et al., 1998; Saigal, Rosenbaum, Szatmari, & Campbell, 1991). Results of these studies indicate that the association becomes stronger as birth weight decreases.

The role of Apgar scores in predicting later LD is not clear from the literature. Andrews et al. (1995) reported that Apgar scores of less than 8 predict LD. Similarly, in the current study, low 5-minute Apgar scores were associated with more than double the risk for LD at age 6 to 8 years, compared to children with higher Apgar scores (≥ 7). Other studies, however, found that low Apgar scores were poorly correlated with long-term intellectual outcome (Blackman, 1988; Seidman et al., 1991). It is possible that these researchers failed to find an association between low Apgar scores and poor cognitive performance because they did not follow the children long enough to identify them with LD, or because their identification procedure was limited to the use of IQ tests only. As stated previously, the central criterion for the identification of LD is a discrepancy between ability and achievement. Without achievement test information, a discrepancy between IQ and achievement cannot be determined.

An interesting finding that needs to be ex-

Table 3.
Whole Sample RR and PAF% Associated With Various Risk Factors Present at Birth on Rates of School-Identified Learning Disabilities at Age 6 to 8.

	n	RR (95% CI)	Risk Factor Prevalence (%)	PAF%
		Child risk factors		
Gestational age				
<37 weeks	19,879	1.6 (1.5–1.7)	9	5
37–42 weeks	216,413	1.0	91	—
>42 weeks	8,327	1.4 (1.2–1.5)	1	0.2
Birth weight				
VLBW (<1500g)	2,465	2.5 (2.1–3.0)	2	2
MLBW (1500–2499g)	15,389	1.5 (1.4–1.6)	6	3
NBW (≥2500g)	226,765	1.0	92	—
5-Minute Apgar				
≤3	394	2.2 (1.5–3.4)	1	1
4 to 6	2,163	2.1 (1.7–2.6)	1	1
≥7	240,940	1.0	98	—
Newborn conditions				
Yes	12,755	1.2 (1.1–1.3)	5	1
No	227,601	1.0	95	—
Congenital abnormalities				
Yes	3,882	1.6 (1.3–1.8)	2	1
No	237,993	1.0	98	—
		Maternal risk factors		
Maternal education				
<12 years	66,704	1.8 (1.7–1.9)	25	14
=12 years	100,279	1.4 (1.3–1.5)	39	11
>12 years	76,184	1.0	36	—
Maternal age				
<18 years	14,385	1.3 (1.1–1.5)	9	3
18–35 years	215,541	1.1 (1.0–1.2)	85	—
>35 years	14,241	1.0	6	—
Mother married				
Yes	164,695	1.0	70	—
No	79,906	1.2 (1.2–1.3)	30	6
Prenatal care began				
1st trimester	171,349	1.0	72	—
2nd trimester	53,137	1.3 (1.2–1.4)	21	5
3rd trimester/none	18,001	1.5 (1.4–1.6)	8	3
Tobacco use				
Yes	47,428	1.5 (1.4–1.6)	19	8
No	196,441	1.0	81	—
Alcohol use				
Yes	8,234	1.4 (1.3–1.6)	3	1
No	235,565	1.0	97	—

Risk Factor Prevalence (%)	PAF%
9	5
91	—
1	0.2
2	2
6	3
92	—
1	1
1	1
98	—
5	1
95	—
2	1
98	—
25	14
39	11
36	—
9	3
85	—
6	—
70	—
30	6
72	—
21	5
8	3
19	8
81	—
3	1
97	—

Table 3.
Continued

	n	RR (95% CI)	Risk Factor Prevalence (%)	PAF%
Medical history factors				
Yes	51,680	1.2 (1.1–1.3)	21	4
No	189,753	1.0	79	—
Labor complications				
Yes	75,857	1.0 (1.0–1.1)	32	1
No	165,756	1.0	68	—

Note. RR = Risk Ratio, CI = 95% Confidence Interval, PAF% = Population Attributable Fraction Percent.

amined further is the risk associated with maternal smoking, which poses a high individual-level risk and is highly prevalent in the population. Our results verify the negative impact of smoking during pregnancy on the intellectual growth of the child and are consistent with results from other studies (Bauman, Flewelling, & LaPrelle, 1991; Fogelman, 1980; Fogelman & Manor, 1988; Fried & Watkinson, 1990; Frydman, 1996; Sexton, Fox, & Hebel, 1990). Future studies should investigate the dose-response relationship of smoking and the prevalence of LD and the interaction of smoking with other risk factors.

Although measures of risk to the individual provide information about which children need early intervention services, they do not address the impact of intervening with those children on a community-wide or population-wide basis (Hollomon, 1998). Public policy makers want to assign funding to interventions that are effective and have an impact on the largest number of people. For this reason, the PFA% can be used to identify a subset of the population that is having a large impact upon rates of a disorder in the community (Scott et al., 1999).

To illustrate the difference between individual-level and population-level risk, consider the 5-minute Apgar score of 3 or less. This risk factor places individuals at a substantial risk for LD. An intervention that successfully prevents all children with Apgar scores ≤ 3 from having an LD placement, however, will only reduce the rate of LD in the study population by 0.6% because only about 5 out of every 1,000 (surviving) births have an Apgar score ≤ 3. On the other hand, an intervention targeting a risk factor that occurs more frequently in the population, such as maternal education < 12 years, which is present in one fourth of all births, would have a much greater potential impact on rates of LD in the population (14%).

Maternal education might operate directly through the child-rearing environment, and it certainly serves as a marker for many of the other risk factors in this study. Thus, whereas mothers with low education are an important *group* to target for intervention, receipt of timely prenatal care and tobacco use are specific risk *behaviors* that have the best potential for reducing the incidence of LD in the population.

Individual-level risk factors and population-level risk factors are both important in determining who could be eligible for services. Child Find professionals and parents should use the individual risk information to determine who should be tested for developmental disabilities. For example, children born with a congenital anomaly had a greater chance of having a learning disability than children without a congenital anomaly. Child Find professionals and parents who have a child with a congenital anomaly should have their child tested for developmental disabilities since they are at increased risk. Professionals who

are designing an intervention would be better served, however, to consider the population-level information that is having the largest impact on LD placements in the population to target services to the group.

One limitation of the current study is the use of school-identified learning disabilities, an admittedly heterogeneous group of children. It is important to keep in mind that the placement of children into special education services is based on professional judgment, which can result in errors. It is also important, however, to examine the characteristics of who is being served in the schools under the LD label, because federal funding is provided for these children. Although these children might not meet the eligibility criteria based on theoretical definitions, they are being serviced in the schools and warrant further study.

Complications in the perinatal period and sociodemographic factors have a negative impact upon a child's later cognitive development. There does not seem to be a one-to-one relationship in most cases, however, between individual events and future difficulties. Although detailed medical records were not available in the current study, the association of LBW with other complications such as preterm birth and oxygen deprivation is well known (Gatten, Arceneaux, Dean, & Anderson, 1994). When multiple complications are present, the child has a greater chance of being identified with LD in the school setting. Integration of descriptive epidemiological findings with more detailed medical records is an important direction for future research.

Concluding, the results of this study describe various risk factors present at birth, which place children at high-risk for a future learning disability. Screening and early identification of these high-risk children are critical, because previous studies have shown that many children classified as LD were not identified for educational services until the elementary school years (Bowe, 1989; Gerber & Levine-Donnerstein, 1989; Scott, Urbano, & Boussey, 1991). Because relying on risk factors alone would lead to over-identification, researchers have begun to work on the screening of children who are at-risk for LD. Scott and colleagues suggested that a screening test composed entirely of tasks that required active cognitive processing would improve early identification of children with mild cognitive impairments (Scott, Fletcher, Jean-Francois, Urbano, & Sanchez, 1998; Scott, Fletcher, & Martell, 2000). Early identification will permit educational interventions to begin at the earliest age possible, and therefore maximize the effect of the intervention. These early intervention services might be the most effective approach to reducing the incidence of school-identified LD.

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