Risk for Reading Disability as a Function of Parental History in Three Family Studies

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ABSTRACT: Inverse Bayesian analyses were applied to data from three large family studies of reading disability to estimate the posterior probability that an offspring will be affected, given that a parent reported a history of learning problems. Prior analyses presented elsewhere (Pennington et al., 1990), suggest that family transmission in these three studies is consistent with major gene or polygene influence. Posterior probability rates are presented in this paper for male to female sex ratios of 3.5:1 and 1:1, with population incidences estimated at 0.05 and 0.10. Results indicate that offspring risk rates are significantly elevated if a parent reports a history of RD. Specifically, an offspring's risk was increased 2 to 80 times over population expectancies when there was an affected parent. While the posterior probabilities and relative risk rates were fairly similar across studies, there was also some variation, which may reflect the different genetic mechanisms operating in these families. This study concludes that both absolute and relative risks are sufficiently increased in families with RD parents to warrant use of family history as a component in clinical evaluation. It is also evident from these results that consideration of the apparent mode of genetic transmission in families may provide even better information as to offspring risk, when family history is obtained.

KEYWORDS: Reading disability, genetics, Bayesian probability, risk, history.

Several pre, peri, and post-natal factors have been shown to place a child at risk for developing a learning disability (Schulman and Leviton, 1978; Dworkin, 1985; Satz and Friel, 1974; Satz, Taylor, Friel, and Fletcher, 1978; Lewis, 1980; DeRuiter, Ferrell, and Kass, 1975; Wissink, Kass, and Ferrell, 1975). Included among these risk factors is a family history of learning problems (Pennington and Smith, 1988). In clinical practice it is commonly observed that a child patient may have other family members who also have learning difficulties. Frequently, a parent will report multiple generations where the patient's relatives had difficulty in reading or spelling, even as adults. Thus it appears that family history may be among the important risk factors in the development of a learning disorder. This conclusion is also suggested by the family and twin studies showing a heritable component to learning disabilities (Pennington and Smith, 1988).

Using data from the Colorado Family Reading Study (DeFries and Decker, 1982; DeFries, Vogler, and LaBuda, 1986), Vogler, DeFries and

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Decker (1985) examined how a child's risk for being reading disabled was modified by the presence of parental self-report history for reading problems. Using inverse Bayesian probability, the increase of risk was anywhere from approximately 4 to 13 times larger if a child had a parent affected according to history, than if the parent reported no history of reading difficulties. The authors concluded that the increase in risk as a function of parental history was high enough to warrant incorporation into a clinical protocol.

For a condition such as reading disability (RD), where genetic influence has been implicated, the probability that an affected parent's offspring will also be affected depends on a number of factors. One of these factors is the type of genetic mechanism involved. For instance, in a condition where there is a completely dominant, fully expressed gene acting, and one parent is affected, we expect, on average, at least 50% of the offspring to also be affected. In the case where many different genes, or polygenes, contribute equally to the condition, such that a certain number of these genes are required for an offspring to be affected, the probability of the condition being expressed in offspring is more difficult to predict, though it is probably less than 50%. The reason why the inheritance patterns of polygenic conditions are more difficult to define precisely, is that they depend more heavily on the genetic make-up of both parents, and which alleles or genes an offspring inherits from either parent, and how these alleles may interact.

We have recently completed complex genetic segregation analyses on four data sets, including the Colorado Family Reading Study, where families were ascertained through an RD adult or child (Pennington, Gilger, Pauls, Smith, Smith, and DeFries, 1990). Three of these data sets yielded results consistent with what would be expected if a single gene was contributing to the transmission of reading disorders in these families. Although major gene influence was not evident in the fourth data set, multi-factorial/polygenic influence appeared to be a significant contributor to the familiality of reading problems. It is noteworthy, that in the three data sets manifesting apparent major gene influence, estimates of the magnitude of the genetic effect, the disease threshold, gene frequency, and sex-dependent penetrances, were all very similar. Genetic heterogeneity is still possible however, and similar parameter estimates across these data sets does not prove that the same gene is operating.

In this paper we repeat the Bayesian analyses of Vogler, DeFies, and Decker (1985) on these data sets, with three major objectives in mind: First, we will examine how, and if, the risk estimates vary across the samples, and how these estimates compare to those reported by Vogler et al. (1985). Second, we will examine whether or not any variability in these estimates is tied to differences in the type of genetic mechanism (polygenic versus major gene) implicated in these families. And third, we will present

posterior probabilities based on calculations representing the upper (3.5:1) and lower (1:1) bounds of the estimated male to female sex ratio for RD in the population. Rates for the two sex ratios will be given for comparison purposes. Where it was once believed that RD males substantially outnumbered RD females, some recent studies have in fact demonstrated that, after controlling for selection biases, the actual sex ratio may approach unity (DeFries, Olson, Pennington, Smith, 1990; Shaywitz, Shaywitz, Fletcher, and Escobar, 1990).

METHOD

Three out of the four family data sets were of sufficient size to be included in this paper: The Iowa Family Study of Reading Disabilities (Gilger, 1990a), the reading disabled families from the linkage work of Smith, Pennington, and colleagues (Smith et al., 1983; Pennington et al., 1984), and data from the Colorado Family Reading Study (DeFries and Decker, 1982; DeFries, Vogler, and LaBuda, 1986) which were also used in the Vogler et al. report. Each sample was ascertained in a different manner from different populations, and the individual methodologies are summarized below.

The Iowa Study

Subjects. As part of an ongoing study of dyslexia, data have been gathered for three generations of 40 families selected through a dyslexic proband seen at the University of Iowa Pediatric Psychology Clinic. Dyslexic probands met the inclusionary and exclusionary criteria detailed in DSM III (American Psychiatric Association, 1987). A further requirement was that all probands demonstrated the "memory deficit" subtype of the University of Iowa diagnostic scheme (Richman, 1983; Lindgren, Richman, and Eliason, 1986). Children possessing the memory subtype are distinguishable from other reading disabled children by their characteristic pattern of memory deficits and largely normal functioning in general intelligence, perceptual-motor skills, and associative reasoning. Specifically, in addition to the classic symptoms of dyslexia, the memory disordered group demonstrates the following characteristics: 1. A verbal IQ within 11 points of their Performance IQ; 2. A Verbal and Performance IQ of at least 90; 3. Scores at least 1 standard deviation below average on more than one memory test (e.g., short or long term verbal and visual memory tests), while showing no deficits in associative reasoning and visuo-perceptual skills. All index cases were between 9 and 18 years of age at the time they were seen in the pediatrics clinic.

Subsequent to identifying an appropriate proband, the participation of relatives was solicited through the mail, and followed-up by telephone. All proband siblings, aunts, uncles, grandparents, and cousins in affected kindreds were asked to take part in the study. Thus far data have been collected for approximately 660 individuals of these RD kindreds. Data on a set of control probands (matched to affected probands on SES, grade, sex and age), and their immediate families have also been collected. Though appropriate matches for all affected probands have not yet been obtained, data are available on approximately 500 individuals from these control families.

Materials and RD Diagnosis. As part of the study, adult subjects complete questionnaires at home and return them by mail. Brief telephone interviews are also conducted. Topics addressed by the surveys pertain to aspects of the respondent's physical and socio-emotional development, and the presence of symptoms suggestive of learning disabilities and behavioral disorders. Adequate validity and reliability of self and parent reports has been demonstrated for a variety of the questionnaire items used in the Iowa study (Gilger, Geary, and Eisele, 1990; Gilger, 1990b).

Archival objective test data (national and state percentile scores) are also obtained for the probands, their siblings, cousins, aunts, uncles, and parents. Such data are made available through the University of Iowa Testing Program, which has maintained extensive records of the Iowa Tests of Basic Skills since their inception in the early 1940s (Hieronymus and Hoover, 1986; Iowa Testing Program, 1987). We have attempted to collect at least one set of scores representing the elementary school years (3rd—8th grades) and at least one set from the high school years for all subjects.

For the purposes of this paper, subjects were classified as either RD or not reading disabled (NRD) through an algorithm using the survey data. Probands and their parents were diagnosed as RD by history (i.e. ever having had special education, difficulty in learning while in school, poor academic achievement in the 1st through 3rd, 4th through 8th, or 9th through 12th grades). These questions and positive responses to them, were found to adequately discriminate between the RD and NRD matched control probands.

The Linkage Kindreds

Subjects. Over ten years ago a collaborative study was begun that was aimed at conducting a linkage analysis of families selected through a dyslexic proband (Smith et al., 1983; Smith et al., 1986). All probands were ascertained through clinics or referred from clinic sources, and only

those pedigrees suggestive of autosomal, major gene transmission (e.g., a three-generation history of familial reading problems) of dyslexia were asked to participate. Thus far, data on approximately 330 subjects from 21 three-generation kindreds have been obtained. Mean proband age in years is 18.9, with a standard deviation of 8.9.

Materials and RD Diagnosis. Subjects were tested and interviewed by trained personnel, though in some cases family members were either unable or unwilling to complete the study. A battery of tests and questionnaires were given to child and adult subjects, and blood samples were taken. Among the surveys was a handedness inventory and a Reading History survey (Finnucci, Isaacs, and Whitehouse, 1982). Medical, socio-emotional, and other general information was also obtained.

Subjects are diagnosed as RD if they report having had a history of reading problems on the Reading History Survey. On the rare occasion that self-report history data was not available, a subject may have been diagnosed as RD if person-to-person interviews with the subject, or information from a blood relative positively indicated reading difficulties.

The Colorado Family Reading Study

Subjects. Subjects were referred for the study by personnel of the Boulder Valley and St. Vrain Valley school districts in Colorado. All probands had IQ's of 90 or above; reading achievement level of one half, or lower, of grade expectancy; chronological age between 7.5 and 12 years; resided with both biological parents; met the exclusionary criteria of DSM III (e.g., no uncorrected visual deficits, no emotional impairments, etc.). Control children were matched to reading-disabled children on the basis of age (within 6 months), sex, grade, school, and neighborhood. Except for reading level, which was normal or above, the controls met the criteria used for the ascertainment of affected probands. In addition to the index cases, data were obtained on the parents and siblings of control and affected families. Data are currently available on approximately 565 individuals from 133 nuclear families selected through a reading disabled child.

Materials and RD Diagnosis. All subjects received an extensive 2—3 hour test battery which included measures of intelligence, academic skills, and specific cognitive abilities. Parental self-report and parent-child report survey information was also obtained on topics related to current and past academic, medical, and socio-emotional status.

RD diagnosis for this paper is the same as for the earlier Vogler et al. study. Specifically, subjects were classified as RD if they responded

positively to a single question addressing serious difficulty in learning to read.

ANALYSES

The ideal way to estimate the risk of being an affected offspring is to ascertain affected and unaffected groups of parents, and examine the frequency with which the children are affected with RD. However, family studies, such as the ones reported herein, are typically retrospective in nature, where the affection status of parents is determined after the family has been ascertained through an affected child. Thus, we cannot directly calculate the offspring probability of being RD, given that a parent is RD. An indirect method however, using an inverse Bayesian probability formula, does provide a means of estimating the likelihood a child will be affected given that the parental reading status is known.

The posterior probability that a child will be affected [P(C/R)], given that a parent is affected, can be found by the following equation (Winkler, 1972):

$$P(C/R) = \frac{P(C)P(R/C)}{P(C)P(R/C) + P(\text{Not C})P(R/\text{Not C})}$$

The parameters in the above equation are defined as follows: P(C) = the prior probability that a child will be RD, or an estimate of the population incidence; P(Not C) = 1 − P(C), or the likelihood that a child will not be RD; P(R/C) = the probability that a parent will be RD given that a child is RD, a value determined from the incidence of RD among parents of probands; P(R/C not C) = the probability that a parent will be RD given that the child is not RD, a value ascertained from the incidence of RD in parents of controls; P(C/R) = the posterior likelihood that a child will be RD given the parental affection status.

For this paper we followed a methodology similar to Vogler, DeFries and Decker (1985). First, we used two estimates of the population base rate for RD: 0.05, and 0.10 percent. We then used these rates to calculate P(C) separately for each sex, assuming a male to female sex ratio of 3.5:1, and again for a ratio of 1:1. Second, P(R/C) was calculated separately for mothers and fathers, and further subdivided by the sex of the proband. Finally, P(R/Not C) was obtained from the Colorado Family study control parents, and as noted in the Vogler et al. paper, 4% of the control fathers, and 3% of the control mothers reported a history of reading problems. There were essentially no differences in parental affection rates as a function of the sex of the proband. Since the Iowa control families were

not all adequately matched to Iowa RD families, their data were not used in the P(R/Not C) estimates.

In the analyses that follow, only the proband nuclear families were used. In the case of the Iowa and Linkage samples, nuclear families other than those of the probands (e.g., cousins) were available. We did not use these additional families because of ambiguity in defining a child proband from each, and a desire for consistency across the three samples, where only proband nuclear family data was always available. Moreover, only those nuclear families where diagnostic data were available for both parents were used. Thus, in some cases the N sizes may deviate slightly from those reported elsewhere in this paper.

RESULTS

Table 1 shows the probability of a mother or father being affected given affected or control offspring [P(R/C) and P(R/Not C)]. Several aspects of the data in Table 1 are noteworthy. First, there is some variability in the parental probabilities across the three studies. Estimates are most similar for the father affection status of male probands, and the mother affection status of female probands. Second, the rates of RD in the parents of RD probands are clearly elevated over population base rates (e.g., 0.05, and 0.10), and over the rates found in controls as well. Third, there are minor differences between the rates we report for the Colorado study and those reported by Vogler et al. This is a consequence of the differences in the

Table 1. Parental affection status of male and female RD and control probands[a]

	N	RD Father	NRD Father	RD Mother	NRD Mother
Iowa Study:					
Male Probands	26	0.35	0.65	0.12	0.88
Female Probands	12	0.17	0.83	0.42	0.58
Linkage Study:					
Male Probands	15	0.53	0.47	0.67	0.33
Female Probands	6	0.83	0.17	0.33	0.67
Colorado Study:					
Male Probands	99	0.30	0.70	0.15	0.85
Female Probands	27	0.41	0.59	0.30	0.70
Controls	182	0.04	0.96	0.03	0.97

[a] RD = Reading Disabled; NRD = Normal.

current (N = 126) and Vogler et al. (N = 174) Colorado samples. Specifically, the original rates reported for fathers of male and female probands were 0.29 and 0.36, respectively, and for mothers these rates were 0.17 and 0.25, respectively (Vogler, DeFries, and Decker, 1985). It is noteworthy that the Linkage study parent rates are somewhat elevated, while the rates in the Colorado and Iowa studies are more similar.

Tables 2 and 3 present probabilities and relative risks for being an RD child given an RD or NRD parent, for assumed sex ratios of 3.5:1 and 1:1, respectively. Examination of Tables 2 and 3 reveals that the probability of affection, given an RD parent, is consistently elevated over sex-specific population incidences for male and female offspring. However, the sex-specific likelihood of being an RD child, given an NRD parent, is not elevated over what we would expect given the population base rates. This is an important finding, since it suggests that it is really the status of the parent that matters in these families, rather than there being some artifact or bias such that children in these three studies are always more likely to be affected irrespective of whether or not a parent is RD.

Table 2. Probability (P[C/R]) that a child will be affected as a function of parental reading ability for a sex ratio of 3.5:1[a]

		Parental Affection Status				Risk[b]	
	P(C)[c]	RD Fa	NRD Fa	RD Mo	NRD Mo	Fa	Mo
Iowa Study:							
Male Child	0.078	0.425	0.054	0.202	0.072	8	3
	0.156	0.618	0.111	0.357	0.145	6	2
Female Child	0.022	0.113	0.019	0.240	0.013	6	19
	0.044	0.207	0.038	0.391	0.027	5	15
Linkage Study:							
Male Child	0.078	0.529	0.039	0.586	0.028	14	21
	0.156	0.710	0.083	0.756	0.059	9	13
Female Child	0.022	0.318	0.004	0.198	0.015	80	13
	0.044	0.488	0.008	0.336	0.038	61	9
Colorado Study:							
Male Child	0.078	0.388	0.058	0.297	0.069	7	4
	0.156	0.581	0.119	0.480	0.139	5	3
Female Child	0.022	0.187	0.014	0.184	0.016	13	12
	0.044	0.321	0.028	0.315	0.032	11	10

[a] Fa = Father; Mo = Mother; RD = Reading Disabled; NRD = Normal.

[b] Relative risk of being affected, rounded to nearest whole number = p(affected/parent affected)/p(affected/parent unaffected).

[c] Population incidence as a function of sex. Overall incidences were 0.05 and 0.10.

Table 3. Probability (P[C/R]) that a child will be affected as a function of parental reading ability for a sex ratio of 1:1[a]

		Parental Affection Status				Risk[b]	
	P(C)[c]	RD Fa	NRD Fa	RD Mo	NRD Mo	Fa	Mo
Iowa Study:							
Male Child	0.050	0.315	0.034	0.174	0.046	9	4
	0.100	0.493	0.069	0.308	0.091	7	3
Female Child	0.050	0.182	0.044	0.424	0.031	4	14
	0.100	0.321	0.088	0.609	0.062	4	10
Linkage Study:							
Male Child	0.050	0.411	0.025	0.540	0.018	16	30
	0.100	0.596	0.052	0.713	0.036	11	20
Female Child	0.050	0.522	0.009	0.367	0.035	58	10
	0.100	0.697	0.019	0.550	0.071	37	8
Colorado Study:							
Male Child	0.050	0.283	0.037	0.208	0.044	8	5
	0.100	0.455	0.075	0.357	0.089	6	4
Female Child	0.050	0.350	0.031	0.345	0.037	11	9
	0.100	0.532	0.064	0.526	0.074	8	7

[a] Fa = Father; Mo = Mother; RD = Reading Disabled; NRD = Normal.
[b] Relative risk of being affected, rounded to nearest whole number = p(affected/parent affected)/p(affected/parent unaffected).
[c] Population incidence as a function of sex. Overall incidences were 0.05 and 0.10.

As in Table 1, there are both similarities and dissimilarities across the three studies for the values shown in Tables 2 and 3. The studies appear most similar for the estimates of the mother's effect on female offspring, and the father's effect on male offspring.

In the last two columns of Tables 2 and 3 are the relative risk estimates given an affected or unaffected parent. It is obvious that relative risk varies depending on the sex of the proband and parent, and the sex-specific population incidences. Relative risk estimates vary from approximately 2 to 80 for a sex ratio of 3.5:1, and from 3 to 58 for a sex ratio of 1:1. Changing the sex ratio from 3.5:1 to 1:1, has the effect of increasing the comparable posterior probabilities for female children and decreasing them for male children.

The variability in the posterior probabilities in Tables 2 and 3 may be a consequence of different selection biases or genetic effects across the three studies. By carefully comparing the within offspring-sex and parent-sex posterior probabilities for being affected, given an affected parent, one can see that on the average, the largest estimates come from the Linkage

sample. On the other hand, there is a tendency for the smallest estimates to come from the Iowa sample. Specifically, the Iowa data set gave the smallest probabilities for 5 out of the 8 possible within child-sex and parent-sex comparisons. While small sample sizes mandate cautious interpretation of these data, they are somewhat consistent with the hypothesis of major (dominant or semi-dominant) gene influence in the Colorado, and especially the Linkage families, and polygenic inheritance in kindreds from Iowa. It is also noteworthy however, that the relative risk estimates and posterior probabilities of the Iowa and Colorado data sets are quite similar in magnitude.

Finally, the Colorado analyses of Vogler et al. indicated that the absolute risk for female offspring was smaller than that for males, though the female relative risk was roughly 1/2 to 2 1/2 times larger, depending on the population incidence used. While the relative risk estimates derived from the current Colorado data reflect this same trend, the Iowa and Linkage data do not for either of the two sex ratios used. For the Iowa sample, relative risks were higher for females only if the mother was RD, and in the Linkage sample, females demonstrated higher risks only if they had an RD father. However, for all three samples, the *average* relative risk estimates were larger for female offspring.

DISCUSSION

In general the results reported in this paper indicate that there is a substantial increase in the childhood risk for RD given an affected parent. Though there were some inconsistencies across studies of the posterior probability estimates, given the vast differences in the diagnostic criteria, design, and populations used in the studies, it is surprising how similar the pattern of results actually were, especially for the Iowa and Colorado samples. Some of the variability observed may reflect parameter instability due to the relatively small number of Iowa and Linkage proband nuclear families. Nonetheless, similar to Vogler, DeFries and Decker (1985), the data we report suggest that consideration of family (parental) history of reading problems may add important information pertinent to the diagnosis and prediction of reading disabilities in children. Our results show that using family data in conjunction with other risk indicators may provide a powerful diagnostic and predictive tool in future clinical and experimental work.

All three projects, particularly the Iowa and Linkage studies, are subject to the response biases prevalent among clinically ascertained or referred samples, and this may have artificially inflated the posterior probabilities and relative risk estimates obtained. While only the Linkage study purposely ascertained families having three or more generations of affected

individuals, it has been our experience that there is a bias towards multiplex families participating in research of this type in general. Therefore, the risk estimates we provide probably represent the upper limits of the "true" probabilities of affection given knowledge of parental reading ability, since our families may have a higher than average genetic loading, or a priori probability towards having RD offspring. Furthermore, it is important to bear in mind that even minor modifications of the population incidence, P(C), or estimated affection rates in control samples, P(R/Not C), can have large effects on the posterior probabilities obtained. In this report we used the same control sample and P(R/Not C) estimates when calculating P(C/R) for all three data sets. Thus, in a sense, the results across data sets are not completely independent. Separate and appropriately matched and identified control samples for each family study may have altered our posterior probabilities. However, confidence can be placed in using the only Colorado control sample, given that P(R/Not C) estimates approximated those we'd expect given general population base rates.

Relative risk estimates seemed higher than expected, particularly for the Linkage sample, and there was a tendency for the lowest risks to come from the Iowa data set. Recall that polygenic factors have been implicated in RD transmission in the Iowa pedigrees, whereas the Colorado and Linkage data sets show major (dominant or semi-dominant) gene influence according to our segregation analyses (Pennington et al., 1990). Therefore, the lower rates in the Iowa sample are in accordance with expectancy.

In summary, the results presented in this paper indicate that parental affection status has a profound impact on the likelihood that offspring will express a reading disorder. The data also suggest that the probability of being affected may vary in response to genetic heterogeneity, or differing modes of genetic transmission operating within families. There is also some evidence that complex sex effects may be operating that alter the posterior probabilities that a child will have RD. Future work, perhaps with different potential predictors of offspring risk, should involve incorporating the mode of genetic transmission into predictive risk models, as well as the parent and offspring sex. This will of course require a better understanding of the genetics and mechanisms behind RD inheritance and expression than is currently available.

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