

ADHD and Dyscalculia: Evidence for Independent Familial Transmission

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Abstract

The familial relationship between dyscalculia and attention-deficit/hyperactivity disorder (ADHD) was assessed. We conducted a familial risk analysis using probands with and without ADHD of both genders and their first-degree relatives. Participants were assessed with structured diagnostic interviews and a cognitive test battery. We found elevated rates of ADHD in relatives of both ADHD proband groups, regardless of dyscalculia status, and elevated rates of dyscalculia in relatives of probands with dyscalculia, irrespective of ADHD status. There was no evidence for cosegregation or assortative mating. Our findings support the hypothesis that ADHD and dyscalculia are independently transmitted in families and are etiologically distinct. These results reinforce the current nosological approach to these disorders and underscore the need for separate identification and treatment strategies for children with both conditions.

Several studies have documented the co-occurrence of attention-deficit/hyperactivity disorder (ADHD) and learning disabilities (LD) in youth. For example, our group reported a significant association between ADHD and LD in a clinical sample of children with ADHD (Semrud-Clikeman et al., 1992). Others have also found associations between these disorders (August & Garfinkel, 1990; Shaywitz et al., 1995). These results have been subsequently replicated in large samples of psychiatrically and pediatrically referred youth of both genders (Biederman et al., 2002). However, most of the literature on the overlap between LD and ADHD has either aggregated mathematical and reading disabilities or focused solely on reading disabilities. Thus, relatively little is known about the specific relationship between mathematical learning disabilities and ADHD. This lack of empirical investigation on dyscalculia in ADHD is unfortunate, considering the evidence documenting an association between dyscalculia and ADHD in children (Gross-Tsur,

Manor, & Shalev, 1996), a 3% to 6% prevalence of dyscalculia (Shalev, Manor, Amir, Wertman-Elad, & Gross-Tsur, 2000), and its associated impairment (Levine, Lindsay, & Reed, 1992).

Because there is strong evidence that both ADHD (Faraone & Doyle, 2000, 2001) and LD (DeFries, Fulker, & LaBuda, 1987; Smith, Pennington, Kimberling, & Ing, 1990) have genetic components, the familial relationship between ADHD and dyscalculia could shed light on the etiology and neurobiology of both disorders. Thus, the examination of relatives of children with ADHD with and without dyscalculia may provide new insights into the relationship between the two conditions and into their respective neurobiological underpinnings.

In this study, we address the familial relationship between ADHD and dyscalculia in a large sample of families ascertained from referred youth of both genders with and without ADHD. We tested competing hypotheses of familial transmission, as proposed by Pauls, Hurst, et al. (1986; see also Pauls, Towbin, Leckman, Zah-

ner, & Cohen, 1986) and Reich and colleagues (Reich, James, & Morris, 1972; Reich, Rice, Cloninger, Wette, & James, 1979). Based on our previous work, we hypothesized that ADHD and dyscalculia would be independently transmitted.

Method

Participants

Data from two identically designed case-control family studies of ADHD were combined. The first study (Biederman et al., 1992) ascertained families on the basis of a male case (ADHD) or control (no ADHD) proband (i.e., the member of a family selected for the research study) child age 6 to 17 years at time of ascertainment. This study of boys with ADHD included 140 probands with ADHD (with 174 siblings and 280 parents) and 120 control probands without ADHD (with 129 siblings and 239 parents). The second study (Biederman et al., 1999) ascertained families on the basis of a female case or control proband child, also age

6 to 17 years at time of ascertainment. In this study of girls with ADHD, we studied 140 probands with ADHD (with 143 siblings and 274 parents) and 122 control probands without ADHD (with 131 siblings and 238 parents). Written informed consent was obtained for all participants; children provided written assent to participate. Potential probands (either ADHD or control) were excluded if they had been adopted or if their nuclear family was not available for study. We also excluded probands if they had major sensorimotor disabilities (e.g., paralysis, deafness, blindness), psychosis, autism, inadequate command of the English language, or a Full Scale IQ (Wechsler, 1974) below 80. All of the ADHD probands met full diagnostic criteria for ADHD according to clinical assessment following the *Diagnostic and Statistical Manual of Mental Disorders*, revised third edition (*DSM-III-R*; American Psychiatric Association, 1987) at the time of the clinical referral; at the time of recruitment for this study, they all had active symptoms of the disorder.

Two independent sources provided the participating children. We selected psychiatrically referred probands with ADHD from consecutive referrals to a pediatric psychopharmacology clinic at the Massachusetts General Hospital (MGH). Pediatrically referred participants with ADHD included pediatric patients from a Health Maintenance Organization (HMO). Within each setting, we selected controls without ADHD from outpatients at pediatric medical clinics.

A three-stage ascertainment procedure was used to select the participants. For probands with ADHD, the first stage was their referral to a psychiatric or pediatric clinic. The second stage confirmed the diagnosis of ADHD by screening all children with a positive diagnosis at the first stage using a telephone questionnaire completed by the mother. The questionnaire asked about the 14 *DSM-III-R* symptoms of ADHD and included questions regarding study exclusion

criteria. The third stage further confirmed the diagnosis made by the telephone questionnaire with face-to-face structured interviews with the mother. Only probands who received a positive diagnosis at all three stages were included in the final analysis. For control probands, we ascertained participants from referrals to medical clinics for routine physical examinations at both the MGH and HMO sites. In Stage 2, the control mothers responded to the *DSM-III-R* ADHD telephone questionnaire. Eligible controls meeting study entry criteria were recruited for the study and received the third stage diagnostic assessment with a structured interview. Only probands classified as not having ADHD at all three stages were included in the control group.

Diagnostic Measures

Psychiatric assessments of probands and their siblings were made with the *Kiddie Schedule for Affective Disorder and Schizophrenia—Epidemiological Version* (K-SADS-E; Orvaschel & Puig-Antich, 1987). Diagnoses were based on independent interviews with the mothers and direct interviews with the child. For children older than 12, data from direct and indirect interviews were combined by considering a diagnostic criterion positive if it was endorsed in either interview. Children younger than 12 years of age were not interviewed directly. Diagnostic assessments of parents were based on direct interviews with each parent using the *Structured Clinical Interview for DSM-III-R* (SCID; Spitzer, Williams, Gibbon, & First, 1990). To assess childhood diagnoses in the parents, we administered modules from the K-SADS-E covering childhood diagnoses.

The K-SADS-E and the SCID provide phrasing for questions about symptoms and standard probes to determine whether a symptom is severe enough to meet criterion. They are scored according to specified diagnostic algorithms that can produce *DSM-III-R* diagnoses. They examine both lifetime and current (i.e., past month)

diagnoses and determine the number of episodes, the length of the longest episode, and the ages at onset and offset.

All assessments were made by raters who were blind to the child's diagnosis and ascertainment site. Different interviewers met with mothers and children in order to maintain blindness to case-control status and in order to prevent information from one informant influencing the assessment of the other. Diagnoses were considered positive if, based on the interview results, *DSM-III-R* criteria were unequivocally met. All diagnostic uncertainties were resolved by a committee of board-certified child and adult psychiatrists who were blind to the proband's ascertainment group, ascertainment site, all data collected from other family members, and all nondiagnostic data (e.g., cognitive functioning). Diagnoses presented for review were considered positive only if a consensus was achieved that criteria were met to a degree that would be considered clinically meaningful. Kappa coefficients of agreement (Cohen, 1968) were computed between raters and three board-certified psychiatrists who listened to audiotaped interviews made by the raters. Based on 173 interviews, the median kappa was .86; kappa was .99 for ADHD, .80 for multiple anxiety disorders, and .83 for major depression. Socioeconomic status (SES) was assessed with the Hollingshead four-factor scale (Hollingshead, 1975).

Cognitive Assessments

Using the methods of Sattler (1988), we estimated Full Scale IQ from the Vocabulary and Block Design subtests of the *Wechsler Intelligence Scale for Children—Revised* (WISC-R; Wechsler, 1974) for participants younger than 17 and the *Wechsler Adult Intelligence Scale—Revised* (WAIS-R; Wechsler, 1981) for participants older than 17. Our interviewers assessed academic achievement with the Arithmetic subtest of the *Wide Range Achievement Test—Revised* (WRAT-R; Jastak & Jastak, 1985) and the *Gilmore Oral*

Reading Test (Gilmore & Gilmore, 1968). These achievement tests were chosen because they were deemed the most appropriate at the time this study was conceived and conducted, during the mid-to-late 1980s. All of the aforementioned cognitive assessments have been shown to exhibit sound psychometric properties (Gilmore & Gilmore, 1968; Jastak & Jastak, 1985; Wechsler, 1974, 1981).

The definition of learning disabilities under the Individuals with Disabilities Education Act (IDEA) amendments of 1997 requires a significant discrepancy between a child's potential and achievement (Federal Register, 1977). Reynolds (1984) provided a thorough review of measurement issues involved in the definition of LD. We used the procedure recommended by him and others (Frick et al., 1991) as follows. We first converted the estimated Full Scale IQ and achievement scores to the z scores z_{IQ} and z_A . We then estimated the expected achievement score, z_{EA} , by the following regression equation:

$$z_{EA} = r_{IQA} \times z_{IQ}$$

where r_{IQA} is the correlation between the IQ and achievement tests. Then the discrepancy score is $z_{EA} - z_A$ and its standard deviation is $\sqrt{1 - r_{IQA}^2}$. We defined as having LD any participant who had discrepant arithmetic or reading scores based on having a value greater than 1.65 on the standardized discrepancy score:

$$\frac{z_{EA} - z_A}{\sqrt{1 - r_{IQA}^2}}$$

Statistical Analysis

Starting with the full sample of 522 probands, we eliminated from the analysis any probands with reading disabilities ($n = 43$) or with missing data on either LD measure ($n = 15$), leaving 464 probands. We excluded probands with reading disabilities to assess the independent association between ADHD and dyscalculia. We then

stratified the relatives of the probands into four groups based on the probands' ADHD and dyscalculia diagnosis. We created the following groups:

1. relatives of controls without dyscalculia (Control; probands, $n = 216$; relatives, $n = 688$);
2. relatives of controls with dyscalculia (Dyscalculia; probands, $n = 13$; relatives, $n = 42$);
3. relatives of youth with ADHD without dyscalculia (ADHD; probands, $n = 209$; relatives, $n = 658$); and
4. relatives of youth with ADHD with dyscalculia (ADHD + Dyscalculia; probands, $n = 25$; relatives, $n = 84$).

Next, we compared these four groups on demographic factors to identify any potentially confounding factors. Finally, we compared the rates of ADHD and dyscalculia across the four relative groups, adjusting for any confounding factors, to determine which hypothesis about the familial association of the two disorders was best supported by the data.

As stated earlier, we tested competing hypotheses of familial transmission as proposed by Pauls and colleagues (Pauls, Hurst, et al., 1986; Pauls, Towbin, et al., 1986) and Reich et al. (1972; Reich et al., 1979). In describing these hypotheses, the expected differences are relative to normal controls.

Hypothesis 1. If ADHD and dyscalculia are independently transmitted, we would expect to find high rates of ADHD in relatives of the ADHD and the ADHD + Dyscalculia groups and an increased rate of dyscalculia in relatives of the ADHD + Dyscalculia group and the Dyscalculia group.

Hypothesis 2. If ADHD + Dyscalculia is an etiologically distinct subtype of ADHD, we would expect to find high rates of ADHD in relatives of both the ADHD and ADHD + Dyscalculia groups and high rates of dyscalculia in relatives of the ADHD + Dyscalculia and Dyscalculia

lia groups. Furthermore, ADHD and dyscalculia should cosegregate in the relatives of the ADHD + Dyscalculia group. We use the term *cosegregate* to indicate that the disorders are transmitted together in families—that is, the degree of comorbidity in the relatives is greater than expected by chance.

Hypothesis 3. If ADHD and dyscalculia share common familial etiological factors but exhibit variable phenotypic expressivity due to other factors, we would expect to find high rates of both ADHD and dyscalculia in the relatives of the ADHD + Dyscalculia, ADHD, and Dyscalculia groups.

Because we are analyzing parents and offspring, the assumption that each observation is independent of all other observations is violated in these data. To account for this, we used robust estimates of variance so that p values would not be underestimated (Liang & Zeger, 1986). Statistical models were fit with the statistical software package STATA (Stata Corp., 2001). Generalized estimating equation models with the logit link and binomial family specification were used to perform logistic regression models to predict binary outcomes. The statistical significance of each covariate in these regression models was determined by Wald's test, and alpha was set at .05. All tests were two-tailed.

Results

Among the 464 probands included in the analysis, the prevalence of dyscalculia in children with ADHD (11%) was significantly higher than among controls (6%), $\chi^2(1) = 3.9$, $n = 464$, $p = .05$. As shown in Table 1, there was a significant difference in age, with the control group being, on average, 1.3 years older than the ADHD group. Also, significant differences were noted in SES, with the Control group having a lower mean SES score (indicating higher social class) than both the Dyscalculia

TABLE 1
Demographic Characteristics of First-Degree Relatives of Probands With and Without ADHD and Dyscalculia

Demographics	Proband status								χ^2	<i>p</i>
	ADHD + Dyscalculia ^a		ADHD ^b		Dyscalculia ^c		Control ^d			
	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%		
Gender (boys)	46	55	330	50	21	50	349	51	4.08	.25
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	χ^2	<i>p</i>
Age	30.4	14.6	31.3	14.7 ^f	30.3	15.4	32.6	15.1	8.66	.03
SES	1.7	1.0 ^g	1.8	0.9 ^g	2.4	1.0 ^f	1.6	0.7	19.15	< .01

Note. ADHD = attention-deficit/hyperactivity disorder; SES = socioeconomic status (lower values indicate higher social class)
^a*n* = 84. ^b*n* = 658. ^c*n* = 42. ^d*n* = 688. ^fVersus Control, *p* < .05. ^gVersus dyscalculia, *p* < .05.

group and the ADHD group. Furthermore, the Dyscalculia group had a higher mean SES score than both the ADHD and the ADHD + Dyscalculia groups. As the age difference was small and unlikely to confound the assessment of our hypothesis, all subsequent analyses were statistically adjusted for SES.

As shown in Figure 1, we found significantly increased rates of ADHD in the relatives of probands with ADHD with and without dyscalculia compared to the relatives of control probands. Also, there was a significantly increased rate of ADHD in relatives of ADHD + Dyscalculia probands compared to relatives of dyscalculia probands. As displayed in Figure 2, there were significantly higher rates of dyscalculia in the relatives of the ADHD + Dyscalculia and the Dyscalculia groups compared to the relatives of the control probands.

Given the increased rates of ADHD and dyscalculia in the relatives of ADHD + Dyscalculia probands, we also tested the cosegregation of these two disorders in this group. We found no significant difference in the rate of dyscalculia in ADHD + Dyscalculia relatives with and without ADHD (see Figure 3). To further examine the elevated rates of ADHD and dyscalculia in this group, we also tested for the presence of assortative mating—that is, the mat-

ing of persons with one disorder to persons with another disorder more often than expected by chance. In ADHD + Dyscalculia relatives, neither mothers with ADHD (*n* = 3) nor mothers without ADHD (*n* = 19) married men with dyscalculia. Also, the 16 fathers without ADHD were not significantly more likely to have a spouse with dyscalculia (6%) than the 6 fathers with ADHD (17%), *z* = 0.73, *p* = .47.

Discussion

This study used familial risk analysis to evaluate the etiological relationship between ADHD and dyscalculia in a large sample of youth with and without ADHD of both genders. The results revealed elevated rates of ADHD in the relatives of both ADHD proband groups, regardless of dyscalculia status, and elevated rates of dyscalculia in relatives of dyscalculia probands, irrespective of ADHD status. There was no evidence for cosegregation or assortative mating. These results best support the hypothesis that ADHD and dyscalculia are independently transmitted in families.

The other, competing hypotheses evaluated were not supported by the data. The hypothesis that ADHD and dyscalculia represent a familial subtype of ADHD predicts elevated rates

of ADHD in the relatives of ADHD probands regardless of dyscalculia status and elevated rates of dyscalculia in relatives of dyscalculia probands irrespective of ADHD status. This hypothesis also predicts cosegregation in the relatives of ADHD + Dyscalculia probands, indicating a significant association between the two disorders in these relatives. Such a finding would suggest that ADHD and dyscalculia are transmitted together in the same persons through families. We found no evidence of such an association, ruling out this hypothesis.

The hypothesis stating that ADHD and dyscalculia share common familial etiological factors but exhibit variable phenotypic expressivity due to other factors predicts high rates of both ADHD and dyscalculia in the relatives of the ADHD + Dyscalculia, ADHD, and Dyscalculia groups. However, we found no evidence of elevated rates of ADHD in Dyscalculia group relatives and no evidence of increased rates of dyscalculia in relatives of ADHD probands, eliminating this hypothesis.

Our findings of independent transmission of ADHD and dyscalculia are consistent with twin studies that examined ADHD and LD, including reading disability (Gilger, Pennington, & DeFries, 1992) and spelling disability (Stevenson, Pennington, Gilger, DeFries, & Gillis, 1993). They are also con-

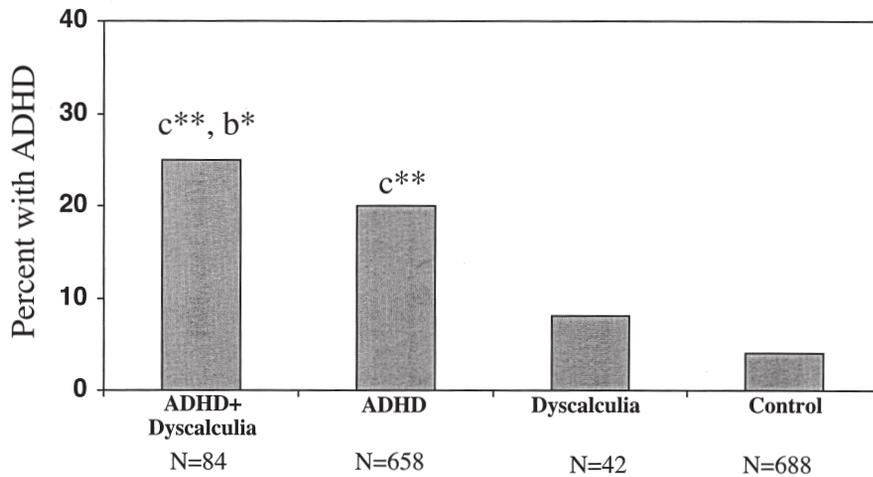


FIGURE 1. Rates of ADHD in the first-degree relatives of ADHD and control probands with and without dyscalculia. *Note.* *N* = number of relatives, ^a = vs. Dyscalculia, ^b = vs. Control; **p* < .05; ***p* < .01.

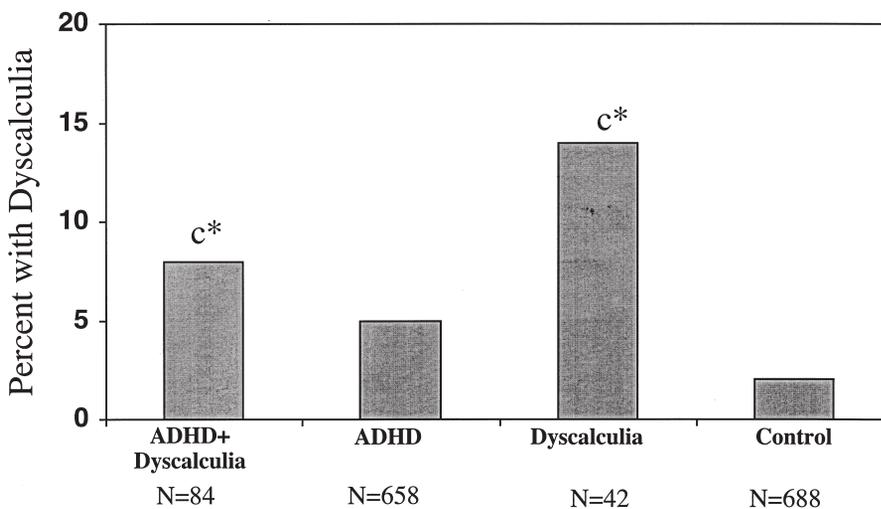


FIGURE 2. Rates of dyscalculia in the first-degree relatives of ADHD and control probands with and without dyscalculia. *Note.* *N* = number of relatives, ^a = vs. Control; **p* < .05; ***p* < .01.

sistent with the findings of another twin study, which found a significant genetic effect for mathematics disability (Alarcon, DeFries, Light, & Pennington, 1997). Other family studies have also found evidence that ADHD and reading or arithmetic disabilities are independent disorders (Faraone et al., 1993; Gilger et al., 1992; Lahey et al., 1988; O'Neill & Douglas, 1991). As probands with reading disabilities

were excluded, this well-powered study provides compelling evidence that dyscalculia by itself is familial and transmitted independently from ADHD. To our knowledge, this specificity of transmission of dyscalculia is a novel finding.

The finding that dyscalculia and ADHD are separate disorders has important clinical implications. Considering that both ADHD and dyscalculia

have significant associated academic dysfunction, children with both disorders are at higher risk for academic failure. The morbidity and disability of ADHD has been well documented (Biederman et al., 1996; Biederman et al., 1999; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993), and dyscalculia has been linked to lower self-esteem, academic motivation (Levine et al., 1992), and academic failure. Whereas ADHD can be treated pharmacologically, dyscalculia requires academic remediation. Furthermore, whereas the assessment of ADHD is clinical, that of dyscalculia is psychometrically defined and requires psychological testing. The diagnosis and treatment of children with ADHD presenting with academic difficulties requires a comprehensive approach, encompassing both psychiatric and psychological components.

The results reported in this study should be considered in light of some methodological limitations. Our sample was predominantly Caucasian and the probands were referred for ADHD. As such, our findings may not generalize to community samples or other ethnic minorities. Future studies should attempt to replicate these findings in community samples and samples ascertained by dyscalculia status. Also, we excluded probands with reading disabilities. Although this approach imposes another limitation on our generalizability, it enhances validity by allowing us to assess the independent association between ADHD and dyscalculia. It is important to note that our results document familiarity and do not necessarily imply genetic influences. Conclusions about the genetic independence of ADHD and dyscalculia await further twin and adoption studies.

Although our definition of dyscalculia was based on measuring discrepancies between IQ and achievement in mathematics, there are other definitions of dyscalculia that we did not assess (Fletcher, Francis, Rourke, Shaywitz, & Shaywitz, 1992; Fletcher et al., 1994). However, it should be noted

that our approach is consistent with both public legal standards (Federal Register, 1977) and other studies in the field (Frick et al., 1991; Reynolds, 1984). In a previous study (Seidman, Biederman, Monuteaux, Doyle, & Earaone, 2001), we found that including low achievement as a method for defining LD (Fletcher et al., 1992; Fletcher et al., 1994) resulted in a small addition of LD cases and did not alter the findings. Also, it is possible that our discrepancy-based method may inappropriately classify participants as having dyscalculia who showed no arithmetic difficulties based on the WRAT-R but who performed particularly well on the IQ measure. However, it should be noted that among the 13 control probands with dyscalculia, the mean WRAT-R Arithmetic score was 80.1 ($SD = 6.6$), 75% had a score below 86, and the highest score was 88. Similarly, among the 25 ADHD probands with dyscalculia, the mean WRAT-R Arithmetic score was 78.5 ($SD = 8.6$), 75% had a score below 85, and the highest score was 91. Considering that all of our dyscalculia probands scored below average in arithmetic achievement and most were at least a full standard deviation below average, our discrepancy-based method of defining dyscalculia did not identify participants who were free from arithmetic difficulties.

Also, our IQ score was based on only two subtests of the WISC-R or WAIS-R (Vocabulary and Block Design) and, thus, could be biased compared to a more comprehensive IQ measurement. However, these two subtests are frequently used for an IQ estimate because their estimate of IQ correlates highly ($r = .90$) with Full Scale IQ based on all subtests (Sattler, 1988). Thus, any bias in our IQ measurement should be minimal and is unlikely to influence our findings. Finally, it is possible that our method of assigning dyscalculia status mistakenly identified participants based on poor test performances attributable to ADHD symptomatology and not to actual dyscalculia, creating false positive dyscalculia diagnoses within the ADHD

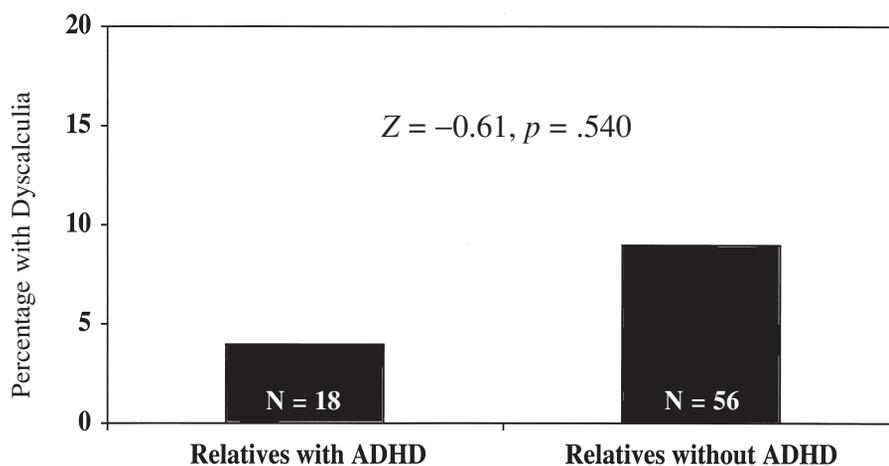


FIGURE 3. Cosegregation of dyscalculia and ADHD in relatives of dyscalculia and ADHD probands.

group. However, several of our findings refute this possibility. First, only a fraction of our probands with ADHD (11%) was classified as having dyscalculia. Second, dyscalculia was elevated in the relatives of probands with ADHD with dyscalculia but not in relatives of probands with ADHD only, despite substantial statistical power to detect an effect in the ADHD group.

Despite these limitations, our findings support the hypothesis that ADHD and dyscalculia are independently transmitted in families, indicating that both disorders are etiologically distinct. No evidence was found to suggest that the comorbid condition is a subtype of ADHD or that the disorders share a common cause and merely exhibit variable phenotypic expressivity. These results reinforce the current nosological (i.e., system of disease classification) approach to these disorders and underscore the need for separate identification and treatment strategies for children with both conditions.

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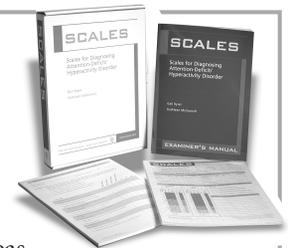
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SCALES

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Gail Ryser and Kathleen McConnell



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