A Twin Study of Mathematics Disability

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Abstract

Although results obtained from recent twin and adoption studies suggest that individual differences in mathematics performance are due in part to heritable influences, no genetic analysis of mathematics disability (MD) has been previously reported. In this article we present data from the first twin sample ascertained for mathematics deficits (40 identical and 23 same-sex fraternal twin pairs in which at least one member had MD). When mathematics performance data from these twin pairs were subjected to a multiple regression analysis, evidence for a significant genetic etiology was obtained. However, tests for the differential etiology of MD as a function of reading performance level were nonsignificant. Results of this first twin study of MD indicate that the condition is significantly heritable, but data from additional twin pairs will be required to test hypotheses of differential etiology more rigorously.

O f the various learning disabilities (LD), only reading disability (RD) has been previously subjected to genetic analysis (DeFries & Alarcón, 1996). Although Kosc (1974) hypothesized that "developmental dyscalculia" has a hereditary, or congenital, basis, the genetic and environmental etiologies of mathematics disability (MD) have not been previously investigated. Nevertheless, results obtained from family, twin, and adoption studies suggest that MD may be heritable. For example, in a review of Cyril Burt's studies of pupils specifically deficient in arithmetic, Barakat (1951) noted that their case histories often included math deficits in other members of the same family, "the number being nearly three times as large as that found in the case histories of ordinary pupils" (p. 154). Results obtained from more recent studies suggest that individual differences in mathematics achievement and performance, within the normal range, are due in part to heritable influences (Gillis & DeFries, 1991; Thompson, Detterman, & Plomin, 1991; Wadsworth, DeFries, Fulker, & Plomin,

1995). Moreover, genetic factors seem to contribute to the observed comorbidity between reading disability and mathematics deficits (Light & DeFries, 1995). Despite these initial inklings, however, the etiology of MD remains unknown. Thus, in a recent comprehensive review of the MD literature, Geary (1993) emphasized the need for behavioral genetic studies: "In comparison with studies of RD, research in the MD area is primitive" (p. 358).

The methodology of behavioral genetics can also be used to assess the validity of subtypes of LD. If different subtypes of MD can be reliably differentiated, different remediation strategies might be developed to target the specific deficits characteristic of each subtype. Three RD–MD combinations have been consistently identified in the LD literature: (a) individuals deficient in reading, spelling, and arithmetic (RSA); (b) those primarily deficient in reading and spelling (RS); and (c) a smaller group primarily deficient in arithmetic (A; Gerber, 1993; Rourke & Del Dotto, 1994; Shafrir & Siegel, 1994). Characteristics of the RS and RSA subtypes include deficits in phonologi-

cal processing, reading, spelling, and short-term memory (Shafrir & Siegel, 1994), with good visual–perceptual and visual–spatial skills (Gerber, 1993). In contrast, individuals with subtype A have adequate verbal– and auditory–perceptual abilities (Gerber, 1993) but perform poorly on visual–spatial, psychomotor, tactile–perceptual, and nonverbal problem-solving tasks (Rourke & Del Dotto, 1994). Thus, at the cognitive level of analysis, the distinction between subtype A and subtypes RS and RSA has been validated by the finding of different cognitive profile. Although there has been little research at the etiological level of analysis on the validity of these subtypes, individuals with subtype A may also have a distinct etiology (Pennington, 1991). The methods used in the present study provide one test of this hypothesis.

A recent twin study of RD (Casto, Pennington, Light, & DeFries, in press) illustrates how the methodology of behavioral genetics can be used to assess the validity of putative subtypes. Twins with RD were classified into three groups: not math disabled, borderline math disabled, and math dis-

abled. Results of a comparison of concordance rates in identical and fraternal twin pairs and regression analyses of reading performance scores indicated that genetic factors may be especially salient as a cause of RD in children with borderline deficits in mathematics; thus, the results of this twin study suggested that mathematics performance may be a valid dimension for diagnosing subtypes of RD.

The purpose of this article was to report results obtained from the first twin study of MD. The etiology of MD was assessed by conducting concordance comparisons in identical and fraternal twin pairs and by fitting the DeFries and Fulker (1985, 1988) basic regression model to mathematics performance data from twins ascertained for math deficits. Although the sample was relatively small, we also conducted a preliminary test of the hypothesis that the etiology of MD differs as a function of reading performance.

Methods

Participants and Measures

Twin pairs tested in the Colorado Learning Disabilities Research Center were systematically ascertained through 27 participating school districts in the state of Colorado (DeFries & Gillis, 1991). All twin pairs in a school were identified by school administrators, and permission was then sought from the parents to review the children's records for evidence of reading and/or mathematics problems. If either member of a twin pair manifested a school history of reading or math problems (e.g., low achievement test scores, etc.), both members of the pair were invited to complete extensive test batteries in laboratories at the University of Colorado and the University of Denver.

At the Institute for Behavioral Genetics, the twins were administered a psychometric test battery that included the Wechsler Intelligence Scale for Children–Revised (Wechsler, 1974) or the Wechsler Adult Intelligence Scale–Revised (Wechsler, 1981), the

Arithmetic subtest of the Wide Range Achievement Test (WRAT; Jastak & Wilkinson, 1984), and the Peabody Individual Achievement Test (PIAT; Dunn & Markwardt, 1970). For the present study, the WRAT Arithmetic subtest and the four PIAT subtests (Reading Recognition, Reading Comprehension, Spelling and Math) were age-adjusted using regression deviation scores, and then standardized using the mean and standard deviation for each measure in a control sample of twins having no school history of reading or math problems. A composite mathematics performance score (MATH) was computed by summing each individual's standardized PIAT Mathematics and WRAT Arithmetic scores. The PIAT Mathematics subtest consists of 84 multiple-choice items with four possible responses for each item. The tester reads an item aloud while the participant observes pictures, numbers, or equations on a card. Item difficulty ranges from simple matching to complex trigonometry and geometry. The WRAT Arithmetic subtest consists of an oral section (counting, numbers, oral problem solving) and a written section with increasingly difficult computations. Level 1 (ages 5 to 11) consists of 15 oral problems and 44 written computations to be completed in 10 minutes. Level 2 (ages 12 and older) consists of 10 oral problems and 56 written computations to be completed in 10 minutes. A reading composite score (READ) was also computed for each twin by employing discriminant weights estimated from an analysis of PIAT Reading Recognition, Reading Comprehension, and Spelling data obtained from an independent sample of 140 nontwin children with RD and 140 nontwin control children (DeFries, Olson, Pennington, & Smith, 1991).

The MD sample included twin pairs in which at least one member had a school history of math problems. In addition, for a twin to be considered an MD proband, he or she had to meet the following criteria: a standardized MATH score of at least 1.5 standard deviations below the mean of the con-

trol sample; a verbal or performance IQ score of at least 90; no evidence of serious neurological, emotional, or behavioral problems; and no uncorrected deficits in visual or auditory acuity. The control sample included twin pairs in which neither member had a school history of math or reading problems; neither had known neurological, emotional, or behavioral problems; and neither had uncorrected deficits in visual or auditory acuity.

Zygosity was determined for same-sex twin pairs using selected items from the Nichols and Bilbro (1966) zygosity questionnaire, which has a reported accuracy of 95%. In cases in which zygosity was doubtful, blood samples were analyzed. Seventy percent of the families of twins with MD were White, 13% were Hispanic, 8% were African American, and 6% were Native American. English was the primary language spoken in the homes.

As of May 31, 1995, the MD sample included a total of 40 pairs of identical (monozygotic [MZ]) twins (19 male, 21 female), and 23 pairs of same-sex fraternal (dizygotic [DZ]) twins (11 male, 12 female). In this sample of 63 twin pairs, 95 children (63 MZ and 32 DZ) met the MD proband criteria. The control sample included 167 MZ twin pairs (75 male, 92 female) and 109 same-sex DZ pairs (60 male, 49 female). At the time of testing, the twins were between the ages of 8 and 20 years, with the MD and control samples having mean ages of 11.54 and 11.87 years, respectively.

In an initial attempt to assess the possible differential etiology of MD as a function of reading performance, the twins with MD were classified into two subtypes: AR (children who had both MD and RD) and A (MD only). If the MD proband also had a school history of reading problems and a READ score in the affected range, the twin pair was included in the AR group. However, if the MD proband had no school history of reading problems and/or a READ score in the nonaffected range, the twin pair was included in the A group. Of the 95 MD probands, 55 were of subtype AR

and 40 were of subtype A. The mean verbal, performance, and full scale IQ scores of the AR probands were 89.5, 96.7, and 92.1, respectively, and the corresponding means for the A probands were 97.0, 100.1, and 98.0, respectively.

Analyses

Members of MZ twin pairs are genetically identical, whereas DZ twins share about one half of their segregating genes, on average. Thus, a comparison of concordance rates in MZ and DZ twin pairs provides a simple, but relatively weak, test for genetic etiology. Because truncate selection was used to ascertain the sample in the present study (i.e., either member of the pair could be a proband), twin pairs concordant for math disability were double-entered to estimate proband-wise concordance (DeFries & Gillis, 1991).

In contrast to a comparison of concordance rates, a multiple regression analysis of selected twin data provides a more versatile and statistically more powerful test for a genetic etiology (DeFries & Fulker, 1985, 1988). When probands are selected for low test scores on a continuous variable, MZ and DZ co-twin scores should both regress, on average, toward the unselected population mean. But, to the extent that the condition is heritable, MZ co-twin means should regress less than DZ co-twin means (DeFries & Fulker, 1985; DeFries, Fulker, & LaBuda, 1987). Therefore, when MZ and DZ proband means are equal, a simple t test between the MZ and DZ co-twin means could be employed as a test of genetic etiology. However, because a multiple regression analysis of selected twin data (DeFries & Fulker, 1985, 1988) is more general, the twin data were also subjected to DF analysis (DeFries & Alarcón, 1996).

The following basic regression model was first fitted to the MATH data:

$$C = B_1 P + B_2 R + \alpha, \qquad (1)$$

where C is the co-twin's MATH score, P is the proband's MATH score, R is the coefficient of relationship (1.0 for MZ twins and 0.5 for DZ twins), and α is the intercept. In this equation, B_1 symbolizes the partial regression of the co-twin's score on the proband's score, and estimates average MZ–DZ twin resemblance. B_2, the partial regression of the co-twin's score on the coefficient of relationship, equals twice the difference between the MZ and DZ co-twin means after covariance adjustment for any difference between the MZ and DZ proband scores. Therefore, B_2 provides a direct test of genetic etiology. With a simple transformation of the data (i.e., each score is expressed as a deviation from the control mean and then divided by the difference between the proband and control means) prior to this analysis (DeFries & Fulker, 1988), B_2 estimates the extent to which proband math deficits can be attributed to genetic factors (h_g^2). As noted, because the present sample was assessed via truncate selection, the data from concordant pairs were double-entered in a manner analogous to that used to calculate proband-wise concordance. Resulting standard errors and significance tests were adjusted accordingly. In subsequent analyses, verbal, performance, and full scale IQs were also added as covariates.

To determine if h_g^2 differed as a function of MD subtype (either AR or A), the basic model was extended to include a main effect and two interactions involving subtype:

$$C= B_1 P + B_2 R + B_3 S + B_4 PS + B_5 RS + \alpha, \qquad (2)$$

where S is the proband's subtype (dummy coded −0.5 for the AR probands and +0.5 for the A probands). The B_4 and B_5 coefficients provide tests of significance for differential twin resemblance and differential h_g^2 as a function of the proband's subtype, respectively. We also conducted a test of differential h_g^2 for MD as a function of reading performance, where S was

the proband's reading score (READ), that is a continuous measure.

Results

As shown in Table 1, 58% of MZ co-twins and 39% of DZ co-twins of MD probands (both AR and A) also had MD, resulting in corresponding proband-wise concordance rates of 0.73 and 0.56, respectively. Although the concordance rate for MZ twins exceeded that for DZ pairs, the difference was not significant ($z = 1.30$, $p = 0.40$) in this small sample. For the twins of subtype A with MD, the MZ and DZ proband-wise concordance rates were also consistent with the hypothesis of a genetic etiology, viz., 0.85 and 0.46, respectively. However, this difference is also nonsignificant ($p = 0.08$).

Distributions of the standardized MATH scores for probands, MZ co-twins, and DZ co-twins are presented in Figure 1. As predicted by the genetic etiology hypothesis, the MZ and DZ co-twin scores regressed differentially toward the mean of the control sample. The mean MATH scores of the MD probands and co-twins (standardized against the mean of 552 control twins) are presented in Table 2. Although the MZ and DZ proband MATH means were very similiar (over 2 standard deviations below the control twin mean), the MZ co-twins regressed only 0.09 standard deviation units, on average, toward the unselected population mean, whereas the DZ co-twins regressed an average of 0.56 standard deviation units. When the proband and co-twin transformed MATH scores were fitted to the basic regression model (Equation 1), $B_2 = h_g^2 = 0.38 \pm 0.18$ ($p = 0.02$, one-tailed), suggesting that almost 40% of the average proband math deficit was due to heritable factors (see Table 3). To assess whether this significant h_g^2 estimate was due to the correlation between MATH and IQ, an extension of the basic model was fitted to the data in which verbal, performance, and full scale IQs were included separately as

TABLE 1
Concordance for Mathematics Disability (MD) in Co-Twins of MD Probands

	Co-twin		
MD proband	Concordant	Discordant	Proband-wise concordance
MZ	23	17	.73
DZ	9	14	.56

TABLE 2
Mean Mathematics Performance Scores

		Standardized[a]		Transformed[b]	
Zygosity	N_{prs}^c	Proband	Co-twin	Proband	Co-twin
MZ	63	−2.54 ± 0.73	−.245 ± 0.85	1.00 ± 0.29	0.96 ± 0.34
DZ	32	−2.44 ± 0.68	−1.88 ± 1.08	1.00 ± 0.28	0.77 ± 0.44

[a]The MATH scores were standardized on the basis of the mean and standard deviation of a sample of 552 controls. [b]The MATH scores were transformed by expressing each score as a deviation from the control MATH mean and then dividing by the difference between the proband and control MATH means. [c]Number of twin pairs following double entry of pairs concordant for MD.

TABLE 3
Fit of Basic Regression Model to Transformed Proband and Co-Twin MATH Scores

Coefficient	Estimate ± SE	t	p
B_1	0.60 ± 0.15	3.98	< 0.001
$B_2 = H_g^2$	0.38 ± 0.18	2.13	.017

Note. B_1 = average MZ–DZ twin resemblance; B_2 = twice the difference between MZ and DZ co-twin means after covariance adjustment for any difference between the MZ and DZ proband scores.

covariates. Resulting estimates of h_g^2 were 0.39 ± 0.18, 0.40 ± 0.18, and 0.41 ± 0.18, respectively, each of which was also significant (p = 0.04, 0.03, and 0.02).

To explore the possible differential etiology of the putative MD subtypes (AR vs. A), mean standardized MATH scores were computed for the probands and co-twins in each group. As shown in Table 4, the average MATH scores for the MZ and DZ probands were similar within each subtype, and in all cases the mean scores of the probands were more than 2 standard deviations below the mean of the unselected population. Moreover, for both subtypes, MZ co-twin MATH scores regressed less toward the control mean than those of DZ co-twins.

The basic regression model (Equation 1) was then fitted to the proband and co-twin transformed MATH scores of each subtype, separately. As shown in Table 5, the resulting h_g^2 estimates for the AR and A subtypes were 0.41 ± 0.21 and 0.32 ± 0.37, respectively, suggesting that math deficits of individuals with the AR subtype may be somewhat more heritable than those of individuals with the A subtype.

To test the significance of the difference between the two h_g^2 estimates, an extension of the DF basic regression model (Equation 2) was fitted to the MATH data of both MD subtypes (AR and A) simultaneously. As expected due to the small sample size, the test for differential h_g^2 was not significant (B_5 = −0.09 ± 0.38, p = 0.82). When the extended model was fitted to MATH data using the proband's composite READ score as the covariate (S), the test for differential h_g^2 was somewhat more powerful, but also nonsignificant (p = 0.65).

Discussion

In the present study, the etiology of mathematics disability was assessed by fitting the basic regression model for the analysis of selected twin data (DeFries & Fulker, 1985, 1988) to mathematics performance data from MD twin pairs tested in the Colorado Learning Disabilities Research Center. The sample included 63 pairs of twins in which at least one member of each had MD; to the best of our knowledge, this was the first sample of twin pairs ascertained for mathematics deficits. Although this sample was relatively small, results of the DF analysis yielded a significant h_g^2 estimate (0.38, p = 0.02), thus providing evidence for the genetic etiology of MD. The h_g^2 estimate was also significant when we controlled for differences in verbal, performance, and full scale IQ.

Although our h_g^2 estimate was significant, it was substantially less than 1. Thus, environmental factors also contribute importantly to proband math deficits. This notion is consistent with previous studies showing that mathematics performance encompasses a variety of skills that are learned sequentially. For example, addition/subtraction, multiplication/division, algebra, and geometry are taught during different developmental stages, and performance on these tasks may be affected by environmental influences shared by family members. In a review of mathematics achievement in the United States, the National Center for Educa-

tion Statistics (1991) reported that "students in homes with resource materials such as newspapers, magazines, and books had higher average mathematics proficiency, as did students who read more pages each day for school and homework" (p. 14). As would be expected, children's success in mathematics is also influenced by the instruction they receive in school (Kameenui & Griffin, 1989). According to Newman and Stevenson (1989), students' performance on math tests, compared with reading tests, is more strongly linked to whether they receive sufficient help from the teacher. Thus, in addition to heredity, environmental factors, such as teaching protocol and parental support in academics, also substantially influence mathematics performance.

Because different forms of intervention are more suitable for some patterns of cognitive assets and deficits than others, it is important to validate ostensible LD subtypes (Rourke & Del Dotto, 1994). To assess the validity of MD subtypes that may have distinct etiologies, we conducted preliminary analyses of math deficits in probands with different levels of reading performance. The h_g^2 estimate for mathematics performance in the AR (comorbid MD and RD) subtype was significant ($p = 0.02$), whereas that for the MD sample without substantial reading deficits (A) was not ($p = 0.20$). However, this result may have been due, at least in part, to the small sample of A probands ($n = 40$). Although the number of twins with MD in the present study was clearly inadequate, we nonetheless tested the hypothesis that the etiology of math deficits differs as a function of the proband's reading performance by fitting an extension of the basic regression model to data from the two putative MD subtypes simultaneously. As expected due to the small sample size, the test for the differential genetic etiology of MD as a function of reading performance was not significant.

MD was diagnosed in the present study using only school history infor-
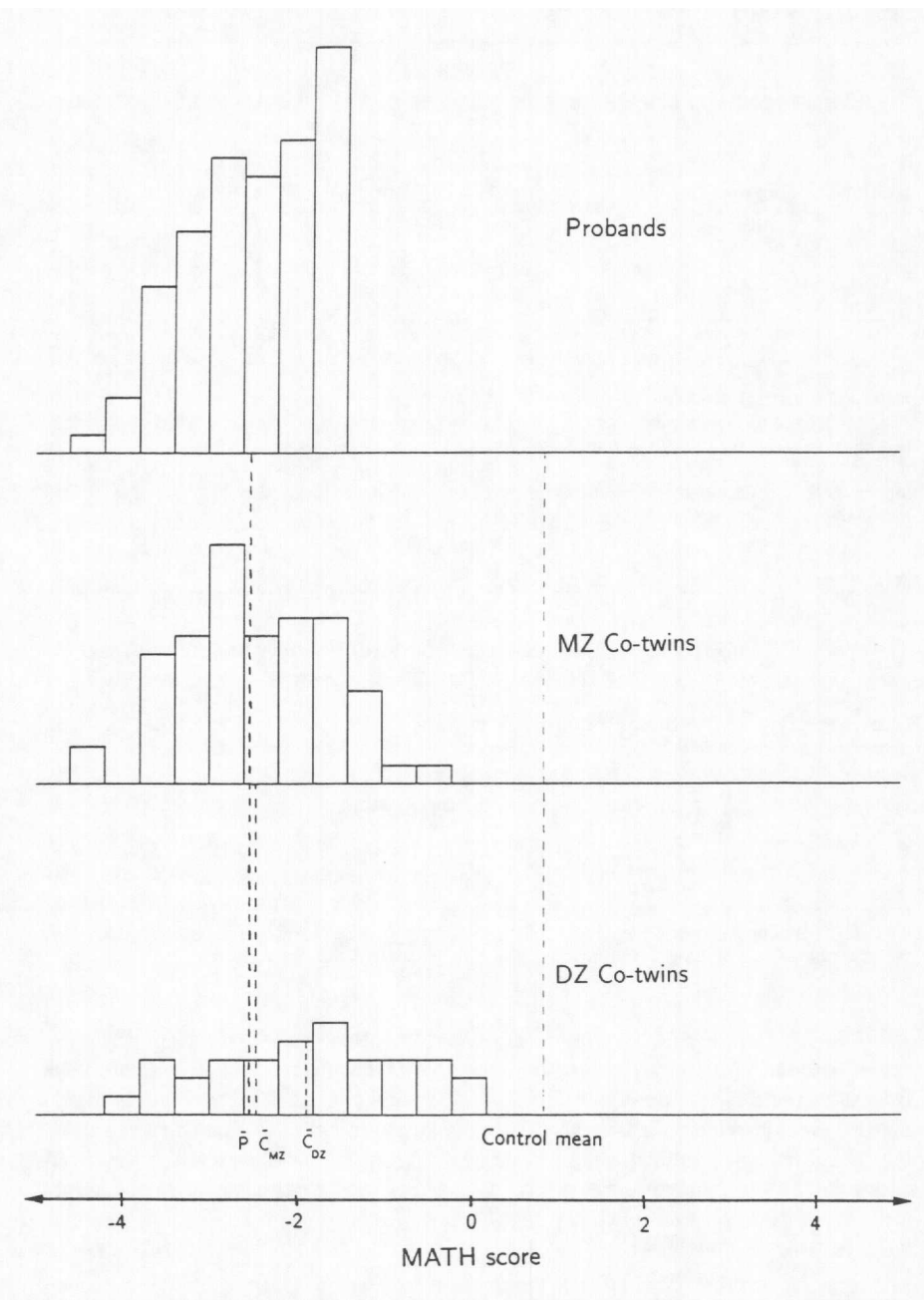
FIGURE 1. Distributions of mathematics performance scores for probands with math disability, MZ co-twins, and DZ co-twins tested in the Colorado Learning Disabilities Research Center.

mation and a composite math performance score based on two psychometric test scores (WRAT and PIAT arithmetic subtests). Moreover, although the results of this first twin study of MD provide evidence for a genetic etiology, the sample is too small for more complex DF analyses (cf. DeFries & Alarcón, in press). Thus, data from additional twin pairs with

MD will be required to test hypotheses of subtype validity more rigorously.

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TABLE 4
Mean Mathematics Performance Scores of MD Twin Pairs by Subtype

Subtype[c]	Zygosity	N[d]	Standardized[a]		Transformed[b]	
			Proband	Co-twin	Proband	Co-twin
AR	MZ	36	−2.62 ± 0.76	−2.45 ± 0.98	1.00 ± 0.29	0.93 ± 0.38
	DZ	19	−2.69 ± 0.71	−1.97 ± 1.04	1.00 ± 0.27	0.73 ± 0.39
A	MZ	27	−2.43 ± 0.69	−2.45 ± 0.67	1.00 ± 0.28	1.01 ± 0.27
	DZ	13	−2.08 ± 0.46	−1.76 ± 1.16	1.00 ± 0.22	0.85 ± 0.56

Note. MD = math disability.
[a]The MATH scores were standardized on the basis of the mean and standard deviation of a sample of 552 controls. [b]The MATH scores were transformed by expressing each score as a deviation from the control MATH mean and then dividing by the difference between the proband and control MATH means. [c]AR symbolizes MD probands with RD, whereas A indicates MD only. [d]Number of twin pairs following double entry of pairs concordant for MD.

TABLE 5
Fit of Basic Regression Model to Proband and Co-Twin Transformed MATH Scores for MD Subtypes

Subtype[a]	Coefficient	Estimate ± SE	t	p
AR	B_1	0.77 ± 0.18	4.37	< .00005
	$B_2 = h_g^2$	0.41 ± 0.21	1.97	.024
A	B_1	0.34 ± 0.34	1.01	< .200
	$B_2 = h_g^2$	0.32 ± 0.37	0.86	.200

Note. MD = math disability.
[a]AR symbolizes MD proband with a reading disability, whereas A indicates MD only.

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AUTHORS' NOTES

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