THE HERITABILITY OF LANGUAGE: A REVIEW AND METAANALYSIS OF TWIN, ADOPTION, AND LINKAGE STUDIES

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Some researchers argue that the ability to acquire and use language is largely the result of innate predispositions that are specific to language (the INNATENESS HYPOTHESIS). If the innateness hypothesis is correct, these predispositions must be encoded for in our DNA. This article reviews more than one hundred genetic studies of language. The results of these studies strongly suggest that genetic factors play a role in the variation in the rate of language acquisition and linguistic proficiency attained by children and adults. Genetic factors account for much of the variance in linguistic abilities among people with written or spoken language disorders and some of the variance in linguistic abilities among normal people. In addition to heritable factors that influence both nonverbal and verbal abilities, there appear to be genetic factors that specifically influence linguistic abilities. Furthermore, some studies suggest that different genetic factors are involved in different aspects of language (e.g. written language vs. spoken language; lexical vs. syntactic abilities).*

This article reviews the results of genetic studies that investigate the extent to which heritable factors play a role in the acquisition and use of language. The key questions that will be addressed are

1. Do heritable factors affect people's ability to acquire and use language?
2. Are heritable factors responsible for (some of) the variation in linguistic abilities observed among 'normal' people, or do heritable factors only account for the variance observed for people diagnosed with language disorders?
3. Are there heritable factors that are specific to linguistic abilities?
4. If genetic factors that are specific to language do exist, do such factors play a role in all aspects of language or just some?
5. If genetic factors play a role in multiple aspects of language, are different factors involved in different aspects of language?

1. INNATENESS AND THE HERITABILITY OF LANGUAGE. To what extent is the ability to acquire and use language the result of innate predispositions that are specific to language (henceforth, the INNATENESS HYPOTHESIS)? Many different types of evidence suggest that language is due (in part) to innate cognitive and neural processes. For example, supporters of the innateness hypothesis point out that human languages share certain universal properties (linguistic arguments), that, even in the absence of negative evidence, children acquire language very quickly and with relatively few errors (learnability arguments), that functional neuroimaging and lesion-deficit correlational studies implicate left perisylvian cortical brain for certain language tasks (neuroanatomical arguments), and that some acquired and developmental disorders preferentially impair or spare language (modularity arguments). Although these data are generally consistent

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with the innateness hypothesis, the data are not all as clean as is sometimes presented. Space does not permit a review of arguments for and against these types of data, but interested readers may want to consult Pinker 1994 and Stromswold 2000 for a pro-innatist perspective of these data and Müeller 1996 and Karmiloff-Smith and colleagues (Elman et al. 1996, Karmiloff-Smith 1991, Karmiloff-Smith & Karmiloff 2001) for a more skeptical view.

Genetic studies of language are another way of investigating the innateness hypothesis. If the innateness hypothesis is correct, the cognitive and neural predispositions that enable us to acquire and use language must be encoded for in our DNA. Thus, if studies reveal that genetic factors play a role in people's linguistic abilities, this supports the innateness hypothesis. But there are at least two reasons why genetic studies might fail to reveal evidence of the heritability of language. One possibility is that the innateness hypothesis is incorrect, and environmental factors determine how facile people are at acquiring and using language. The other possibility is that language *is* the result of our genetic endowment, but everyone has exactly the same genetic endowment when it comes to language. Here's how the second possibility works: for a genetic study to reveal that a trait is heritable, genetic factors must account for some of the variation observed among individuals for that trait. If individuals do not differ genetically with respect to a given trait, then heritability estimates for that trait will be zero even if the trait is completely the result of what is in our DNA. In other words, language may be universal to all people in the same way that all (normal) people have exactly ten fingers: both are the result of people's genetic endowment, but genetic factors account for none of the variance in people's linguistic abilities or number of fingers (see e.g. Chomsky 1980). Alternatively, linguistic ability may be more like height than it is like the number of fingers people have. As Lieberman (1984, 1991, 1994) has argued, it is possible that, even if language is a genetically encoded, species-universal trait, some people's genetic endowments might predispose them to be more linguistically adept than others, in the same way that some people are genetically predisposed to be taller than others, even though all people have height. (For evolutionary arguments on why this would be so, see Tooby & Cosmides 1990.)

Some studies suggest that normal people vary in their ability to acquire and use language. Although the course of acquisition is remarkably similar for all children acquiring a given language (for a summary, see Stromswold 2000), some children learn language more rapidly than others. For example, some normal twenty-four-month-old children say only a handful of words, whereas others say over 300 words (e.g. Fenson et al. 1994, Goldfield and Reznick 1990). Brown (1973) and Cazden (1968) investigated when three English-speaking children acquired fourteen grammatical morphemes, and found that, although all three children acquired these morphemes in the same order, the children acquired them at different ages. For example, all three children mastered the third person singular verbal inflection *-s* after they had mastered the homophonous plural and possessive morphemes, but one child mastered the *-s* verbal inflection at two years, three months (2;3), whereas the other two children did not achieve mastery until ages 3;6 and 3;8. Similar individual differences in rates of acquisition have been reported for questions (Stromswold 1988, 1995), auxiliaries (Stromswold 1989, 1990), and datives, verbal particles and related constructions (Snyder & Stromswold 1997, Stromswold & Snyder 1995). In addition, some studies suggest that, although all normal adults have a basic level of linguistic competence in their native language, some adults are more linguistically adept than others. For example, individual differences have been reported in verbal fluency (Day 1979), in the interpretation of novel compound nouns

(Gleitman & Gleitman 1970), in sentence processing (Bever et al. 1989, Just & Carpenter 1992), and in the ability to acquire a second language as a school-aged child (Masoura & Gathercole 1999) or as an adult (Fillmore 1979). Even among native speakers of the same dialect, there are individual differences in grammaticality judgments (Cowart 1997, Nagata 1992, Ross 1979, Schütze 1996). We can't tell whether these individual differences in linguistic ability are the result of environmental and/or genetic factors, but the fact that such differences exist suggests that people may differ in their genetic endowments for language.

Although, as a species, people have exactly ten fingers and genetic factors do not account for any of the variance in the number of fingers normal people have (people may have fewer than ten fingers because of environmental factors such as accidents or prenatal exposure to thalidomide), genetic syndromes such as Alpert syndrome can cause people to have fewer than ten fingers (oligodactyly) and genetic syndromes such as Laurence-Moon-Biedl syndrome can cause people to have more than ten fingers (polydactyly). Similarly, even if genetic factors do not account for any of the variance in linguistic abilities observed for normal individuals, there might be genetic syndromes that result in language disorders (see Jung 1989 for a description of genetic syndromes that affect language). For this reason, in the review that follows, genetic studies are divided according to whether they investigate the heritability of language disorders (such studies are reported primarily in §§3, 6, 13 and 14) or normal language abilities (such studies are reported primarily in §§7, 8, 9, 10, and 12).

2. Method

2.1. Locating studies. In the summer of 1994, the computerized bibliographic databases PsycINFO, ERIC, and Medline were searched for papers on the genetics of language. Boolean searches were performed to cull any paper, chapter, or book written in English that contained the letter strings *language*, *linguistic*, *articul*, or *speech*, and the letter strings *hereditary*, *genetic*, *famil*, *twin*, *adoption*, *chromosom*, *linkage*, *pedigree*, *sex-ratio*, *segregation*, *aggregation*, *DNA*, or *RNA* in its journal title, article title, key words, or abstract. In the spring of 2001, this procedure was repeated for just the PsycINFO and Medline databases. To find recent studies that had yet to appear in these databases, in June 2001, targeted searches were done of the Web of Science and the World Wide Web (using the search engine Google). The strings searched for were the names of authors who had previously published genetic studies of language, and conventional abbreviations for chromosomal regions that have been implicated in language disorders (e.g. 15q21). The results of the searches were examined by hand and all appropriate papers were obtained. Only twin, adoption, and linkage studies of language are comprehensively reviewed here. (For review of other types of genetic studies, see Stromswold 1998.) Because my purpose is to review the evidence for genetic factors specific to language, studies that investigated progressive or acquired language disorders, or language impairments that are secondary to hearing impairment, motor dysfunction, frank neurological disorders, psychiatric disorders, or mental retardation were excluded. Studies of stuttering were also omitted. (For discussion of the genetics of stuttering, see Felsenfeld et al. 2000, Yairi et al. 1996). The reference sections of all articles deemed appropriate for inclusion were searched for additional studies not included in the computerized bibliographic databases. In addition to the studies obtained using the method just described, a few papers that were written or published after the computerized search were included if they met the criteria described above.

2.2. Studies included. Because of the scarcity of genetic studies of spoken language and the growing consensus that reading and spelling disabilities are often manifestations

of a more pervasive language impairment (see §9.1), genetic studies of written language were included in this review. Many papers report interim results of ongoing studies and, subsequently, the same subjects appear in many papers. If the only difference between an earlier and a later paper was that the results for more subjects were reported in the later paper (e.g. Plomin et al. 1993, Reznick et al. 1997), the results of the earlier paper were not included in the review.

It is generally believed that members of same-sex twin pairs are more likely to be similar to one another than members of opposite-sex twin pairs. (But, for evidence that this is not always the case, see Dale et al. 1998, Eley et al. 1999.) Therefore, because identical twins are necessarily the same sex, whereas fraternal twins may be opposite sex, if one does not exclude opposite-sex fraternal twin pairs, heritability estimates may be inflated. Thus, in the studies reviewed, data for opposite-sex fraternal twin pairs were excluded, as were data from studies that did not distinguish between same-sex and opposite-sex fraternal twins (e.g. Levi & Bernabei 1976). Because written and spoken language impairments are more likely to be diagnosed in boys (see Shaywitz et al. 1990, Stromswold 1998), if the sex ratios for identical and fraternal twin pairs in a study are very different, this could affect heritability estimates. If a higher percentage of identical than fraternal twin pairs are male, this will tend to inflate heritability estimates for language disorders, whereas if a higher percentage of identical than fraternal twin pairs are female, this could reduce heritability estimates for language disorders. Furthermore, if twin studies of normal language use and development inadvertently include some impaired twins (as might occur, for example, if language disability just represents the low end of the continuum of linguistic ability), different sex ratios for identical and fraternal twins could skew heritability estimates in twin studies of normal language. For this reason, the sex ratios for identical and fraternal twin pairs are given for each study reviewed. Studies were not excluded, however, if they did not provide the sex breakdown for twins.[1] Over one hundred papers met the inclusionary and exclusionary criteria outlined above. Of these, about 60% were twin studies, 30% were genetic linkage studies, and 10% were adoption studies.

2.3. ASCERTAINMENT OF SUBJECTS. In the studies reviewed, individuals with spoken or written language disorders were located by reviewing clinic, school, medical, or twin-study records, by sending questionnaires to clinicians and participants in twin studies and twin clubs, by placing advertisements in local papers and twin publications, and by testing large cohorts of people and identifying those who performed poorly. Nonimpaired subjects were located through birth and school records, twin clubs, newspaper advertisements, messages posted to computer newsgroups for twins, and referrals from various agencies.

3. FAMILY AGGREGATION STUDIES OF SPOKEN LANGUAGE DISORDERS. If developmental language disorders are heritable, then the incidence of language disorders should be greater among relatives of people with language impairment (probands) than among relatives of people without language impairments (controls): more probands than controls should have a positive family history for language impairments (i.e. more probands should have at least one language-impaired relative) and a higher percentage of probands' relatives than controls' relatives should have a history of language impairment. Stromswold (1998) reviewed eighteen family aggregation studies of spoken language

[1] As is standard practice in behavioral genetic studies, most of the studies partialled out effects of age and sex before performing further analyses.

impairment (SLI). The incidence of positive family history was significantly greater for probands than controls in all seven studies that collected data for both probands and controls. In these seven studies, the reported incidence of positive family history for probands ranged from 24% to 78% (mean 46%, median 35%). For controls, positive family history rates ranged from 3% to 46% (mean 18%, median 11%). Of the studies reviewed, eleven reported the percentage of probands' relatives who were impaired. For probands, the percentage of family members who were impaired ranged from 20% to 42% (mean 28%, median 26%). For controls, the percentage of family members who were impaired ranged from 3% to 19% (mean 9%, median 7%).[2] The incidence of impairment was significantly higher among proband relatives than among control relatives in seven of the eight studies that made such a comparison.

Although familial aggregation data are consistent with some spoken language disorders having a genetic component, it is possible that children with language-impaired parents or siblings are more likely to be linguistically impaired themselves because they are exposed to deviant language (the DEVIANT LINGUISTIC ENVIRONMENT HYPOTHESIS, henceforth, DLEH). The DLEH is consistent with studies that report that parents are more likely to use simple, directive speech when talking to their language-impaired children than parents talking to normal children (Cramblit & Siegel 1977). However, as Conti-Ramsden and colleagues (Conti-Ramsden 1990, Conti-Ramsden & Friel-Patti 1984, Conti-Ramsden & Dykins 1991) point out, children's language impairments may cause parents to use simplified speech, rather than vice versa. In other words, parents may use simple, directive speech because they cannot understand their impaired children and the children do not understand them if they use more complicated language.

The DLEH makes a number of predictions that are not borne out (for more details, see Stromswold 1998). Contrary to the DLEH prediction that the most severely impaired children should come from families whose members have severe language impairments, Byrne et al. (1974) found that children with profound language impairments were less likely to have positive family histories of language impairment than children with moderate language impairments, and Tallal et al. (1991) found no differences in the language abilities of children who did versus those who did not have a positive family history of language disorders. According to the DLEH, the deficits exhibited by language-impaired children are the result of the children 'copying' the ungrammatical language of their parents. Thus, the DLEH predicts that language-impaired children should have the same type of impairment as their relatives. But Neils and Aram (1986) found that 38% of parents with a history of a speech and language disorder said that the disorder they had was different from their children's disorder. According to the DLEH, parents with a history of spoken language impairment who are no longer impaired should be no more likely to have language-impaired children than parents with no such history. Contrary to this prediction, Neils and Aram (1986) found that a third of the probands' parents who had a history of a spoken language disorder no longer suffered from the disorder as an adult. Contrary to the DLEH prediction that all children with SLI should have at least one close relative with a language impairment, in the studies reviewed in Stromswold 1998, over half of the language-impaired children had

[2] The reason the rates of positive family history and impaired family members are high for controls is that some studies were quite lenient about what counted as evidence of a language impairment in family members. For example, Tallal et al. (1989a) counted as affected any family member who had a history of speech or language disorder (excluding isolated articulation disorder), dyslexia, learning disability, or poor school performance (having to repeat a grade).

no first degree relatives with impairments. Although the DLEH predicts that birth order might affect how likely a child is to exhibit a language disorder, birth order does not affect the severity or likelihood of developing language disorders (e.g. Tomblin et al. 1991). In our society, mothers typically have the primary responsibility for child-rearing, and the DLEH predicts that the correlation of language status should be greatest between mother and child. But Tomblin (1989) found that among the family relations he studied (mother-child, father-child, male sibling-child, female sibling-child), the relationship was weakest between mother and child. In addition, Tomblin and Buckwalter (1994) found a ratio of impaired fathers to impaired mothers of 2.7:1, Neils and Aram (1986) found a ratio of 1.4:1, and Tallal et al. (1989b), Whitehurst et al. (1991), and Lewis (1992) all found the ratio was approximately 1:1.

4. QUANTIFYING THE ROLE OF GENETIC FACTORS IN LANGUAGE.

4.1. OVERVIEW OF TWIN METHODOLOGIES. The results of family aggregation studies suggest that genetic factors play a role in many cases of spoken language disorders. Unfortunately, such studies do not allow one to quantify the extent to which genetic and environmental factors play a role in spoken language disorders. One way of teasing apart the strands of environmental and genetic factors is to determine whether identical (monozygotic, MZ) twins are more similar to one another than are fraternal (dizygotic, DZ) twins. While MZ and DZ twins share essentially the same pre- and postnatal environment, MZ twins share 100% of their genetic material, whereas, on average, DZ twins share only 50% of their genetic material.[3] Therefore, if MZ twin pairs' linguistic abilities are more similar than those of DZ twin pairs, this suggests that genetic factors play a role in language. But if MZ twin pairs are no more similar to one another than DZ twin pairs, this suggests that the heritability of language is negligible.

Geneticists seek to determine the extent to which individual differences in a trait are the result of genetic and environmental factors. Putting aside the possibility of interactions and correlations between genetic and environmental factors (see §15), the variation for a trait in a population (the phenotypic variance) is due to genetic variance plus environmental variance. Heritability (h^2) is a measure of the proportion of the phenotypic variance that is due to genetic variance (Falconer 1960, 1989). In twin studies, environmental factors that may contribute to phenotypic variance are divided into factors that co-twins share (common environmental factors, c^2) and those they do not share (specific environmental factors, e^2). Shared environmental factors include, for example, the linguistic input children receive (assuming parents of twins speak the same way to both). Nonshared environmental factors include, for example, illnesses or accidents that only one co-twin experiences.

4.2. TWIN CONCORDANCE ANALYSES OF LANGUAGE DISORDERS. One way to determine whether MZ co-twins are more alike than DZ co-twins is to compare the concordance rates for MZ and DZ twins. Twins are concordant for a trait if both express the trait or neither expresses it. Twins are discordant for a trait if one exhibits the trait and the other does not. If the concordance rate for language disorders is significantly greater for MZ than DZ twins, this suggests that genetic factors play a role in language disorders such as dyslexia and specific language impairment (SLI). If there is no significant difference in the concordance rates for MZ and DZ twins, this suggests that genetic

[3] As discussed in §§11.2 and 11.3, these assumptions may be faulty.

STUDY	TYPE OF DISORDER	METHOD USED TO LOCATE TWINS	METHOD USED TO EVALUATE LANGUAGE
Bakwin 1973	dyslexia	twin clubs in NYC	parent report
Colorado Study of Reading Disability	dyslexia	public school districts in Colorado	standardized tests
Matheny et al. 1976	dyslexia	subset of twins from the Louisville Twin Study	school records
Stevenson et al. 1987	dyslexia	subset of twins from Stevenson et al. who performed poorly	standardized tests
Zerbin-Rudin 1967	dyslexia	published case studies	varied
Bishop et al. 1995, 1996	SLI	ads in UK newspapers	standardized & nonstandardized tests
Bishop et al. 1999	SLI	subset of twins in Bishop et al. 1995, 1996 plus poor performers in new sample of twins	standardized & nonstandardized tests
Lewis & Thompson 1992	SLI	subset of twins from Western Reserve Twin Project	parent report of history of speech/language therapy
Stromswold & Rifkin 1996	SLI	clinics and special-needs schools in NJ	analyses of spontaneous speech; standardized & nonstandardized tests
Tomblin & Buckwalter 1994	SLI	public schools in Iowa	school speech/language pathologist report
Tomblin & Buckwalter 1998	SLI	clinics, twin clubs, high-risk infant registry in midwestern US	standardized tests
Twins Early Development Study (TEDS)	SLI	TEDS twins in bottom fifth percentile on vocabulary and/or nonverbal measures	parent report of language development

TABLE 1. Ascertainment of impaired twins.

factors play little role in developmental language disorders. As shown in Tables 1 and 2, the studies reviewed in this article used different methods to locate (ascertain) twins. Because proband concordance rates are less affected by the way twins are ascertained than concordance rates calculated using other methods such as the pairwise concordance method (McGue 1992), the proband method shown in 1 was used to calculate concordance rates for studies included in this review.

(1) Probandwise concordance rates
number of affected individuals in concordant pairs ÷ total number of affected individuals

A conservative test of the significance of the difference in proband concordance rates for MZ and DZ twins can be obtained by calculating the *Z*-scores for the difference between proportions as shown in 2 where p_1 and p_2 are the probandwise concordance rates for MZ and DZ twins, respectively, N_1 and N_2 are the number of MZ and DZ twin pairs, and p_p is set to .5 (Snedecor and Cochran 1980).

$$(2)\quad Z = \frac{p_1 - p_2}{\sqrt{p_p(1 - p_p)\left(\frac{1}{N_1} + \frac{1}{N_2}\right)}}$$

STUDY	METHOD USED TO LOCATE TWINS	METHOD USED TO EVALUATE LANGUAGE
Bishop et al. 1999	twins ages 7–13 Hertfordshire, Cambridgeshire schools	standardized & nonstandardized tests
Chubrich 1971	twin clubs in Buffalo, NY	standardized tests
Colorado Study of Reading Disability	public school districts in Colorado (normal controls)	standardized tests
Fischer 1973	birth records & twin clubs in Philadelphia	standardized & nonstandardized tests
Foch & Plomin 1980	twin clubs, newspaper articles, & advertisements	standardized tests
Ganger 1998, Ganger et al. 1999	twin clubs, twin computer newsgroups, ads in twin publications	parent report; analyses of spontaneous speech
Hohnen & Stevenson 1999	schools in metro London	standardized tests
Koch 1966	twin clubs, advertisements, referrals in greater Chicago area	standardized test
Louisville Twin Study	birth & school records, twin clubs in Louisville, KY	standardized tests
Mather & Black 1984, Locke & Mather 1989	Purdue & Indiana University Medical Centers, twin clubs in Indiana & Louisville, KY	analyses of spontaneous speech; standardized & nonstandardized tests
Mittler 1969	public health registries in 2 English counties	standardized tests
Munsinger & Douglass 1976	twin clubs in San Diego	standardized tests
Osborne et al. 1968	referrals from Atlanta GA	standardized tests
Reznick et al. 1997	review of Colorado birth records	standardized & nonstandardized tests
Segal 1985	twin clubs, schools, recreation centers, & physicians, NYC, Chicago	standardized test
Stevenson et al. 1987	twins born 1967–68, living in London	standardized tests
Twins Early Development Study (TEDS)	twins born 1994, England & Wales	parent report
Thompson et al. 1991	Birth & school records in Ohio	standardized tests

TABLE 2. Ascertainment of normal twins (major studies only).

Z-scores are normally distributed when N_1 and N_2 are reasonably large, and the value of p_p is not extreme (Snedecor & Cochran 1980).[4]

4.3. CORRELATIONAL ANALYSES. When the data obtained are continuous (e.g. scores on standardized language tests) rather than dichotomous (presence or absence of a language disorder), one can study whether MZ twins are linguistically more similar to one another than are DZ twins by comparing performance on language tests. For example, the correlation coefficient (r) provides a measure of how highly correlated twins' scores are with their co-twins' scores. The intraclass correlation coefficients are calculated separately for MZ and DZ twins. If the MZ intraclass correlation coefficient is significantly greater than the DZ intraclass correlation coefficient, this suggests that

[4] A good rule of thumb is that Z-scores can be used if the product of the smaller of N_1 and N_2 and the smaller of P_p and $(1 - P_p)$ is greater than or equal to 5. I am grateful to Dorothy Bishop and Jeffrey Gilger for discussions on significance testing for proband-wise concordance rates.

genetic factors play a role in language. One can test whether the intraclass correlation coefficients for MZ and DZ twins are significantly different by transforming the correlation coefficients into Fisher z's. When N_1 and N_2 equal the number of MZ and DZ twin pairs, respectively, the quantity given in 3 is distributed as Z (Snedecor & Cochran 1980).

$$(3)\quad Z = \frac{z_1 - z_2}{\sqrt{\frac{1}{N_1 - 3} + \frac{1}{N_2 - 3}}}$$

A number of different formulas have been used to estimate the size of genetic and environmental factors using MZ and DZ correlation coefficients. The most widely used measure, Falconer's estimate of heritability (Falconer's h^2), is calculated by doubling the difference between the MZ and DZ correlations ($h^2 = 2\,[r_{mz} - r_{dz}]$) (Falconer 1960, 1989). An estimate of the role played by environmental factors shared by co-twins (c^2) can be computed by subtracting Falconer's h^2 from the MZ correlation ($c^2 = r_{mz} - h^2$). Artifacts such as systematic rater bias—the tendency of parents or experimenters to give similar scores to co-twins—may inflate the value of c^2. An estimate of the influence of environmental factors that co-twins do not share (e^2) can be calculated by subtracting the MZ correlation from one ($e^2 = 1 - r_{mz}$). The value of e^2 includes variance due to measurement error and contrast effects of rater bias that artificially inflate differences between twin scores.[5] Estimates of the effects of genetic factors, shared environment factors, and nonshared environment factors sum to 1.

4.4. DeFries-Fulker analyses. If a disorder is actually just the low end of the normal distribution for a quantitative trait, concordance analyses may lose information by artificially dichotomizing a quantitative trait into affected versus nonaffected categories. DeFries and Fulker (1985) developed a technique called the DF extremes analysis in which MZ and DZ twins' performance on language tests are compared using multiple regression analyses. If MZ and DZ probands are ascertained by deviant scores on a standardized test, then co-twins' scores on the same test should regress toward the mean score of an unselected population. Because MZ twin pairs are genetically identical whereas DZ twin pairs share only half their genetic material, to the extent that linguistic abilities are hereditary, DZ co-twins' scores should regress to the population mean more than MZ co-twins' scores (DeFries & Fulker 1985). Standardized scores can be fit to the regression equation given in 4, where C is the predicted score for the co-twin, P is the proband score, R is the coefficient for degree of genetic similarity (1.0 for MZ twins and .5 for DZ twins), A is the regression constant, B_1 is the partial regression of the co-twin score on the proband (a measure of twin resemblance that is independent of zygosity), and B_2 is the partial regression of the co-twin score on the coefficient of the relationship. B_2 is equal to twice the difference between the means for MZ and DZ co-twins (adjusted for differences between MZ and DZ probands). If B_2 is significant, this indicates that there is a significant heritable effect. An estimate of the extent to which a disorder is heritable (h^2_g) can be calculated by dividing B_2 by the difference between the means for probands and an unselected population. (For more information, see DeFries & Fulker 1985.)

$$(4)\quad C = B_1P + B_2R + A$$

[5] To save space, Falconer's estimates of effects of shared and nonshared environments are not always given. However, these can easily be calculated from the numbers provided.

Group differences heritability (h^2_g) is a measure of the heritability of a trait in a population suffering from a disorder, whereas individual differences heritability (h^2) is a measure of the heritability of a trait in the general population. As discussed in §1, genetic factors might play a role in the linguistic abilities of people suffering from language disorders but play no role in the linguistic abilities of normal individuals. It is possible that extreme scores on a test may be more heritable than scores for the general population. DeFries and Fulker (1988) developed an augmented multiple regression technique that may be used for comparing normal MZ and DZ twins' performance on tests and thereby get an estimate of h^2. The idea of the augmented DF analysis is basically the same as the DF extremes analysis: in general, co-twins' scores will regress to the mean score. If linguistic abilities are heritable, then DZ co-twins' scores should regress more than MZ co-twins' scores. An additional term (PR), which represents the product of proband score (P) and the coefficient of the relation (R), is added to the DF extremes regression equation. If there exist genes that contribute to the variance among the language-disordered population but not to variance among the general population, h^2_g will be greater than h^2 (DeFries & Fulker 1988).

4.5. COVARIATION ANALYSES. Recall that two of the goals of this article are to determine whether the genetic factors that affect language (if they exist) are specifically linguistic and whether the same genetic factors are involved in all aspects of language. These questions may be addressed using bivariate analysis and Cholesky modeling techniques. Although it is not possible within the scope of this review to go into the details of bivariate analysis and Cholesky modeling (see de Jong 1999), the idea is fairly straightforward (but see Loehlin 1996 for a cautionary note). Whereas in univariate analyses, a twin's performance on test A is compared with his co-twin's performance on that same test, in bivariate analyses, a twin's performance on test A is compared with his co-twin's performance on test B. Genetic influence on the phenotypic correlation between test A and B (bivariate heritability) is estimated by the extent to which the MZ crosstwin correlation is greater than the DZ crosstwin correlation. In contrast, genetic correlation (R_G) estimates the extent to which the same genetic factors affect A and B regardless of their contribution to the correlation between A and B. Genetic correlation for two traits may be high yet bivariate heritability low, and vice versa. For example, genetic factors might play a substantial role for both traits A and B, but if completely different genetic factors are responsible for A and B, genetic correlation would be zero. Conversely, genetic factors might play only a modest role for traits A and B, but if the same genetic factors are responsible for both traits, genetic correlation will be high. If one wants to determine the relationships among more than two variables, one can use Cholesky decomposition modeling techniques in which one successively adds additional factors and at each step uses bivariate analysis techniques to determine the extent to which the same genetic and environmental factors play a role for these variables (see de Jong 1999 for details).

4.6. METAANALYSES. Most of the twin studies included in this review involved relatively small numbers of twins and, hence, these studies have only enough statistical power to detect relatively large genetic or environmental effects. One way to get around this problem is to compare the results of small studies and determine whether the findings of these studies are similar. If the results of all or most of the studies are similar, it increases the likelihood that the results are not due to chance alone. If studies are similar enough, it may be possible to perform metaanalyses in which the results of studies are combined. Two types of metaanalyses were performed on studies that com-

pared MZ and DZ twins' concordance rates for language disorders. Although published versions of older studies generally do not provide enough information for DF extreme analyses, most papers provide the information necessary to calculate proband concordance rates. Thus, the first type of metaanalysis involved calculating the MZ and DZ proband concordance rates for individual studies using the formula given in 1. The formula in 2 was used to determine whether the difference in MZ and DZ concordance rates was significant. Concordance rates from individual studies were used to calculate mean MZ and DZ concordance rates. In order to determine whether, across the studies, concordance rates were significantly greater for MZ twins than for DZ twins, *t*-tests were performed in which the MZ and DZ concordance rates from individual studies served as data points. Rates were also compared using sign-tests which, although less statistically powerful, do not require that data be distributed normally. The second type of metaanalysis involved combining all of the twins from the different studies into one large pool. Overall concordance rates for the pooled MZ and DZ twin data were calculated using the formula in 1 and the significance of the difference was tested using the formula in 2.

Although many studies of unselected (normal) twins do not provide the information necessary to do DF analyses, most papers provide MZ and DZ correlation coefficients (or the information necessary to calculate them). For these studies, two types of metaanalyses were performed. In the first type—unweighted metaanalysis—MZ and DZ correlation coefficients reported in individual studies were treated equally regardless of how many twin pairs participated in each study. Sign- and *t*-tests were used to determine whether MZ correlations were significantly different than DZ correlations, and unweighted mean correlation coefficients ($\bar{r}$) were calculated for MZ and DZ twins. In the second type of metaanalysis, correlation coefficients from individual studies were weighted according to how many twin pairs participated in each study and weighted mean ($\bar{r}_w$) MZ and DZ correlational coefficients were calculated. Following Rosenthal and Rosnow (1984), weighted mean Fisher's $\bar{z}$'s were calculated using the formula given in 5.

$$(5)\quad \bar{z} = \frac{\sum (N_j - 3)z_j}{\sum (N_j - 3)}$$

MZ and DZ weighted mean Fisher's $\bar{z}$'s were transformed into weighted mean correlation coefficients ($\bar{r}_w$). The formula in 3 was used to determine if the weighted mean MZ and DZ correlation coefficients were significantly different, with N_1 and N_2 equal to the mean number of MZ and DZ twin pairs in the studies included in the metaanalysis.[6]

5. Twin studies of language disorders

5.1. Concordance analyses. As shown in Tables 3 and 4, ten studies included information necessary to calculate concordance rates for written or spoken language disorders (Bakwin 1973, Bishop et al. 1995, Dale et al. 1998, DeFries & Gillis 1993, Lewis & Thompson 1992, Matheny et al. 1976, Stevenson et al. 1987, Tomblin & Buckwalter 1994, 1998, Zerbin-Rudin 1967). Six of the studies relied solely on data from questionnaires or clinical records for diagnosis of disorders, but, in four of the studies (Bishop et al. 1995, DeFries & Gillis 1993, Stevenson et al. 1987, Tomblin & Buckwalter 1998), the twins' language abilities were assessed through direct testing.

[6] Using the mean number of MZ and DZ twin pairs for N_1 and N_2 is extremely conservative and may lead to Type II errors (incorrectly failing to reject the hypothesis that MZ and DZ twins are the same), but it should not cause Type I errors (incorrectly rejecting the hypothesis that MZ and DZ twins are the same).

STUDY	NUMBER OF TWIN PAIRS	DIAGNOSIS	PROBAND CONCORDANCE
Bakwin 1973	31 MZ (19 M, 12 F) 31 DZ (19 M, 12 F)	dyslexia	Overall $mz = 91\%$; $dz = 45\%$*** Male $mz = 91\%$; $dz = 59\%$* Female $mz = 91\%$; $dz = 15\%$****
DeFries & Gillis 1993	133 MZ (58 M, 75 F) 98 DZ (57 M, 41 F)	dyslexia (PIAT scores)[a]	$mz = 68\%$; $dz = 43\%$***
Matheny et al. 1976	17 MZ (sex N/A) 10 DZ (sex N/A)	dyslexia or problems with written language	$mz = 87\%$; $dz = 33\%$**
Stevenson et al. 1987	18 MZ (sex N/A) † 30 DZ (sex N/A) †	reading & spelling retardation (Neale & Schonell tests)	Neale read $mz = 33\%$; $dz = 29\%$ Schon read $mz = 35\%$; $dz = 31\%$ Spelling $mz = 50\%$; $dz = 33\%$
Zerbin-Rubin 1967	17 MZ (sex N/A) 33 DZ (sex N/A)	word blindness	$mz = 100\%$; $dz = 50\%$***
MEAN			$\bar{x}_{mz} = 76.2\%$; $\bar{x}_{dz} = 40.6$**
OVERALL††	212 MZ, 199 DZ		$mz = 74.9\%$; $dz = 42.7\%$ DZ****

TABLE 3. Concordance rates for written-language disorders.

All significance levels are for one-tailed tests * $p < .05$; ** $p < .01$; *** $p < .001$, **** $p < .0001$.
† Number of twin pairs varied according to diagnosis.
†† Mean and overall rates include Stevenson et al.'s Schonell reading-retarded data
[a] Peabody Individual Achivement Test for Word Recognition

STUDY	TWIN PAIRS	DIAGNOSIS	PROBAND CONCORDANCE
Bishop et al. 1995	63 MZ (52 M, 11 F) 27 DZ (20 M, 7 F)	SLI (by test scores)	strict criteria $mz = 70\%$; $dz = 46\%$* broad criteria $mz = 94\%$; $dz = 62\%$**
Dale et al. 1998	75 MZ (sex N/A) 60 DZ (sex N/A)	TEDS twins with small vocabulary at age 2 (bottom 5% by parent report)	$mz = 81\%$; $dz = 42\%$****
Lewis & Thompson 1992	32 MZÝ (24 M, 8 F) 25 DZÝ (18 M, 7 F)	Received speech or language therapy	any disorder $mz = 86\%$; $dz = 48\%$** Articulation $mz = 98\%$; $dz = 36\%$**** LD $mz = 70\%$; $dz = 50\%$ Speech delay $mz = 83\%$; $dz = 0\%$*
Tomblin & Buckwalter 1994	56 MZ (sex N/A) 26 DZ (sex N/A)	SLI (questionnaire to speech pathologists)	$mz = 89\%$ MZ vs. 55% DZ**
Tomblin & Buckwalter 1998	40 MZ (sex N/A) 23 DZ (sex N/A)	poor composite language score (>1 SD below mean)	$mz = 96\%$; $dz = 69\%$**
MEAN†			$\bar{x}_{mz} = 84.4\%$; $\bar{x}_{dz} = 52.1$****
OVERALL†	266 MZ, 161 DZ		$mz = 83.7\%$; $dz = 48.3\%$****

TABLE 4. Concordance rates for spoken-language disorders.

All significance levels are for one-tailed tests: * $p < .05$; ** $p < .01$; *** $p < .001$, **** $p < .0001$
LD = learning disorder, SLI = specific language impairment
† Overall and mean rates include Lewis & Thompson's 'any diagnosis' data and Bishop et al.'s (1995) strict criteria data.

For MZ twins, concordance rates ranged from 35% (Stevenson et al. 1987) to 100% (Zerbin-Rudin 1967), with a mean rate of 80.3%.[7] For DZ twins, concordance rates ranged from 31% (Stevenson et al. 1987) to 69% (Tomblin & Buckwalter 1998), with a mean rate of 46.3%. In all ten studies, concordance rates were greater for MZ than DZ twin pairs, with the differences being significant in all of the studies except Stevenson et al. 1987. The difference between MZ and DZ concordance rates was significant by *t*-test, $t(9) = 7.32$, $p < .0001$, and sign-test, $p < .0001$. When the 478 MZ twin pairs and 360 DZ twin pairs from all ten studies were pooled together, the overall concordance rates were 79.9% for MZ twins and 46.2% for DZ twins ($Z = 9.67$, $p < .0001$).

If spoken language disorders and written language disorders are distinct entities, including twins with both types of disorders in one analysis could either hide a significant difference or create a spuriously significant difference. As shown in Table 3, for the five twin studies of written language disorders, MZ concordance rates ranged from 33% (Stevenson et al. 1987) to 100% (Zerbin-Rudin 1967), with a mean rate of 76.2%, and DZ concordance rates ranged from 31% (Stevenson et al. 1987) to 50% (Zerbin-Rudin 1967), with a mean rate of 40.6% ($t(4) = 3.80$, $p < .05$, sign-test $p < .05$). Pooling together the 212 MZ and 199 DZ twin pairs with written language disorders yielded overall concordance rates of 74.9% for MZ twins and 42.7% for DZ twins, $Z = 6.53$, $p < .0001$. As shown in Table 4, for the five twin studies of spoken language disorders, MZ concordance rates ranged from 70% (Bishop et al. 1995) to 96% (Tomblin & Buckwalter 1998), with a mean rate of 84.4%, and DZ concordance rates ranged from 42% (Dale et al. 1998) to 69% (Tomblin & Buckwalter 1998), with a mean rate of 52.1%, $t(4) = 11.62$, $p < .0005$, sign test $p < .05$. Pooling together the 266 MZ and 161 DZ twin pairs with spoken language disorder yielded overall concordance rates of 83.6% for MZ twins and 50.2% for DZ twins ($Z = 6.68$, $p < .0001$.)

The finding that concordance rates were significantly greater for MZ twins indicates that genetic factors play a significant role in developmental language disorders. The concordance rates for written and spoken language disorders were reasonably similar, with mean and overall concordance rates for spoken language disorders being approximately 10 percentage points higher than the rates for written language disorders. However, the fact that the mean and overall MZ concordance rates for both written and spoken language disorders were about 33 percentage points higher than the DZ concordance rates is consistent with genetic factors playing an equal role in both types of impairments.

If MZ twin pairs have exactly the same genetic and environmental makeup, we would expect 100% concordance for MZ twins. The fact that the mean and overall concordance rates for MZ twins were less than 100% and only one of the ten studies reported 100% concordance suggests that (1) MZ twins don't have identical genetic and environmental backgrounds, (2) the expressivity or penetrance of language disorders is incomplete (some people have the genotype for language disorder, but do not have a clinically apparent impairment), or (3) researchers failed to diagnose language disorders in some

[7] Concordance rates include Lewis and Thompson's (1992) 'any diagnosis' results, Stevenson et al.'s (1987) 'Schonell reading-retarded' results, and Bishop et al.'s (1995) 'strict criteria' results. Zerbin-Rudin (1967) reported the results of 34 sets of DZ twins, but mentioned that one set was an opposite-sex pair. This opposite-sex pair was excluded and, the Gricean assumption was made that all of the other DZ twin pairs were same sex. In their analyses, Tomblin and Buckwalter (1998) included results obtained from identical and nonidentical sets of triplets. In order to make Tomblin and Buckwalter's (1998) study more similar to the other studies, I excluded from my analyses twin pairings obtained from triplets. I am grateful to Bruce Tomblin for providing the data that allowed me to do so.

of the MZ co-twins. The twin study method assumes that, like nontwin siblings, DZ twins share, on average, 50% of their genetic material. Thus, to the extent that language disorders are due solely to genetic factors, we would expect the concordance rates for DZ twins to be similar to those of nontwin siblings. Stromswold (1998) found that in ten family aggregation studies of spoken language disorders that reported affectedness rates for siblings, the rate of affectedness among probands' siblings ranged from 15% to 49% (mean 30%). Thus, consistent with language disorders being due largely to heritable factors, the affectedness rate for nontwin siblings is quite similar to the overall DZ pairwise concordance rate for spoken language disorders (26%).[8]

Although some studies compared impaired MZ and DZ twins' performance on language tests, because these studies examined different aspects of language using different types of tests, it is not possible to perform the types of metaanalyses described in §4.6. Therefore, such results will be reviewed individually, beginning with studies of spoken language disorders (§5.2) and then proceeding to studies of written language disorders (§5.3).

5.2. ANALYSES OF SLI TWINS' TEST PERFORMANCE. Bishop et al. (1995) compared the performance of 63 MZ and 27 DZ SLI twin pairs on a battery of language tasks.[9] Twins' articulation abilities were assessed using the Edinburgh Articulation Test (Anthony et al. 1971), in which children are asked to pronounce a list of words that is designed to determine whether children can produce English consonants correctly in word-initial and word-final position. In terms of the percent of consonants that SLI twin pairs correctly pronounced, the articulation abilities of the MZ SLI twins were more similar than those of the DZ SLI twins, $r_{mz} = .72, r_{dz} = -.19, Z = 4.55, p < .0001$, Falconer's $h^2 = 1.82$.[10] A number of researchers have suggested that developmental language disorders are due to problems in the perception, processing, or recall of auditory or phonological information (e.g. Adams and Gathercole 2000, Eisenson 1968, Gathercole & Baddeley 1990a, Merzenich et al. 1996, Tallal & Piercy 1973a, 1973b, 1974, Tallal et al. 1991). Bishop and colleagues (1996) assessed their SLI twins' phonological short term memory via Gathercole et al.'s (1994) nonsense-word repetition test: children listen to and repeat phonologically possible (in English) nonsense words that are between 2 and 5 syllables long. The DF group heritability estimate (h^2_g) for phonological short-term memory was 1.25, which indicates that heritable factors accounted for all of the SLI twins' variance in performance on this task. Bishop et al. (1995) assessed the

[8] Affectedness rates for nontwin siblings are compared with pairwise twin concordance rates because the method for calculating affectedness rates for nontwin siblings (# affected siblings/total number of siblings) is more similar to the method for calculating pairwise twin concordance rates (number of concordant twins/total number of twins) than is the method for calculating proband-wise twin concordance rates (number of affected twins who are members of concordant twin pairs/total number of affected twins).

[9] I am grateful to Dorothy Bishop for providing the correlation coefficients for all of the tests performed in Bishop et al. 1995.

[10] Extreme Falconer's heritability estimates (i.e. estimates that are greater than 1 or negative) may result if there are interactions between genetic and environmental factors. For example, if MZ twins are treated more similarly than DZ twins, this may lead to heritability estimates greater than 1. Negative heritability estimates could result from environmental-genetic interactions such as the fact that MZ cotwins are more likely to compete for in utero resources (i.e. twin-twin transfusion syndrome) and such competition may inflate MZ co-twins differences (see Falconer 1989). If genetics plays no role, then one would expect correlation coefficients for both MZ and DZ twins to be randomly distributed around 0 and, on average, this would result in negative heritability estimates half the time. If the number of either MZ or DZ twin pairs is small, this may also result in extreme heritability estimates because a few extreme points could artificially inflate correlation coefficients.

SLI twins' vocabulary by having them point to pictures of words. The MZ correlation coefficient was significantly greater than the DZ correlation coefficient, $r_{mz} = .81$, $r_{dz} = .43$, $Z = 2.76$, $p = .003$, with heritable factors accounting for 76% of the SLI twins' performance on the vocabulary test. They assessed the SLI twins' morphosyntactic abilities using the comprehension subtests of the Wechsler Intelligence Scale for Children-Revised (WISC-R, Wechsler 1974) or, for older children, the Wechsler Adult Intelligence Scale-Revised (WAIS-R, Wechsler 1986), the Clinical Evaluation of Language Function (CELF, Semel et al. 1980) sentence repetition subtest (in which children are asked to repeat increasingly more complex sentences) and Bishop's (1983) Test for Reception of Grammar (TROG) in which children must choose from among four pictures the picture that corresponds to a sentence where choosing correctly depends on syntactic rather than lexical information. Comparison of MZ and DZ twins' correlation coefficients revealed significant effects of heritability for scores on the Wechsler comprehension test ($r_{mz} = .74$, $r_{dz} = .18$, $Z = 3.18$, $p = .0008$, Falconer's $h^2 = 1.12$), and the CELF repetition test ($r_{mz} = .80$, $r_{dz} = .17$, $Z = 3.88$, $p < .0001$, Falconer's $h^2 = 1.26$), but not for scores on the TROG ($r_{mz} = .56$, $r_{dz} = .32$, $Z = 1.25$, $p = .11$, Falconer's $h^2 = 1.48$). Bishop et al. (1995) also analyzed SLI subjects' scores on vocabulary and morphosyntactic tests using DF extremes analyses. DZ co-twins' language scores regressed significantly more than MZ co-twins' scores for all 4 tests ($p < .01$), and the DF group heritability estimates indicate that heritable factors account for all of the variance on three tests (Vocabulary $h^2_g = 1.35$; Wechsler $h^2_g = 1.10$; TROG $h^2_g = 1.09$), and over half of the variance on the fourth test (CELF $h^2_g = .56$). However, when performance on the Raven's (1986) matrices test of nonverbal abilities was controlled for, Falconer's and DF extreme analyses revealed no significant genetic effects for any of these tests. However, as Bishop (p.c.) cautions, even though the Raven's matrices test is considered a nonverbal test, the SLI children's poor verbal abilities may have adversely affected performance on this test. Hence, controlling for performance on the Raven's matrices test may have inadvertently eliminated some of the genetically influenced variance in linguistic abilities.[11]

Testing many of the same twins that were studied in Bishop et al. 1995, Bishop and colleagues (1999) identified 27 MZ and 21 DZ twin pairs in which at least one co-twin did poorly on Tallal's pure tone repetition test (Tallal & Piercy 1973a,b). In this test, subjects listen to and repeat a sequence of 100 Hz and 300 Hz tones. The tone sequences range in length from two tones to seven tones, with inter-tone intervals of 10 or 70 msec. DF-extremes analyses revealed no evidence that performance on this test was affected by heritable factors ($h^2_g = .11$). Bishop et al. (1999) also identified 25 MZ and 22 DZ twin pairs in which one co-twin did poorly on Gathercole et al.'s (1994) nonsense-word repetition task. In contrast to the findings for pure tone sequences, DF-extremes analyses revealed that heritable factors accounted for all of the variance in the SLI children's ability to repeat nonsense words ($h^2_g = 1.17$).

Tomblin and Buckwalter (1998) performed DF extreme analyses on the data from 40 pairings of MZ SLI twins and 23 pairings of DZ SLI twins who were between the ages of four and sixteen at the time of testing. At least one member of each twin pairing had poor language achievement as defined by a composite expressive and receptive language score of at least one standard deviation below the mean. (The tests used to

[11] Bishop reported to me that she is doing additional nonverbal tests (WISC-R block design and picture completion subtests) on a subset of the children and that preliminary results provided a different picture than those for Raven's matrices.

obtain the composite language score varied according to the age of the twins, see Tomblin & Buckwalter 1998.) The DF group heritability estimate (h^2_g) for poor language achievement was .45 and the individual heritability estimate (h^2) for language was .48. Because some of Tomblin and Buckwalter's twins had low nonverbal IQs (nonverbal IQs of between 70 and 85), these heritability estimates could reflect the role genetic factors play in general cognitive ability, rather than the heritability of language. When they performed the same analyses on the data from the 30 MZ and 16 DZ twin pairings in which both twins had nonverbal IQs of above 85, DZ co-twins' scores regressed significantly more than MZ co-twins' scores ($h^2_g = .47$). The similarity of the h^2_g's for the populations of twins that included twins with low IQs and those that did not suggests that the heritability of language disorders is not merely the result of heritability of low cognitive function.

Tomblin and Buckwalter (1998) included results from five twin pairings obtained from sets of triplets. Bruce Tomblin (p.c.) kindly provided the data necessary to calculate correlations when twin pairings from triplets were removed. The intraclass correlational coefficients for language were .66 for MZ twins and .33 for DZ twins, $Z = 1.61$, $p = .054$, with 66% of composite language scores attributable to genetic factors and 34% attributable to nonshared environmental factors. Bivariate analyses of normalized nonverbal IQ and normalized language scores yielded a modest bivariate heritability of .21, with an estimate of bivariate nonshared environmental factors of .80. The genetic correlation between IQ and poor language achievement was less than .01. These results indicate that, although genetic factors play a modest role in the phenotypic correlation between nonverbal IQ and language skills, different genetic factors influence nonverbal IQ and language.

Dale et al. (1998) used the DF extremes method to analyze the data from 75 MZ and 60 DZ two-year-old twin pairs in which at least one of the co-twins had a vocabulary size in the bottom 5% for the entire Twins Early Development Study (TEDS) population. Vocabulary size was measured by parents' responses on a short version of the MacArthur Communicative Development Inventory vocabulary checklist (MCDI, Fenson et al. 1994). DZ co-twins' vocabulary scores regressed significantly more than MZ co-twins' scores, with genetic factors accounting for 73% of the variance in the vocabulary size of probands, shared environmental factors accounting for 18% of the variance, and nonshared environmental factors accounting for the remainder. Because of the enormous number of twins in the TEDS project, the researchers were able to compare the role genetic factors play in vocabulary size for twins with very small vocabularies versus the role genetic factors play in vocabulary size for the entire population of twins (i.e. the population of all twins regardless of their vocabulary size). DF analyses performed on the entire population of twins indicate that genetic factors account for only 25% of the variance in vocabulary size across the entire range of vocabulary sizes, with shared environmental factors accounting for 69% of the variance in vocabulary size. Thus, results from the TEDS twins suggest that genetic factors play a greater role in vocabulary size for children who are slow word-learners than for children who learn words at a normal rate.

Eley et al. (1999) investigated whether genetic factors play equal roles in verbal and nonverbal delays in the TEDS two-year-olds. They performed separate DF extreme analyses for twin pairs in which at least one co-twin scored in the bottom 5% in language (language delay), in nonverbal abilities (nonverbal delay), in a composite of language and nonverbal abilities (general delay), in both language and nonverbal abilities (i.e. bottom 5% in both verbal and nonverbal measures, comorbid delay), in language but

not nonverbal abilities (language-only delay), and nonverbal but not language abilities (nonverbal-only delay). Language development was measured by parental report of vocabulary size on the MCDI. Nonverbal development was measured using the Parent Report of Children's Abilities (PARCA). In the PARCA, parents administer standard nonverbal cognitive tasks and report on nonverbal cognitive abilities (Saudino et al. 1998). Eley et al. (1999) found that heritability estimates were greater for groups that included children in the bottom 5% for language (the language delay, language-only delay, and comorbid delay groups), than for groups that did not include such children (the nonverbal delay, nonverbal-only delay, and general delay groups). For example, DF extremes analyses of the language-only delay group yielded a heritability estimate of .78, with shared environment estimate of .20, whereas for the nonverbal-only group, the heritability estimate was .22 (nonsignificant), with a significant shared environment estimate of .54. It is possible that genetic factors merely affect the earliest stages of language acquisition, with genetically at-risk children quickly catching up with children who are not at risk. Eley et al. 2001 investigated genetic stability by comparing the extent to which genetic and environmental factors contribute to verbal and nonverbal delays when the TEDS twins were two and three years of age. As was the case at two years of age, at age three, heritable factors played a greater role in language delay than in nonverbal delay (e.g. the language-only delay h^2_g was .62, whereas the nonverbal-only delay h^2_g was .29). Using a variant of bivariate analyses to quantify genetic continuity, Eley et al. (2001) compared the performance of twins who were in the bottom 5% at age two (age two probands) with the performance of their co-twins at age three. If the MZ cross-age correlation is significantly greater than DZ cross-age correlation, this indicates that there is genetic continuity, that is, genetic factors play a substantial role at both ages. Although genetic continuity was greatest for TEDS twins who suffered from both verbal and nonverbal delays ($h^2_g = .87$), genetic continuity was twice as great for verbal-only delay than nonverbal-only delay (verbal-only $h^2_g = .48$ vs. nonverbal-only $h^2_g = .22$).

Although the TEDS results summarized above suggest that genetic factors play a greater role in the language delay than in nonverbal delay, bivariate analyses of verbal and nonverbal delays are necessary to rule out the possibility that the same genetic factors are involved in both types of delay but to different degrees. Purcell and colleagues (2001) conducted bivariate analyses on the TEDS data for two-year-olds. They found that when they used small vocabulary size as the method for selecting probands (language delay probands), the genetic correlation between verbal delay and nonverbal delay was 100%. This indicates that when probands are selected for verbal delay, the same genetic factors are responsible for verbal and nonverbal delay. That is, the same genes that cause verbal delay are responsible for the lowered nonverbal scores of verbal-delay probands. When they selected probands based on low scores on the PARCA nonverbal test (nonverbal delay probands), the genetic correlation between nonverbal delay and verbal was 36%. This indicates that the genes responsible for nonverbal delay are largely different from the genes responsible for lowered verbal scores in nonverbal-delay probands. This asymmetry suggests that the low verbal group is more genetically homogenous than the low nonverbal group.

Stromswold and Rifkin (1996) studied a set of MZ and a set of DZ SLI twins, analyzing data collected every month for over two years. Even though all four children were diagnosed as having SLI, for all measures (age of acquisition, rate of different type of errors, indices such as mean length of utterance and type/token ratios, and performance on comprehension and production tests) and all areas of language assessed

(phonology, vocabulary, morphology, and syntax), the MZ co-twins were more similar to one another than the DZ co-twins were. And where the MZ co-twins continued to exhibit very similar linguistic profiles over the course of the study, the DZ co-twins became linguistically more different as time passed.

5.3. DYSLEXIC TWINS' TEST PERFORMANCE. Multivariate analyses of data from the ongoing Colorado Twin Study of Reading Disabilities have been invaluable in uncovering the environmental and genetic factors that are associated with dyslexia (Alarcón et al. 1997, Castles et al. 1999, Casto et al. 1996, DeFries & Fulker 1985, DeFries & Gillis 1993, DeFries et al. 1987, Light et al. 1998, Olson et al. 1989). Using the data obtained from 196 MZ and 155 DZ Colorado study twin pairs in which at least one co-twin scored in the dyslexic range on standardized tests, Light et al. (1998) calculated DF heritability estimates for phonologically based and overall reading ability. Phonologically based reading was assessed by a test in which twins had to pronounce nonsense words (*calch*, *posket*) as quickly as possible. Overall reading ability was assessed by the Peabody Individual Achievement Test (PIAT) reading and spelling tests (Dunn & Markwardt 1970). For phonologically based reading, heritable factors accounted for 52% of the variance, with nonshared environmental factors accounting for 36% of the variance. For overall reading ability, heritable factors accounted for 70% of the variance, with nonshared environmental factors accounting for 27% of the variance. Also as part of the Colorado Twin Study, Olson et al. (1989) tested 64 MZ and 53 DZ dyslexic twin pairs and 104 nondyslexic twin pairs on a battery of tests designed to assess different aspects of reading. The phonological coding tests included a speeded nonsense-word reading test and a test in which twins had to read three nonsense words (e.g. *kep*, *kap*, *ket*) and decide which word sounded like a real English word. In an orthographic coding test, twins had to pick the actual word from a word-pseudohomophone pair (e.g. *room*/*rume*, *explain*/*explane*). Children were also given a speeded reading test in which they had two seconds to read words, and the PIAT word-recognition test (Dunn & Markwardt 1970), in which they chose the correct spelling of a word from among four alternative spellings. DF extreme analyses revealed that DZ co-twins' scores regressed significantly more than MZ co-twins' scores ($p < .001$) for all tests but the orthographic coding test (PIAT $h^2_g = .40$; timed reading $h^2_g = .39$; phonological coding $h^2_g = .93$; orthographic coding $h^2_g = -.16$). When test scores were adjusted for IQ, the results were essentially unchanged, suggesting that there are genetic factors affecting reading independent of those that affect IQ. Based on these results, Olson et al. (1989) concluded that phonological coding deficits are significantly heritable and account for most of the heritable variance in reading disability, whereas orthographic coding deficits are not significantly heritable and account for much of the environmental variance in reading disability. In a follow-up study of even more twins from the Colorado twin study, Castles et al. (1999) used an extended version of the DF-extreme technique to further quantify the genetic contribution to phonologically and orthographically based dyslexia. In this study there were 592 pairs of dyslexic twins (272 MZ, 320 DZ).[12] Children were classified as having a phonologically based reading disorder if they had difficulty reading nonsense words, and as having an orthographically based reading disorder if they had difficulty reading irregularly spelled words (e.g. *eye*, *fruit*, *yacht*). The phonological dyslexic group consisted of 208 twin pairs (97 MZ, 111 DZ). For

[12] Unfortunately, Castles et al.'s (1999) study included opposite-sex DZ twins. Despite this fact, I have included the study because it makes a unique contribution to the study of the heritability of language.

the phonological group, 67% of the variance in performance was attributable to heritable factors ($p < .001$), and 27% was attributable to shared environmental factors ($p < .05$). The orthographic dyslexic group consisted of 195 twin pairs (89 MZ, 106 DZ). For the orthographic group, 31% of the variance in performance was attributable to heritable factors ($p < .05$), and 63% was attributable to shared environmental factors ($p < .001$). Based on these findings and the results of supplementary analyses, Castles et al. (1999) conclude that, although genetic and environmental factors play significant roles in both types of dyslexia, genetic factors play a much larger role in phonologically based dyslexia, and environmental factors play a much larger role in orthographically based dyslexia. Analyzing data from the same dyslexic twins studied by Castles and colleagues (1999), Gayan and Olson (1999) found that genetic and shared environmental factors significantly affect ALL aspects of reading (PIAT word recognition $h^2_g = .45$, $c^2_g = .49$; phonemic awareness $h^2_g = .56$, $c^2_g = 24$; phonological decoding $h^2_g = .61$, $c^2_g = .24$; orthographic coding $h^2_g = .58$, $c^2_g = .20$). Contrary to Olson et al.'s results (1989), Gayan and Olson (1999) concluded that heritable factors play just as great a role in orthographic decoding as phonological decoding and shared environmental factors do not play a greater role in orthographic than phonological decoding.

Children may have difficulty learning to read because they have a specific problem with reading (dyslexia) or because they are not very bright. Indeed, one of the most common ways of identifying dyslexic children is to look for children with a large discrepancy between IQ and reading level (see Francis et al. 1991, Meyer 2000, Stanovich 1994, Toth & Siegel 1994). When the dyslexic twins in the Colorado twin study were divided into three groups based on full-scale IQ, Olson and colleagues (1999) discovered that as IQ increased, heritability estimates for PIAT word recognition increased significantly and shared environment estimates decreased significantly (low IQ $h^2_g = .32$, $c^2_g = .63$; medium IQ $h^2_g = .39$, $c^2_g = .56$; high IQ $h^2_g = .54$, $c^2_g = .37$). Subsequent analyses revealed that the heritability of reading disability differs as a linear function of full-scale IQ (Wadsworth et al. 2000). These results suggest that IQ is relevant for the diagnosis of reading disability and that environmental influences may be more salient as a cause of reading difficulties in children with lower IQ scores.

Many dyslexic children also do poorly in mathematics (see Alarcón et al. 1997, Casto et al. 1996, Light et al. 1998, and references therein). Light et al. (1998) analyzed the math, phonological reading, overall reading, and verbal IQ scores of dyslexic Colorado twins to determine the reason for this comorbidity. Using Cholesky decomposition modeling, the researchers determined that the genetic correlation between overall reading and math scores was .36, with genetic factors common to verbal IQ accounting for 63% of this correlation and genetic factors common to phonological reading accounting for an additional 20% of the correlation. These findings suggest that deficits in verbal IQ and phonological decoding can result in both math and reading disorders.

5.4. STUDIES OF LANGUAGE IMPAIRMENTS. Depending on what aspect of language was being assessed and the technique used to assess it, results of twin studies suggest that between 25% and 100% of the variance in language-impaired children's linguistic performance is due to genetic factors. It is not clear whether these genes are specific to language. For written language, the results of the Colorado Study of Reading Disabilities suggest that, in addition to genetic factors that influence both reading and IQ, some of the genetic factors that affect reading ability in dyslexic twins are specific to language. With respect to spoken language disorders, the results of Tomblin and Buckwalter's

1998 study indicate that the heritability of SLI is not just a reflection of the heritability of low cognitive ability. Bishop et al. (1995), however, found that when they controlled for performance on a nonverbal IQ test, genetic factors did not play a significant role in their SLI twins' performance on any of the linguistic tests administered. If the nonverbal IQ test they used is a good specific measure of nonverbal ability, these results suggest that the genetic factors that influence language performance are not specific to language.

The TEDS results for 2-year-old twins suggest that although genetic factors are more important for verbal delays than nonverbal delays (Eley et al. 2001, Eley et al. 1999), when probands are selected based on small vocabulary size, the same genetic factors are responsible for verbal and nonverbal delays (Purcell et al. 2001). However, Purcell et al.'s verbal-delay probands were selected solely because they had small vocabularies. Some of these twins may suffer from SLI, but others may have low-normal IQs, and still others may be ''late-talkers'' who will eventually outgrow their verbal impairment (Mills et al. 1997, Paul 1993, Weismer et al. 1994). By definition, children with SLI are specifically impaired in language but not in nonverbal cognition. Hence, if one wishes to investigate the genetic specificity of SLI, then one should do bivariate analyses on the verbal and nonverbal scores for TEDS twins with low verbal scores but normal nonverbal scores (verbal-only delay twins). Because the majority of late-talkers who are going to catch up with their normal peers do so by age three or four (Paul 1993), it makes sense to calculate verbal-nonverbal genetic correlations at age three and four. If the genetic correlation for verbal-only delay twins is high at three and four, this suggests that the genes involved in SLI are not specific to language.

The results of the twin studies reviewed in this section suggest that genetic factors play a substantial role in the development of language abilities among children who are having difficulty learning to talk or read. But it is possible that genetic factors play little or no role in the variation in linguistic abilities observed for normal individuals (§1), and also possible that genetic factors account for some of the variation among normal individuals for certain aspects of language but not others. For these reasons, I now review studies that investigate normal twins' lexical, phonological, morphosyntactic, and written language skills.[13]

6. Vocabulary

6.1. Description of studies. As shown in Table 5, eight twin studies compared normal MZ and DZ twins' performance on tests of spoken vocabulary (Dale et al.

[13] Numerous twin studies have established that MZ twins perform more similarly on IQ tests than do DZ twins (e.g. Bouchard & McGue 1981). To the extent that IQ tests directly or indirectly tap verbal abilities (see Osborne et al. 1968), the MZ-DZ IQ difference is consistent with genetic factors affecting language. However, it is possible that nonverbal portions of IQ tests account for all or most of the heritability of full-scale IQ. Verbal IQs of 270 MZ and 220 DZ twin pairs were examined in four studies (Koch 1966, Segal 1985, Stevenson et al. 1987, Wilson 1975). For verbal IQ, MZ correlations were significantly greater than DZ correlation in all four studies, with Falconer's estimates of heritability ranging from .50 (Stevenson et al. 1987) to .84 (Segal 1985). For full-scale IQ, the weighted mean correlation coefficients were .84 for MZ twins and .54 for DZ twins ($Z = 3.30$, $p = .0005$, Falconer's $h^2 = .60$). For nonverbal IQ, the $\bar{r}_w$ was .80 for MZ twins and .57 for DZ twins ($Z = 2.60$, $p = .0047$, Falconer's $h^2 = .46$). For verbal IQ, the $\bar{r}_w$ was .82 for MZ twins and .49 for DZ twins ($Z = 3.31$, $p = .0004$, Falconer's $h^2 = .66$). Thus, heritable estimates suggest that genetic factors play a greater role in verbal IQ (66%) than performance IQ (46%). In addition, Segal found that the MZ twin pairs' performance was significantly more similar than DZ twin pairs' performance on 4 of 5 verbal subtests (the difference was marginally significant for the fifth subtest), but only 3 of 5 performance subtests. Taken together, these results suggest that heritable factors play a greater role in verbal IQ than nonverbal IQ.

STUDY	NO. OF TWIN PAIRS	AGES	TESTS	CORRELATION COEFFICIENTS	FALCONER'S HERITABILITY ESTIMATE
Reznick et al. 1997	144–204 MZ (F > M)†	14 mos	preferential looking	Preferential looking	Preferential looking
	128–172 DZ (M > F)†	20 mos	expressive & receptive	14 mos r_{mz} = .06; r_{dz} = .07	14 mos = .02
		24 mos	vocabulary (parent report)	20 mos r_{mz} = .23; r_{dz} = .24	20 mos = .02
				24 mos r_{mz} = .26; r_{dz} = .21	24 mos .10
				Expressive vocabulary	Expressive vocab
				14 mos r_{mz} = .38; r_{dz} = .37	14 mos .02
				20 mos r_{mz} = .76; r_{dz} = .63	20 mos .26**
				24 mos r_{mz} = .79; r_{dz} = .60	24 mos .38***
				Receptive vocabulary	Receptive vocab
				14 mos r_{mz} = .64; r_{dz} = .49	14 mos .30*
				20 mos r_{mz} = .68; r_{dz} = .61	20 mos .14
				24 mos r_{mz} = .71; r_{dz} = .62	24 mos .18
Ganger et al. 1999	57 MZ (27 M, 30 F)	$\bar{x}$ = 18	Age vocabulary size = 25	r_{mz} = .98; r_{dz} = .92	.12***
	35 DZ (22 M, 13 F)	mos	words (diary study)		
Dale et al. 1999	1008 MZ (466 M, 542 F)	24 mos	Vocabulary size at age 2	r_{mz} = .96; r_{dz} = .82	.28****
	959 DZ (485 M, 474 F)		(MCDI parental report)		
Fischer 1973	11 MZ (3 M, 8F)	3.5 yrs	Stanford-Binet	Stanford r_{mz} = .91; r_{dz} = .45	Stanford-Binet .92*
	10 DZ (4 M, 6 F)		PPVT	PPVT r_{mz} = .71; r_{dz} = .48	PPVT .46
			Mehrabian vocabulary	Mehrabian r_{mz} = .72; r_{dz} = .01	Mehrabian 1.41*
				MEAN r_{mz} = .78; r_{dz} = .21	MEAN .94
Mather & Black 1984	50 MZ (21 M, 29 F)	3–6 yrs	PPVT	r_{mz} = .78; r_{dz} = .44	.68**
	29 DZ (11 M, 18 F)	$\bar{x}$ = 4.5			
Foch & Plomin 1980	53 MZ (25 M, 29 F)	5–12 yrs	McCarthy & WISC-R	r_{mz} = .81; r_{dz} = .68	.26
	31 DZ (17 M, 16 F)	$\bar{x}$ = 7.6	vocabulary		
Segal 1985	70 MZ (33 M, 37 F)	5–13 yrs	WISC-R vocabulary &	Vocab r_{mz} = .78 vs. .42	Vocab .72**
	35 MZ (22 M, 13 F)	$\bar{x}$ = 8.0	similarities	similar r_{mz} = .76 MZ vs. .29	Similar .94***
				MEAN r_{mz} = .77; r_{dz} = .36	MEAN .82**
Thompson et al. 1991	146 MZ, 132 DZ	6–12 yrs	WISC-R vocabulary &	r_{mz} = .60; r_{dz} = .41*	.38
		$\bar{x}$ = 9.8	verbal fluency		
WEIGHTED MEAN	1247 MZ, 1152 DZ	<3 yrs		r_{mz} = .95; r_{dz} = .80	.29****
WEIGHTED MEAN	330 MZ, 237 DZ	3–12 yrs		r_{mz} = .71; r_{dz} = .45	.53*
WEIGHTED MEAN	1577 MZ, 1389 DZ	2–12 yrs		r_{mz} = .93; r_{dz} = .76	.33****

TABLE 5. Vocabulary.

Significance levels are for one-tailed tests: * $p < .05$ ** $p < .01$; *** $p < .001$, **** $p < .0001$

† Number of twins in Reznick et al. (1997) varied for different measures and different ages

2000, Fischer 1973, Foch & Plomin 1980, Ganger et al. 1999, Mather & Black 1984, Reznick et al. 1997, Segal 1985, Thompson et al. 1991). The ages of the twins varied greatly from study to study. The twins studied by Ganger et al. (1999) were between 10 and 30 months of age, Reznick et al. (1997) tested twins at 14, 20, and 24 months of age, and the twins in the TEDS study were 24 months old (Dale et al. 2000). Fischer (1973) tested twins on a monthly basis for eight months starting between 2.5 and 3.5 years of age, and the twins in Mather and Black (1984) were between 3 and 6 years of age (mean 4.5 years). The twins in the other three studies were between 5 and 13 years of age (mean age 7.6 years for Foch & Plomin's twins, 8.0 years for Segal's twins, and 9.8 years for Thompson et al.'s twins).

Depending on the ages of the twins being studied, different methods were used to assess spoken vocabulary. Reznick et al. (1997) used a preferential looking task in which word comprehension is assessed by determining whether infants look at a display that depicts a word or a display that depicts a distractor word (Reznick 1990). Reznick and colleagues also assessed twins' expressive and receptive language via parental report and experimenter observation. Dale et al. (2000) used the short MCDI parental checklist (Fenson et al. 1994) to assess twins' expressive vocabularies. Ganger et al. (1999) used parents' daily diaries to determine the age at which twins had acquired twenty-five words (i.e. cumulative vocabulary $=$ 25 words).[14] Fischer (1973) assessed expressive vocabulary via the Stanford Binet IQ vocabulary subtest (Terman & Merrill 1960) in which children name pictures. Fischer assessed receptive vocabulary via the Mehrabian (1970) vocabulary test and the Peabody Picture Vocabulary Test (PPVT, Dunn 1965) in which children point to named pictures. Mather and Black (1984) also used the PPVT to assess receptive vocabulary. Foch and Plomin (1980) assessed vocabulary by having children define words taken from the McCarthy (1972) scales and the vocabulary subtest of the WISC-R IQ test. Segal (1985) assessed vocabulary by having children define words and explain how pairs of words are similar. Thompson et al. (1991) assessed vocabulary by having children define words on the WISC-R vocabulary subtest and by having children take a verbal fluency test in which they list as many words as they can that meet a given requirement, for example, names of animals.

6.2. FALCONER'S ESTIMATES OF THE HERITABILITY OF VOCABULARY DEVELOPMENT. Table 5 summarizes eight studies that assessed the spoken vocabularies of a total of 1553 MZ and 1365 DZ twin pairs. The correlations were larger for MZ twins than for DZ twins for all eight studies and for all measures except Reznick et al.'s (1997) preferential looking results for 14- and 20-month-old twins. The MZ-DZ differences were significant for all studies and measures with the exception of Foch and Plomin's (1980) data ($Z = 1.26$, $p = .10$), Fischer's (1973) PPVT data ($Z < 1$) and mean data ($Z = 1.40$, $p = .08$) and Reznick et al.'s (1997) preferential looking data ($Z < 1$ at all 3 ages), expressive vocabulary data at 14 months ($Z < 1$) and receptive vocabulary data at 20 months ($Z = 1.10$, $p = .14$) and 24 months ($Z = 1.48$, $p = .07$). Falconer's h^2's for individual measures ranged from $-.02$ (for Reznick et al.'s preferential looking data for 14- and-20-month-old twins) to 1.41 (for Fischer's Mehrabian vocabulary data). Weighted and unweighted metaanalyses were performed using the mean data from Fischer 1973 and Segal 1985 and Reznick et al.'s expressive vocabulary data from

[14] I am grateful to Jennifer Ganger for providing updated results.

twenty-four-month-old twins.[15] MZ correlations were greater than DZ correlations in all eight studies, $p < .01$. For the eight studies, the unweighted $\bar{r}$ was .81 for MZ twins and .57 for DZ twins, $t(7) = 4.64$, $p = .002$, with 48% of variance in spoken vocabulary attributable to genetic factors, 33% attributable to environmental factors shared by co-twins, and 19% attributable to environmental factors not shared by co-twins. The weighted correlation coefficient ($\bar{r}_w$) was .93 for MZ twins and .76 for DZ twins, $Z = 6.10$, $p < .0001$, with 33% of the variance in spoken vocabulary attributable to genetic factors, 60% attributable to shared environmental factors, and 7% attributable to nonshared environmental factors.

Different factors may be involved in the earliest stages of vocabulary acquisition compared to later stages of acquisition (Carey 1985, 1994). For this reason, separate metaanalyses were performed for twin studies that examined vocabulary acquisition in two year olds versus those that studied older children. The three studies that assessed spoken vocabulary in late infancy (Dale et al. 2000, Ganger et al. 1999, Reznick et al. 1997) included a total of 1247 MZ and 1152 DZ twin pairs. For these studies, the unweighted $\bar{r}$ was .91 for MZ twins and .78 for DZ twins, $t(2) = 3.43$, $p = .075$, with 26% of variance in spoken vocabulary attributable to genetic factors, 65% attributable to shared environmental factors, and 9% attributable to nonshared environmental factors. The weighted metaanalysis revealed an $\bar{r}_w$ of .95 for MZ twins and .80 for DZ twins, $Z = 10.26$, $p < .0001$. According to this analysis, in late infancy, genetic factors account for about a third (29%) of the variance in vocabulary size and shared environmental factors account for two-thirds (66%) of the variance. Nonshared environmental factors account for little of variance in vocabulary size (5%).

Five studies assessed spoken vocabulary in children over the age of two (Fischer 1973, Foch & Plomin 1980, Mather & Black 1984, Segal 1985, Thompson et al. 1991). These studies included a total of 330 MZ and 237 DZ twin pairs. For older twins, the unweighted $\bar{r}$ was .75 for MZ twins and .44 for DZ twins ($t(4) = 4.77$, $p = .009$), with 62% of variance in spoken vocabulary attributable to genetic factors, 13% attributable to shared environmental factors, and 25% attributable to nonshared environmental factors. The weighted metaanalysis of late vocabulary acquisition revealed an $\bar{r}_w$ of .71 for MZ twins and .45 for DZ twins ($Z = 2.12$, $p = .017$). According to this analysis, genetic factors accounted for the majority of the variance in older children's spoken vocabulary (53%) with shared environmental factors accounting for only 18% of the variance and nonshared environmental factors accounting for 29% of the variance.

6.3. DF ESTIMATES OF THE HERITABILITY OF VOCABULARY DEVELOPMENT. Four studies used the augmented DF method to estimate the heritability of spoken vocabulary in unselected (normal) populations. In Ganger et al.'s (1999) study, heritability played a small role in age of acquisition of the 25th word (11%), whereas shared environmental factors played a substantial role (87%). In Dale et al. (2000), genetic factors accounted for 25% of vocabulary size in 2-year-old children, with 69% of vocabulary size attributable to shared environmental factors. In Reznick et al. (1997), shared environmental factors played a significant role in expressive vocabulary at all three ages (35% at 14 months, 49% at 20 months, and 40% at 24 months), whereas genetic factors played a significant role only in expressive vocabulary at 24 months (38%). For receptive vocab-

[15] Essentially the same results are obtained if the correlation coefficients that correspond to the median values of h^2 for Fischer 1973 and the WISC-R vocabulary subtest for Segal 1985 are used. Metaanalyses used Reznick et al.'s (1997) expressive vocabulary data for 24-month-old twins because these data were most like those of Dale et al. 2000 and Ganger et al. 1999.

ulary, shared environmental factors played a significant role at all three ages (34% at 14 months, 52% at 20 months, and 51% at 24 months), whereas genetic factors played a significant role only in receptive vocabulary at 14 months (28%). Analyses of Reznick et al.'s (1997) preferential looking data revealed that genetic factors did not play a significant role at any age and shared environmental factors played a significant role (22%) at 20 months, but not at 14 or 24 months. Thompson et al. (1991) estimated the genetic influence on school-age children's spoken vocabularies to be 54%, with shared environmental factors playing little role (8%) in spoken vocabulary.

6.4. THE ROLE OF NONVERBAL COGNITIVE ABILITIES ON SPOKEN VOCABULARY. It is possible that genetic factors are responsible for learning lexical items, but that these genetic factors are related to general cognitive abilities and not to specifically linguistic abilities. Using the Cholesky modeling procedure, Reznick et al. (1997) determined that the most parsimonious model for 14-month-old twins revealed a genetic factor that influenced nonverbal abilities and receptive vocabulary (genetic correlation, $R_G = .38$). In addition, there was a distinct genetic factor that was specific to receptive vocabulary. For 20-month-old twins, there was a genetic factor that influenced nonverbal performance and a distinct genetic factor that influenced both expressive and receptive vocabulary $R_G = .77$), but not nonverbal abilities. However, at 24 months of age, a single genetic factor affected twins' nonverbal abilities and expressive and receptive vocabulary (nonverbal and expressive vocabulary $R_G = .61$; nonverbal and receptive vocabulary $R_G = .89$; expressive and receptive vocabulary $R_G = .82$). In contrast with these results, using the same type of analyses, Price et al. (2000) found that for the 2-year-old TEDS twins, only 21% of the variance in expressive vocabulary size was explained by scores on the PARCA nonverbal cognitive measure, and the R_G between nonverbal and expressive vocabulary measures was only .30. This finding suggests that the genetic factors that influence nonverbal abilities and spoken vocabulary do not, for the most part, overlap. Robinson (1999) analyzed the verbal (mainly vocabulary) and nonverbal items of the Bayley Scales of Infant Development (Bayley 1969) data collected from 220 Louisville Twin Study twin pairs who were 18 and 24 months when the Bayley was administered. Univariate analyses revealed significant heritable effects for verbal abilities at 18 and 24 months ($h^2 = .33$ and .16, respectively) and for nonverbal ability at 18 months ($h^2 = .54$). Multivariate analyses of data collected at 18 months revealed that there were heritable factors unique to language and heritable factors that influenced both verbal and nonverbal abilities. At 24 months, a new genetic factor appeared that only accounted for variance in language scores. Robinson (1999) also found evidence of domain-specific genetic stability for verbal and nonverbal scores. Genetic factors that influenced language scores at 18 months influenced language scores at 24 months, and genetic factors that influenced nonverbal scores at 18 months influenced nonverbal scores at 24 months, whereas genetic factors for language scores at 18 months did not influence nonverbal scores at 24 months, nor did genetic factors for nonverbal scores at 18 months influence verbal scores at 24 months.

6.5. VOCABULARY TWIN STUDIES. Depending on the technique used to measure heritability and vocabulary and the age of the twins studied, between a quarter and a half of the variance in normal twins' spoken vocabulary is attributable to heritable factors. The developmental trends observed in the two longitudinal studies (Reznick et al. 1997, Robinson 1999) were consistent with the results of the other studies taken as a group. As children get older, heritable factors and nonshared environmental factors play an increasing role in spoken vocabulary, and shared environmental factors play a decreas-

ing role. Of the three studies that examined the specificity of the genetic factors that influence language, one (Dale et al. 2000) suggests that at 24 months there are genetic factors that influence expressive vocabulary size that are distinct from those genetic factors that influence both vocabulary and nonverbal abilities. The second study (Robinson 1999) suggests that there are both language-specific and general genetic factors at 18 months, and that another language-specific genetic factor emerges at 24 months. In contrast, the third study (Reznick et al. 1997) suggests that specific-to-language genetic factors play a role in vocabulary size at 14 and 20 months, but not at 24 months. Although the TEDS results suggest that genetic factors play a larger role in the vocabulary of slow word learners than most children, the genetic factors that influence vocabulary size in slow learners overlap completely with genetic factors that influence nonverbal ability, whereas for normal TEDS twins, there are genetic factors that are specific to language.

7. Phonology and articulation

7.1. Phoneme discrimination and phonological awareness. Five studies compared normal MZ and DZ twins' ability to perform various phonological tasks (see Table 6). Fischer (1973) compared normal 2- and 3-year-old MZ and DZ twins' ability to discriminate between minimally different phonemes as measured by a minimal pairs test in which children listen to a word (e.g. *goat*) and choose which of two pictures depicts the word where the words depicted are phonological minimal pairs (e.g. *goat* and *coat*). Fischer found no evidence that genetic factors played a significant role in phoneme discrimination ($h^2 = .22, Z < 1$), although this may reflect the small sample size (11 MZ and 10 DZ twin pairs) and the fact that both MZ and DZ twins did so well on the minimal pairs test that there was very little variance to be explained by any factors. Hohnen and Stevenson (1999) used a battery of tests to arrive at a composite measure of 6- and 7-year-old twins' phonological awareness. This battery included tests that assess children's ability to blend the individual phonemes of words together to form words (children hear /b/ /a/ /t/ and must say the word /bat/), to say a word when one of the phonemes is removed (children hear the word /sid/ and must pronounce the word when the /d/ is deleted). In addition, for the 7-year-old twins, the composite measure included a rhyme detection test in which children decide which of the words in a set does not rhyme (in the set *hen*, *ten*, *pen*, and *bed*, *bed* does not rhyme). Hohnen and Stevenson (1999) tested 32 sets of 6-year-old MZ twins and 28 sets of 6-year-old DZ twins. The MZ correlation for phonological awareness was significantly greater than the DZ correlation, $r_{mz} = .87$, $r_{dz} = .59$, $Z = 2.40$, $p < .01$, with 56% of 6-year-old twins' performance attributable to genetic factors, 31% attributable to shared environmental factors and 13% attributable to nonshared environmental factors. For the 34 sets of 7-year-old MZ twins and 32 sets of 7-year-old DZ twins, the MZ r was .92 and the DZ r was .53, $Z = 3.89$, $p < .0001$, with 78% of the variance in phonological awareness attributable to genetic factors, 14% attributable to shared environmental factors, and 8% due to nonshared environmental factors. Averaging together the phonological awareness data for 6- and 7-year-old twins, the weighted $\bar{r}_{mz}$ was .90 and the weighted $\bar{r}_{dz}$ was .56, $Z = 3.15$, $p < .001$, with heritable factors accounting for 68% of the variance in children's performance and shared environmental factors accounting for 22% of the variance. If we combine Hohnen and Stevenson's phonemic awareness data (1999) with Fischer's phonemic discrimination data (1973), heritable factors account for 65% of the variance in children's phonemic abilities ($Z = 2.48$, $p < .01$) and shared environmental factors account for 23% of the variance.

STUDY	TWIN PAIRS	AGE	TESTS	CORRELATIONS	FALCONER'S HERITABILITY ESTIMATE
Bishop et al. 1999	51 MZ (sex N/A) 49 DZ (sex N/A)	7–13 yrs $\bar{x}$ = 9.9	pure tone repetition nonword repetition	Pure tone r_{mz} = .60; r_{dz} = .49 Nonword r_{mz} = .64; r_{dz} = .29	Pure tone .22 Nonword .71**
Fischer 1973	11 MZ (3 M, 8 F) 10 DZ (4 M, 6 F)	3.5 yrs	phoneme discrimination (minimal pairs test)	r_{mz} = .64; r_{dz} = .53	.22
Hohnen & Stevenson 1999	6 yr: 32 MZ, 28 DZ 7 yr: 34 MZ, 32 DZ	$\bar{x}$ = 5.8 $\bar{x}$ = 7.0	phonological awareness composite score	6-yr-olds r_{mz} = .87; r_{dz} = .59 7-yr-olds r_{mz} = .92; r_{dz} = .53 OVERALL $\bar{r}_{Wmz}$ = .90; $\bar{r}_{Wdz}$ = .56	6-yr-olds .56** 7-yr-olds .78**** OVERALL .68***
Matheny & Bruggemann 1973	64 MZ (35 M, 29 F) 37 DZ (16 M, 21 F)	3–8 yrs	articulation test	Male r_{mz} = .84; r_{dz} = .56 Female r_{mz} = .90 MZ; r_{dz} = .83 OVERALL $\bar{r}_{Wmz}$ = .87; $\bar{r}_{Wdz}$ = .74 SES-M r_{mz} = .85; r_{dz} = .56 SES-F r_{mz} = .90; r_{dz} = .58 OVERALL-SES $\bar{r}_{Wmz}$ = .90; $\bar{r}_{Wdz}$ = .57	Male .56* Female .14 OVERALL .26 SES-M .58* SES-F .64** OVERALL-SES .66**
Mather & Black 1984	50 MZ (21 M, 29 F) 29 DZ (11 M, 18 F)	3–6 yrs $\bar{x}$ = 4.5	articulation test	r_{mz} = .96 MZ, r_{dz} = .92 DZ	.08

TABLE 6. Articulation & phonology.

Significance levels are for one-tailed tests: * $p < .05$; ** $p < .01$; *** $p < .001$, **** $p < .0001$

Hohnen and Stevenson (1999) also used the DF method to estimate the contributions of genetic and environmental factors on phonological awareness. For the 6-year-old twins, the DF estimates (52% genetics, 35% shared environment, 14% nonshared environment) were quite similar to estimates obtained using Falconer's method. For the 7-year-old twins, the DF method yielded a somewhat lower estimate of the effects of genetic factors (62% genetics, 28% shared environment, 10% nonshared environment) than Falconer's method (78% genetics, 14% shared environment, 8% nonshared environment). Hohnen and Stevenson also performed covariation analyses to determine the extent to which the genetic factors that affect phonological awareness are the same as those affecting nonverbal IQ. Cholesky decomposition revealed that genetic factors affecting nonverbal IQ accounted for 29% of the variance in phonological awareness for 6-year-old twins. Genetic factors that affect vocabulary and morphosyntax accounted for an additional 23% of the variance in phonological awareness, with genetic factors specific to phonological awareness accounting for a further 9% of the variance. Results were somewhat different for the 7-year-old twins: genetic factors affecting nonverbal IQ accounted for 18% of phonological awareness, with genetic factors affecting vocabulary and morphosyntax accounting for an additional 67% of the variance in phonological awareness. In contrast with the findings for the 6-year-old twins, genetic factors specific to phonological awareness explained none of the variance in phonological awareness observed for the 7-year-old twins.

7.2. Auditory and phonological short-term memory. Gathercole and colleagues have argued that individual differences in phonological short-term memory affect the rate of first and second language acquisition among normally developing children (Adams & Gathercole 1995, 1996, Gathercole & Baddeley 1990b, 1993, Gathercole & Adams 1993, Gathercole & Pickering 1999, Gathercole et al. 1992, 1997, 1999, Masoura & Gathercole 1999). Bishop et al. (1999) tested 51 MZ and 49 DZ unselected (i.e. normal) twin pairs' short-term memory for sequences of pure tones (Tallal & Piercy 1973a,b) and 2- to 5-syllable-long nonsense words (Gathercole et al. 1994). Although heritable factors did not significantly influence children's ability to repeat sequences of tones ($h^2 = .22$, $Z < 1$), heritable factors accounted for about three-quarters of the variance in normal twins' ability to repeat nonsense words ($h^2 = .71$, $Z = 2.26$, $p = .01$).

7.3. Articulation. Although there are a number of twin studies that investigate articulation abilities, to date there are no twin studies of normal phonological development. Four studies (Dixon et al. 1995, Locke & Mather 1989, Matheny & Bruggemann 1973, Mather & Black 1984) compared normal MZ and DZ twins' articulation abilities as measured by the Templin-Darley Screening Test of Articulation (1969). This test consists of a list of words designed to determine whether children can produce American English consonants correctly. Dixon et al. (1995) analyzed the articulation scores of 256 twins and 124 of their nontwin siblings and via factor analysis came up with scores for five articulation factors. These scores were then analyzed using multiple regression techniques. The results of these analyses suggest that articulation of the phoneme /r/ is largely the result of genetic factors, whereas environmental factors play a greater role in the articulation of the phonemes /l/, /w/, and /j/. Matheny and Bruggemann (1973) found that for the male twins they studied, the MZ correlation for articulation was significantly greater than the DZ correlation, regardless of whether socioeconomic status (SES) was controlled for, with heritable factors accounting for 56% of the variance in non-SES adjusted scores and 58% of the variance in SES-adjusted scores. For

female twins, the MZ correlation was only significantly greater than the DZ correlation when SES was controlled for. For non-SES adjusted scores, only 14% of the variance in girls' articulation was attributable to heritable factors, whereas 64% of the variance in girls' SES-adjusted articulation scores was attributable to genetic factors.[16] Matheny and Bruggemann (1973) also found that the MZ correlation (boys' and girls' scores combined) was significantly greater than the correlations for pairs of nontwin siblings, MZ-nontwin sibling pairs, and DZ-nontwin sibling pairs (all p's $< .05$). In Mather & Black 1984, MZ and DZ correlations were extremely high (.96 and .92, respectively), and only marginally different from one another ($Z = 1.46, p = .07$). Locke and Mather (1989) reanalyzed the articulation data for 13 pairs of age-matched and sex-matched MZ and DZ twins in Mather and Black's (1984) original articulation study. Although these 26 twin pairs were not considered language impaired, Locke and Mather (1989) selected them because at least one member of each twin pair made frequent errors ($>$ 15%) on the articulation screening test. MZ co-twins were no more likely than DZ co-twins to mispronounce a word in exactly the same way (Locke & Mather 1989). MZ co-twins, however, were significantly more likely to mispronounce the same target words than DZ co-twins (rates of shared errors were 82% for MZ twins and 56% for DZ twins).[17] Children who shared neither genetic nor family environment (but were matched for age, sex, race, dialect, and SES) were significantly less likely to have shared errors than were MZ twins, but they were not significantly less likely to have shared errors than DZ twins (Locke & Mather 1989).

As shown in Table 6, a total of 114 MZ and 66 DZ pairs of normal twins were assessed by Matheny and Bruggemann (1973) and Mather and Black (1984). For articulation scores not adjusted for SES, the unweighted $\bar{r}_{mz}$ was .90 and the unweighted $\bar{r}_{dz}$ was .77 ($t(2) = 1.72, p = .28$). The $\bar{r}_w$ was .92 for MZ twins and .85 for DZ twins ($Z = 1.28, p = .10$), with only 15% of children's performance attributable to genetic factors and 77% attributable to shared environmental factors. When Matheny and Bruggemann's SES-adjusted articulation scores were used, the unweighted $\bar{r}_{mz}$ was .90 and the unweighted $\bar{r}_{dz}$ was .69 ($t(2) = 2.44, p = .14$). The weighted $\bar{r}_w$ was .93 for MZ twins and .79 for DZ twins ($Z = 1.90, p = .03$), with 26% of performance attributable to genetic factors and 66% attributable to shared environmental factors.

7.4. PHONOLOGY AND ARTICULATION TWIN STUDIES. Family aggregation and pedigree studies suggest that phonological impairments may be heritable (see Stromswold 1998). Consistent with this, Bishop and colleagues report that genetic factors play a large role in SLI twins' articulation abilities and phonological short-term memories (Bishop et al. 1995, 1996, 1999), but not in their short-term memories for sequences of pure tones (Bishop et al. 1999). For normally developing children, heritable factors account for about two-thirds of the variance in phonemic awareness and phonological short term memory, but less than a quarter of the variance in phoneme discrimination or tone repetition. Hohnen and Stevenson's (1999) findings indicate that some of the genetic factors that influence normal children's phonological awareness are specific to language and some are not, but specific-to-phonology genes play little or no role in phonological

[16] SES is an important factor because children's pronunciations were judged against standard American English dialect.

[17] Overall, DZ twins performed significantly better than MZ twins (71% and 55% respectively). However, the lack of a significant correlation between number of errors and number of shared errors suggests that the greater similarity of MZ twin pairs is not merely the result of there being more opportunities for shared errors among MZ twins.

awareness. Genetic factors play a modest, but significant role in normal twins' articulation abilities, accounting for about a quarter of children's performance on articulation tests. However, as a *Language* referee pointed out to me, one cannot tell from these studies whether what is being measured is the role genetic factors play in motor skills generally, oral motor skills generally (i.e. genetic factors that influence behaviors such as chewing), or specifically linguistic motor skills.

8. Morphology and syntax

8.1. Twin studies of morphosyntax. Twelve studies (shown in Table 7) assessed aspects of normal twins' language other than simple vocabulary, phonological awareness/discrimination, articulation, reading, or writing (Chubrich 1971, Dale et al. 2000, Fischer 1973, Ganger 1998, Ganger et al. 1999, Hohnen & Stevenson 1999, Koch 1966, Mather & Black 1984, Mittler 1969, Munsinger & Douglass 1976, Segal 1985, Thompson et al. 1991). Unfortunately, metaanalyses cannot be performed on the results of these studies because the tests used and the aspects of language assessed are too diverse. Therefore the results of each study will be reviewed individually, starting with studies that examined morphosyntactic abilities of very young children.

Ganger (1998) investigated the heritability of morphosyntax via analyses of bimonthly speech samples collected from four sets of MZ twins and four sets of DZ twins who were not part of the diary study. Despite the small sample size, for rate of correct tense- and agreement-marking, the mean MZ correlation was significantly greater than the mean DZ correlation for both first order correlations ($\bar{r}_{mz} = .83$; $\bar{r}_{dz} = .44$) and for correlations when age was partialled out ($\bar{r}_{mz} = .69$; $\bar{r}_{dz} = .14$). For mean length of children's utterances (MLU) in morphemes, the MZ-DZ differences were not significant for either first order or partial correlations. Ganger (1998) also studied the heritability of morphosyntactic abilities using information that parents recorded in language diaries. (Different twins were used in the spontaneous speech study and the language diary study.) For a study of past tense acquisition, at least three times a week for an hour-long period, twins' parents wrote down all the sentences their twins said that referred to something that had already happened. For the 15 sets of MZ twins and 9 sets of DZ twins who overregularized irregular past tense verbs (e.g. **eated* for *ate*) for more than a few weeks, the MZ correlation for overregularization rate was significantly greater than the DZ correlation ($r_{mz} = .97$; $r_{dz} = .75$, $Z = 2.24$, $p = .013$), with DF and Falconer's estimates of .44 for heritable factors and .53 for shared environment factors.[18] For regular past tense rates (i.e. overregularized forms excluded), the MZ and DZ correlations were not significantly different ($r_{mz} = .97$; $r_{dz} = .95$, $Z < 1$), and shared environment factors accounted for most of the variance in past tense rates. The DF method yielded a nonsignificant estimate of heritability of .05 and a significant shared environment estimate of .93.

At least three times a week, parents wrote down all of the multiword sentences their twins produced in an hour. Ganger et al. (1999) used these diaries to determine the age at which members of 33 MZ twin pairs and 23 DZ twin pairs first used productive word combinations. The correlation coefficients were .90 for MZ twins and .50 for DZ twins. Falconer's and DF estimates indicate that genetic factors account for 81% of the variance in age of productive word combinations, with shared and nonshared environmental factors accounting for only 9 and 10% of the variance, respectively. Ganger

[18] Ganger (1998) also compared MZ and DZ correlations for age of first overregularization. However, Ganger (1998:127–28) argues that the age of first overregularization data are unreliable.

STUDY	TWIN PAIRS	AGE	TESTS AND MEASURES	CORRELATION	FALCONER'S HERITABILITY ESTIMATE
Ganger 1998	15 MZ; 9 DZ 16 MZ, 10 DZ	2.5–3.5 yrs	overregularization & past tense rates (diary study)	overreg $r_{mz} = .97$; $r_{dz} = .75$ past tense $r_{mz} = .97$; $r_{dz} = .95$	overreg .44* past tense .04
Ganger et al. 1999	33 MZ (12M, 21 F) 23 DZ (10M, 13 F)	$\bar{x} = 20$ months	age of first productive word combinations (diary study)	$r_{mz} = .90$; $r_{dz} = .50$	.81***
Ganger 1998	4 MZ, 4 DZ	2.5–3.5 yrs	tense/agreement & MLU (spontaneous speech)	Tense: $\bar{r}_{mz\cdot} = .83$; $\bar{r}_{dz} = .69$ MLU: $\bar{r}_{mz\cdot} = .80$; $\bar{r}_{dz} = .44$	Tense .28* MLU .26
Dale et al. 2000	1008 MZ; 959 DZ	2 yrs	parent report of syntax	$r_{mz} = .85$; $r_{dz} = .65$	.40***
Fischer 1973	11 MZ (3 M, 8 F) 10 DZ (4 M, 6 F)	2.5–3.5 yrs	repetition tests morphology tests grammar comprehension analyses of spontaneous speech	MZ-DZ significant for 4 of 10 repetition measures. MZ-DZ insignificant for both morphology tests MZ-DZ significant for grammar comprehension MZ-DZ significant for 1 of 4 speech analysis measures	repetition $\bar{h}^2 = .44$ morphology $\bar{h}^2 = -.58$ grammar comp 1.52* speech analysis $\bar{h}^2 = .82$ OVERALL $\bar{h}^2 = .55$
Mittler 1969	28 MZ (17 M, 11 F) 33 DZ (16 M, 17 F)	4 yrs	ITPA (total score & grammatical closure, verbal expression, vocal association, and comprehension subtests)	TOTAL $r_{mz} = .90$; $r_{dz} = .62$ grammar $r_{mz} = .82$; $r_{dz} = .59$ verbal $r_{mz} = .63$; $r_{dz} = .35$ assoc $r_{mz} = .81$; $r_{dz} = .63$ compreh $r_{mz} = .52$; $r_{dz} = .72$	TOTAL .56** grammar .46* verbal .56 assoc .36 compreh −.38
Mather & Black 1984	50 MZ (21 M, 29 F) 29 DZ (11 M, 18 F)	3–6 yrs $\bar{x} = 4.5$	Berko morphology ITPA grammatical closure ITPA verbal expression	Berko $r_{mz} = .97$; $r_{dz} = .92$ grammar $r_{mz} = .68$; $r_{dz} = .40$* verbal $r_{mz} = .53$; $r_{dz} = .36$	Berko .10* grammar .56* verbal .34
Koch 1966	33 MZ 30 DZ	6 yrs	analysis of grammatical errors	MZ twins errors more similar in type & frequency than DZ twins	No available information
Hohnen & Stevenson 1999	6 yr 32 MZ, 28 DZ 7 yr 34 MZ, 32 DZ	$\bar{x} = 5.8$ $\bar{x} = 7.0$	composite vocabulary and morphosyntax	6-yr-olds $r_{mz} = .80$; $r_{dz} = .50$ 7-yr-olds $r_{mz} = .90$; $r_{dz} = .59$	6-yr-olds .60* 7-yr-olds .62***
Segal 1985	70 MZ (33 M, 37 F) 35 DZ (22 M, 13 F)	5–13 yrs $\bar{x} = 8.0$	general language comprehension test	$r_{mz} = .65$; $r_{dz} = .43$	.44
Munsinger & Douglass 1976	37 MZ (14 M, 23 F) 37 DZ (16 M, 21 F)	$\bar{x}_{mz} = 8$ $\bar{x}_{dz} = 10$	composite expressive & receptive morphosyntax	$r_{mz} = .83$; $r_{dz} = .44$	.79**
Thompson et al. 1991	146 MZ; 132 DZ	6–12 yrs $\bar{x} = 9.8$	general language achievement test	$r_{mz} = .87$; $r_{dz} = .71$	.32***

TABLE 7. Morphosyntax.

All significance levels are for one-tailed tests: * $p < .05$; ** $p < .01$; *** $p < .001$
ITPA = Illinois Test of Psycholinguistic Abilities

et al. (1999) did not do bivariate analyses to determine if the same genetic factors influence acquisition of vocabulary and word combinations because there were only a small number of twin pairs for whom both measures were available and because the variance in vocabulary scores was very low. They argue, however, that even if the same genetic factors are responsible for both aspects of language, the greater heritability estimate for word combinations than for vocabulary indicates that genetic factors play a greater role in early syntactic development than early lexical development.

Dale et al. (2000) used a shortened form of the MCDI grammar checklist (Fenson et al. 1994) to assess almost 2000 TEDS twin pairs' grammatical development at age two. In the grammar checklist, parents were asked whether their child combined words. Parents were also given twelve pairs of sentences and were asked to choose which sentence in each pair sounded more like what their child would say (e.g. *baby crying, baby is crying*). From these data, Dale and colleagues calculated a grammatical development score for each child. The grammatical development correlations were .85 for MZ twins and .65 for DZ twins, with Falconer's estimates of 40% for genetic factors, 45% for shared environment factors and 15% for nonshared environment factors. DF analysis of these data yielded very similar results (grammatical development heritability = 39%, shared environment = 48%; specific environment = 13%). Bivariate analysis revealed that only 11% of the variance in TEDS twins' grammatical development scores was explained by their PARCA nonverbal scores. The R_G between nonverbal and grammar scores was only .29, which suggests that the genetic factors that influence vocabulary size and nonverbal ability are largely distinct. But the same genetic factors appear to influence acquisition of grammar and vocabulary items: bivariate analysis revealed that 28% of the variance in grammatical development was explained by vocabulary scores, with 69% of the variance due to shared environmental factors. However, the genetic correlation between grammar and vocabulary was .61. These results indicate that, although genetic factors play a modest role in the phenotypic correlation between grammar and vocabulary acquisition, to a large extent, the same genetic factors influence both (Dale et al. 2000).

Even though Dale et al. 2000 and Ganger et al. 1999 both used parent report measures to assess the grammatical abilities of 2-year-old twins, the heritability estimate for grammar in Dale et al. 2000 is half what Ganger et al. 1999 obtained. What accounts for this difference? On the one hand, Dale et al.'s study has 35 times more subjects than Ganger et al.'s study, and so the Dale group's heritability estimate is more likely to be statistically robust (i.e. if a different group of parents completed the same checklist, a very similar heritability estimate would be obtained). On the other hand, Ganger et al.'s productive word combination measure is almost certainly a more accurate reflection of children's syntactic development. In determining the age at which twins began to use multiword utterances, Ganger and colleagues counted only word combinations in which each word was used in at least one other multiword utterance (e.g. *what dat* was counted only if *what* was used in another multiword utterance, and *dat* was used in another multiword utterance). In Dale et al. 2000, parents simply reported whether or not their child combined words. It is likely that some parents said their children combined words even though all their multiword utterances were unanalyzed routines (e.g. *whatdat?*). Even more worrisome is Dale et al.'s reliance on parents' judgments of which sentences sound like sentences their child might say. It is extraordinarily unlikely that the children uttered any of these exact sentences in the checklist, so parents had to act as amateur developmental linguists, deciding which sentence in each pair was the more plausible one.

As part of her study of the genetics of different components of language, Fischer (1973) assessed 11 MZ and 10 DZ 2- and 3-year-old twins pairs' morphosyntactic abilities via a battery of tests. Although Fischer's Falconer's h^2's were reasonably large for many spontaneous speech measures, because her sample size was small, heritable factors played a significant role for only one spontaneous speech measure, the number of morphologically complex words (Falconer's $h^2 = 2.18$). For the four measures of spontaneous speech that might reasonably serve as indices of syntactic development (the number of correctly inflected verbs, the number of pronouns, the number of morphologically complex words, and MLU in words),[19] the mean r_{mz} was .51 and the mean r_{dz} was .10 (Falconer's $h^2 = .82$, $Z < 1$). Fischer also used the Osser et al. 1969 and Mehrabian 1970 repetition tests. Of the five measures used to assess children's repetitions, the number of insertions the children made was the only measure for which heritable factors played a significant role for both Ossler and Mehrabian tests (Falconer's $h^2 = 1.02$ and .84, respectively). For the Mehrabian repetition test, significant heritable effects were found for the number of deletions children made and the number of sentences children imitated perfectly (Falconer's $h^2 = .20$ and 1.12, respectively). For the ten repetition measurements, the mean r_{mz} was .77 and the mean r_{dz} was .55 (Falconer's $h^2 = .44$, $Z < 1$). Fischer found a significant genetic effect (Falconer's $h^2 = 1.52$) for the Harvard grammatical comprehension test (Fraser et al. 1963), but not for the Berko (1958) 'Wug' test of productive morphology, which tests children's ability to produce inflected forms of nonsense words (e.g. *one wug, two wug_*), or the Mehrabian 1970 test of morphology, which tests children's ability to produce inflected forms of real words. The fact that the different morphosyntactic tests and measures yielded such different results is worrisome and reflects either limitations of the tests or the small number of twins in Fischer's 1973 study. The small sample size also makes it impossible to do bivariate analyses to determine whether the same genetic factors play a role in twins' performance on the articulation, vocabulary, and morphosyntactic tests that Fischer administered.

Chubrich (1971) tested the longitudinal morphosyntactic development of 4 MZ twin pairs and 2 DZ twin pairs between the ages of 3.5 and 4.5 years of age. Every month for a year, Chubrich administered the Berko Wug test and a picture-matching comprehension test. Chubrich failed to find any significant MZ-DZ differences, although this is probably due to the small sample size and the monthly repetition of tests (which likely increased correlations for both MZ and DZ twins and, thus, may have obscured heritability effects).

Analyzing data from 28 pairs of 4-year-old MZ twins and 33 pairs of 4-year-old DZ twins, Mittler (1969) found a significant genetic effect on overall performance on the Illinois Test of Psycholinguistic Abilities (ITPA, Kirk et al. 1968) and on the ITPA grammatical closure subtest (Falconer's $h^2 = .56$ for both), but not on any of the other ITPA subtests.[20] As part of their larger study of the heritability of different aspects of

[19] The other two measures Fischer (1973) used (number of words and number of utterances) are not good candidates for measures of morphosyntactic development (see Brown 1973).

[20] The ITPA is based on Osgood's (1957) communication model and its nine subtests are designed to test the skills Osgood believed were necessary for communication. Some of the subtests do not tap linguistic abilities per se (e.g., nonverbal short term memory, ability to indicate gestures associated with common objects). Table 7 includes overall results and results for subtests that intuitively seem to tap linguistic abilities. The grammatical closure subtest is the ITPA subtest that most directly measures morphosyntactic abilities. In this subtest, children must complete sentences with the correct inflectional or derivational morphological form of a word (e.g., here is an apple, here are two—; this mother is writing a letter, this is the letter she—; this stick is long, this stick is even—). In the comprehension subtest, children are asked yes/no questions

language, Mather and Black (1984) assessed the morphosyntactic abilities of 50 MZ and 29 DZ twin pairs who were between the ages of 3 and 6 (mean age 4.5). Morphosyntactic abilities were assessed using the Berko Wug test and the grammatical closure and verbal expressions subtests of the ITPA. Genetic factors played a small but significant role in performance on Berko's morphology test (Falconer's $h^2 = .10, Z = 2.06, p = .02$). Genetic factors also significantly affected performance on the ITPA grammatical closure subtest (Falconer's $h^2 = .56, Z = 1.66, p = .05$), but not on the ITPA verbal expression subtest (Falconer's $h^2 = .34, Z < 1.0$).[21] Unfortunately, because Mather and Black (1984) did not test their subjects' nonverbal IQs, one cannot determine whether the genetic factors that influence performance on language tests are distinct from those that affect general (nonverbal) cognitive ability. Furthermore, they do not provide the information necessary to do bivariate analyses to determine whether the same genetic factors affect articulation, vocabulary, and morphosyntactic performance.

Koch (1966) analyzed the spontaneous speech of 6-year-old twins. For the 33 MZ and 30 DZ twin pairs included in this study, Koch concluded MZ twins were more similar than DZ twins in terms of the types and frequency of morphosyntactic errors. Unfortunately, Koch did not provide the information necessary to determine whether these MZ-DZ differences were statistically significant.

As part of their study of reading abilities of normal 6- and 7-year-old twins, Hohnen and Stevenson (1999) developed a composite measure of vocabulary and syntactic abilities. This composite measure included scores on Bishop's (1983) Test for Reception of Grammar, the British Picture Vocabulary Scale (a receptive vocabulary test), the WISC-R Vocabulary subtest (in which children define words), the WISC-R similarities subtest (in which children say how words are similar), and a test in which children repeat a story and their retellings are scored for sentence length and information content. For 6 year olds, the r_{mz} was .80 and the r_{dz} was .50, and, thus according to Falconer's method 60% of the variance in their performance was attributable to heritable factors, and 20% was attributable to shared environmental factors. Compared to Falconer's estimates, DF analysis yielded a somewhat lower estimate of heritability (43%) and a somewhat higher estimate of shared environment effects (35%). Cholesky decomposition analyses of 6-year-old twins' data revealed that genetic factors affecting nonverbal IQ accounted for 45% of the variance for the composite vocabulary/syntax measure, with genetic factors specific to vocabulary/morphosyntax accounting for 31% of performance, and nonshared environmental factors specific to morphosyntax making up the balance. The picture was quite similar for Hohnen and Stevenson's (1999) 7-year-old twins. The r_{mz} was .90 and the r_{dz} was .59, with Falconer's estimates of .62 for genetic factors and .28 for shared environmental factors. DF analysis again yielded a somewhat lower estimate for genetic factors (.50) and a somewhat higher estimate for shared environment factors (.39) than Falconer's estimates. Cholesky decomposition analyses of 7-year-old twins' data revealed that genetic factors affecting nonverbal IQ accounted for 42% of variance in vocabulary/syntax scores, with 18% attributable to genetic factors specific to vocabulary/morphosyntax, 29% attributable to shared environmental

such as 'Do apples fly?'. The association subtest is a verbal analogies subtest (e.g. 'I sit on a chair; I sleep on a—'). The verbal expressions subtest requires children to describe a series of simple objects.

[21] Mather and Black (1984) also measured the complexity of twins' spontaneous speech using Lee's (1974) Developmental Sentence Score (DSS) and Brown's (1973) mean length of utterance (MLU) in morphemes. However, because less than half of the twins produced at least 50 sentences as required for DSS or MLU measures, and Mather and Black do not say what the MZ and DZ sample sizes were for these measures, one cannot tell whether genetic factors played a significant role on these measures.

factors specific to vocabulary/morphosyntax and 12% attributable to nonshared environmental factors specific to vocabulary/morphosyntax.

In a study of 70 MZ and 35 DZ twin pairs who were between the ages of 5 and 13 years old (mean age 8 years), Segal (1985) found that genetic factors did not play a role in general language comprehension as measured by the WISC-R comprehension subtest. Munsinger and Douglass (1976) used the Assessment of Children's Language Comprehension test (Foster et al. 1972) and the Northwestern Syntax Screening Test (Lee 1971) to assess the comprehension and production of morphosyntax of 37 MZ twin pairs and 37 DZ twin pairs. Because the MZ twins were, on average, two years younger than the DZ twins (mean ages 8 and 10 years, respectively), Munsinger and Douglass (1976) divided each child's language score by his chronological age. In order that heritability estimates for language not be confounded with nonverbal IQ, Munsinger and Douglass did a regression analysis to partial out the effect of nonverbal IQ (as measured by the WISC-R picture completion subtest) on the combined score of the two tests. Genetic factors played a significant role on the age- and nonverbal IQ-adjusted scores on the combined tests (Falconer's $h^2 = .79$, $Z = 2.95$, $p = .002$), with shared environmental factors accounting for only 5% of the variance. In a study of 146 MZ and 132 DZ twin pairs between the ages of 6 and 12 (mean age 9.8 years), Thompson et al. (1991) found that, on a test of general language achievement (the Metropolitan Language Achievement Test, Prescott et al. 1986), genetic factors accounted for 32% ($p < .001$) of the variance in twins' performance and shared environmental factors accounted for 55% of the variance.

8.2. MORPHOSYNTACTIC RESULTS. Despite the relative paucity of large-scale twin studies of morphological and syntactic abilities and the impossibility of obtaining an overall heritability estimate for morphosyntax, the results of existing studies suggest that genetic factors play a role in children's comprehension and production of syntax and morphology. The MZ correlation was greater than the DZ correlation for 33 of 36 morphosyntactic measures ($p < .0001$ by sign test). (The three exceptions were Fischer's two morphology tests and Mittler's ITPA comprehension subtest.) The MZ correlation was significantly greater than the DZ correlation for all of the measures used in five studies, two-thirds of the measures used in one study, and half of the measures used in two studies. There were only four studies in which the MZ-DZ differences were not significant for the majority of the measures used. Furthermore, in all twelve studies reviewed, the MZ correlation (or the mean correlation for studies that reported more than one measure) was greater than the DZ correlation ($p < .0001$ by sign test).

A number of observations can be made about the studies reviewed. First, they are quite heterogeneous in the tests performed and the results obtained. Even within a study, tests that were designed to assess the same aspect of language sometimes yielded different results. Of the 17 morphosyntactic tests and measures used by Fischer (1973), for example, 6 revealed significant MZ-DZ differences and 11 did not. Similarly, of the three morphosyntactic tests administered by Mather and Black (1984), only two (the ITPA Grammatical Closure subtest and the Berko Wug test) yielded significant MZ-DZ differences. My second observation is that, in general, studies with large numbers of twin pairs were more likely to report significant MZ-DZ differences than studies including relatively few twin pairs. With the exception of Segal 1985, all of the studies that had at least fifty twin pairs found significant genetic effects. The effect of sample size can also be seen by comparing the results that different studies obtained for the Berko Wug test. Although Chubrich (1971), Fischer (1973), and Mather and Black

(1984) all gave this test to preschool-age children, only Mather and Black found significant MZ-DZ differences. This almost certainly reflects the fact that Mather and Black's study had 3.5 times as many twin pairs as Fischer's study and 6 times as many twin pairs as Chubrich's study. The third observation is that MZ-DZ differences were more likely to be significant for those tests and measures that clearly tap morphosyntactic abilities. For example, Mittler (1969) and Mather and Black both found significant MZ-DZ differences for the ITPA grammatical closure subtest but not for other ITPA subtests. Perhaps the reason Segal (1985) failed to find a significant MZ-DZ difference on the WISC-R comprehension subtest even though her study included over one hundred twin pairs is that morphosyntactic knowledge is not required to answer two-thirds of the items on this test.

Three studies performed analyses that help answer the question of how specific the genetic factors that influence morphosyntactic are. Munsinger and Douglass (1976) found that even when nonverbal IQ was partialled out, genetic factors played a significant role in performance on morphosyntactic tests, suggesting that there are genetic factors for morphosyntax above and beyond the genetic factors that influence general nonverbal ability. The bivariate analyses Dale and colleagues (2000) performed on the TEDS data from 24-month-old twins suggest that the genetic factors that influence syntactic development are largely different from those that influence nonverbal cognitive ability, but that the genetic factors influencing syntactic development are largely the same as those that affect vocabulary development. However, the high genetic correlation for vocabulary and grammar could reflect problems with the way lexical and grammatical development was measured in the TEDS study. The bivariate and Cholesky analyses that Hohnen and Stevenson (1999) performed on 6- and 7-year-old twins' data suggest that although genetic factors that influence performance on nonverbal cognitive tasks have a large effect on children's composite vocabulary/morphosyntax score (between 40 and 45% of the variance being explained by such factors), genetic factors specific to vocabulary/morphosyntax play a moderate role in children's vocabulary/syntax scores (accounting for between 20 and 30% of the variance). Furthermore, Hohnen and Stevenson (1999) found that, in addition to the genetic factors that influence nonverbal IQ and vocabulary/morphosyntax, there are genetic factors that specifically influence phonological awareness (in 6-year-olds, but not 7-year-olds).

Based on the results of existing twin studies, genetic factors do not seem to play a greater role in morphosyntactic abilities of older children, but this may reflect the relative paucity of large twin studies of older children that include 'pure' measures of morphosyntax. One of the goals of the TEDS study is to compare the role that genetic factors play at age 2, 3, and 4. Thus, if the TEDS project includes measures sensitive to aspects of morphosyntax that develop at ages 3 and 4, then future results from the TEDS project will help determine whether genetic factors have a greater influence on morphosyntactic ability at some ages than at others.

9. Written language

9.1. The relationship between written and spoken language. There is a growing consensus that at least some reading disabilities are language-based (see Scarborough 1990, Scarborough & Dobrich 1990, Stanovich 1988, and references therein). Prospective studies of kindergarten and preschool-age children later diagnosed as dyslexic have revealed that some dyslexic children exhibit weaknesses in phonemic awareness (Scarborough 1990, 1991a, Share et al. 1984, Stanovich et al. 1984), phonological production (Scarborough 1990, Silva et al. 1985), receptive and expressive vocabulary

(Scarborough 1990, Share et al. 1984, Stanovich et al. 1984), syntactic comprehension and production (Scarborough 1990, 1991a, 1991b, Share et al. 1984), and syntactic awareness (Tunmer & Grieve 1984, Tunmer et al. 1988). In a follow-up study of 3-year-old children with spoken-language disorders, Silva et al. (1987) found that such children frequently had reading difficulties later in life. In another follow-up study of preschool-age children with spoken-language disorders, Aram et al. (1984) found that after ten years, although many of the children had 'outgrown' their spoken-language impairment, over 90% had impairments in reading, spelling, or mathematics. In retrospective studies, parents report that dyslexic children are more likely to have a history of spoken language impairments than are children without dyslexia (Ingram et al. 1970, Rutter & Yule 1975). Genetic studies also suggest that dyslexia and SLI are related. For example, in a study of 13 identical and 5 fraternal twins, Johnston et al. (1984) found that preschool (spoken) language skills were the best predictor of later reading ability, accounting for 33% of the variance in reading-age discrepancy. Studies have also revealed that children with developmental phonological disorders have significantly more nuclear and extended family members with dyslexia than do control children (Gilger et al. 1998, Lewis 1992, Lewis et al. 1989)

9.2. HERITABILITY ESTIMATES FOR READING. Five studies of normally developing twins contained the information necessary to calculate Falconer's estimates of heritability (Foch & Plomin 1980, Hohnen & Stevenson 1999, Osborne et al. 1968, Stevenson et al. 1987, Thompson et al. 1991). Osborne et al. 1968 and Foch & Plomin 1980 used tests that primarily assess children's ability to read words in isolation. Stevenson et al. 1987 used tests that assess ability to read words in isolation (Schonell reading test, Schonell & Schonell 1960) and to read prose (Neale reading tests, Neale 1967). Hohnen & Stevenson 1999 developed a composite literacy score that measured children's ability to read prose and words in isolation. For the seven year olds in their study, Hohnen and Stevenson's literacy measure also included tests of children's ability to read irregularly spelled words—*pint*, *answer*—and nonsense words—*kule*, *tocken*—and to detect the correct spelling of a word in a word-pseudohomophone pair—*room*/*rume*. As shown in Table 8, the MZ correlations were greater than the DZ correlation coefficients for all five studies and for all reading tests, with Falconer's h^2's ranging from .18 for the Neale Reading Accuracy test in Stevenson et al. 1987 to 1.53 for the Heim Self-judging Vocabulary test (Vandenberg 1965) in Osborne et al. 1968. In a metaanalysis that used the mean correlation coefficients for Osborne et al. (1968) and Stevenson et al. (1987), the unweighted $\bar{r}$ was .82 for MZ twins and .55 for DZ twins ($t(5) = 5.23$, $p = .003$), with heritable factors accounting for 53% of the variance in reading ability, and shared environment accounting for 28% of the variance. The heritability estimate based on weighted mean correlations was slightly less (the $\bar{r}_w$ was .86 for MZ twin pairs and .66 for DZ twin pairs, $Z = 2.84$, $p = .002$), with 41% of the variance in reading ability attributable to heritable factors, 45% attributable to shared environment factors, and 14% due to nonshared environment.[22]

Thompson et al. (1991), Hohnen & Stevenson (1999), and Light et al. (1998) also used the DF method to calculate heritability estimates. DF heritability estimates were .29 for the twins in Thompson et al. 1991, .60 for the 6-year-old twins and .59 for the 7-year-old twins in Hohnen and Stevenson's 1999 study. As part of the Colorado Twin

[22] When the MZ and DZ correlations that correspond to the median values of h^2 for Osborne et al. 1968 and Stevenson et al. 1987 were used, essentially the same results were obtained. For weighted metaanalyses, the $\bar{r}_w$ was .87 for MZ twins and .66 for DZ twins, $Z = 2.90$, $p = .002$, Falconer's $h^2 = .42$.

STUDY	TWIN PAIRS	AGE	TESTS	CORRELATION COEFFICIENTS	FALCONER'S HERITABILITY ESTIMATE
Hohnen & Stevenson 1999	6 yr 32 MZ, 28 DZ	$\bar{x} = 5.8$	composite reading score	6-yr-olds $r_{mz} = .95$; $r_{dz} = .71$	6-yr-olds .48***
	7 yr 34 MZ, 32 DZ	$\bar{x} = 7.0$		7-yr-olds $r_{mz} = .92$; $r_{dz} = .61$	7-yr-olds .62***
Foch & Plomin 1980	52 MZ (25 M, 29 F)	5–12 yrs	PIAT	$r_{mz} = .74$; $r_{dz} = .46$	.56*
	32 DZ (17 M, 16 F)	$\bar{x} = 7.6$			
Thompson et al. 1991	146 MZ, 132 DZ	6–12 yrs	Metropolitan Reading Achievement Test	$r_{mz} = .94$; $r_{dz} = .79$	.30****
		$\bar{x} = 9.8$			
Stevenson et al. 1987	97 MZ (45 M, 52 F)	13 yr old	Neale Accuracy	Neale Accuracy $r_{mz} = .62$; $r_{dz} = .53$	Neale Accuracy .18
	107 DZ (48 M, 59 F)		Neale Reading	Neale Comp $r_{mz} = .71$; $r_{dz} = .49$	Neale Comp .44**
	Schonell Reading		Comprehension Tests	Schonell Read $r_{mz} = .61$; $r_{dz} = .73$	Schonell Read .19
	96 MZ (45 M, 51 F)		Schonell Reading Test	MEAN $r_{mz} = .65$; $r_{dz} = .51$	MEAN .28
	106 DZ (48 M, 58 F)				
Osborne et al. 1968	33 MZ, 12 DZ	13–18 yrs	WR Vocabulary	WR Vocab $r_{mz} = .61$; $r_{dz} = .40$	WR Vocab .42
			Heim Self-judging Vocab	Self-judge $r_{mz} = .63$; $r_{dz} = -.13$	Self-judge 1.53*
				Heim Vocab $r_{mz} = .86$; $r_{dz} = .38$	Heim Vocab .96**
			Heim Vocabulary	MEAN $r_{mz} = .70$; $r_{dz} = .22$	MEAN .96**
WEIGHTED MEAN†	403 MZ, 342 DZ			$r_{mz} = .86$; $r_{dz} = .66$	.41**

TABLE 8. Reading.

Significance levels are for one-tailed tests: * $p < .05$; ** $p < .01$; *** $p < .001$, **** $p < .0001$

† includes Osborne et al. 1968 and Stevenson et al. 1987 mean data

WR Vocabulary = Wide Range Vocabulary Test, PIAT = Peabody Individual Achievement Test for Word Recognition

Study of Reading Disorders, Light and colleagues (1998) studied the reading abilities of 132 MZ and 91 DZ normal twin pairs. For phonologically based reading, genetic factors accounted for 68% of normal twins' performance (versus 52% for dyslexic twins). For overall reading ability, genetic factors accounted for 42% of normal twins' performance (versus 70% for dyslexic twins).

Two twin studies of normal reading ability include analyses that help answer the question of just how specific to reading the heritable factors that influence reading are. Stevenson et al. 1987 found that for both MZ and DZ twins, correlations for reading scores that were adjusted for IQ were lower than for nonadjusted scores, but that Falconer's h^2 were as high or higher for IQ-adjusted scores as for nonadjusted scores on the Neale and Schonell reading tests (IQ-adjusted Falconer's h^2: Neale Reading Accuracy = .18, Neale Reading Comprehension = .51, Schonell Reading = .73, mean h^2 = .33). The MZ-DZ difference for IQ-adjusted correlations was significant for the Neale comprehension test ($Z = 2.07$, $p = .019$), and marginally significant for the mean correlations for the reading tests ($Z = 1.45$, $p = .074$). In a more sophisticated set of analyses, Hohnen and Stevenson (1999) used Cholesky decomposition to determine the extent to which the genetic and environmental factors affecting literacy were general cognitive factors, general linguistic factors, factors associated with phonological awareness, or factors specific to literacy. For the 6-year-old twins, genetic factors affecting nonverbal IQ accounted for only 11% of the variance on the literacy index, with genetic factors specific to vocabulary/morphosyntax accounting for 17% of the variance, and genetic factors specific to reading accounting for 30% of the variance. Although genetic factors specific to phonological awareness did not influence literacy, 37% of reading performance was attributable to shared environmental factors that were specific to phonological awareness. The picture was almost exactly the same for the 7-year-old twins. Genetic factors affecting nonverbal IQ accounted for 12% of variance on the literacy index, with genetic factors specific to vocabulary/morphosyntax accounting for 31% of the variance, and genetic factors specific to reading accounting for 18% of the variance. Although genetic factors specific to phonological awareness did not influence literacy, 30% of 7-year-old twins' reading performance was attributable to shared environmental factors that were specific to phonological awareness.

9.3. TWIN STUDIES OF SPELLING. In addition to examining genetic influences on reading, Osborne et al. 1968 and Stevenson et al. 1987 studied the effects of heritable factors on normal children's spelling abilities. Osborne et al. 1968 assessed spelling abilities of 33 pairs of MZ twins and 12 pairs of DZ twins using the Heim Spelling Achievement Test (Vandenberg 1965). The correlation coefficients for spelling scores were .82 for MZ twins and .26 for DZ twins ($Z = 2.35$, $p = .009$, Falconer's h^2 = 1.12). For the 95 pairs of MZ twins and 106 pairs of DZ twins tested by Stevenson and colleagues, the correlation coefficents on the Schonell Spelling Test (Schonell & Schonell 1960) were .76 for the MZ twins and .50 for the DZ twins ($Z = 3.13$, p = .0009, Falconer's h^2 = .53). Combining the results of the two studies, the unweighted $\bar{r}$ was .79 for MZ twins and .38 for DZ twins. The $\bar{r}_w$ was .78 for MZ twins and .48 for DZ twins ($Z = 2.76$, $p = .003$, Falconer's h^2 = .60). The Stevenson study found that heritability estimates for IQ-adjusted spelling scores were higher than heritability estimates for nonadjusted scores (73% and 53%, respectively), suggesting the existence of heritable factors, but not general intelligence that influence spelling abilities.

9.4. NORMAL TWINS' WRITTEN LANGUAGE. Results of existing twin studies of reading and spelling indicate that heritable factors account for between a third and a half of

the variance in normal children's reading abilities and about two-thirds of their variance in spelling abilities. Although about 10% of the variance in reading is due to genetic factors that also affect nonverbal IQ, 20 to 30% of the variance is due to genetic factors that also influence morphosyntax and vocabulary, and 20 to 30% of the variance is attributable to genetic factors that are specific to reading. Genetic factors specific to phonological awareness explain none of the variance in reading ability for normal children (Hohnen & Stevenson 1999). Analyses of the Colorado twin study data (Light et al. 1998) indicate that, for phonologically based reading, genetic factors accounted for slightly more of the variance for normal twins than for dyslexic twins (68% versus 52%), whereas for overall reading ability, genetic factors accounted for less of the variance for normal twins than for dyslexic twins (42% versus 70%).

10. SUMMARY OF TWIN STUDY RESULTS

10.1. THE ROLE OF GENETICS IN NORMAL AND ABNORMAL LANGUAGE. The twin studies reviewed suggest that heritable factors are responsible for much of the variance in language-impaired people's linguistic abilities and some of the variance in normal people's linguistic abilities. For both written-language and spoken-language disorders, mean and overall concordance rates were approximately 30 percentage points higher for MZ twins than for DZ twins, with genetic factors accounting for between one-half and two-thirds of the written and spoken language abilities of language-impaired people. In studies of normal twins, depending on the aspect of language being tested, between one-quarter and one-half of the variance in linguistic performance was attributable to genetic factors. In Dale et al.'s (1998) TEDS study, genetic factors accounted for three times as much of the variance in vocabulary size for slow word-learners as for children who learn words at a normal rate. Similarly, Bishop et al. 1999 found that heritable factors accounted for more of the variance in nonsense-word repetition in SLI children than in normal children (100% versus 70%). Results of the Colorado Twin Study of Reading Disability (Light et al. 1998) indicate that genetic factors account for more of the variance in overall reading ability in dyslexic twins than in normal twins, but genetic factors account for slightly more of the variance in phonologically based reading for normal twins.

10.2. PROBABLE GENETIC FACTORS SPECIFIC TO LANGUAGE. Studying twin pairs in which at least one co-twin performed poorly on verbal tests, Eley et al. (2001, 1999), Olson (1994), Stevenson et al. (1987), and Tomblin and Buckwalter (1998) performed analyses that suggest that there are genetic factors specific to language. But Bishop et al. (1995) found that once they partialled out performance on a nonverbal test, genetic factors did not significantly influence their SLI subjects' performance on linguistic tasks (but see §5.3 and note 11). In a similar vein, Purcell et al. (2001) found that the same genetic factors accounted for much of the comorbidity of verbal and nonverbal delays in the TEDS subjects. However, as I noted in §5.4, the analyses Purcell and colleagues performed are not really the appropriate ones to test the genetic specificity of the deficits observed in SLI children.

A number of studies of normal twins suggest that there are language-specific genetic factors. For example, even when nonverbal IQ was partialled out, Munsinger and Douglass (1976) found that genetic factors played a significant role in performance on morphosyntactic tests, and Stevenson et al. (1987) found the same thing for reading and spelling. Using bivariate and Cholesky analyses, Hohnen and Stevenson (1999) found that language-specific genetic factors affected six- and seven-year-olds' performance on vocabulary/morphosyntax, phonological awareness, and reading tests. Performing

similar analyses, Dale et al. (2000), Price et al. (2000) and Robinson (1999) found that language-specific genetic factors affected two-year-olds' spoken-language development. In contrast to these findings, Reznick et al. (1997) found evidence of language-specific genetic factors at 20 months of age, but not at 14 or 24 months of age.

10.3. GENETIC FACTORS AFFECT MANY ASPECTS OF LANGUAGE. Studies of language-disordered twins indicate that genetic factors play a role in many, if not all, aspects of language. For example, MZ concordance rates were greater than DZ concordance rates for both written-language and spoken-language disorders. This suggests that genetic factors play a role in both types of language disorders. Bishop et al. (1995) found that genetic factors influenced their SLI twins' performance on phonological short-term memory, articulation, vocabulary, and morphosyntactic tests (although initial analyses suggest that the genetic factors involved may be related to general cognitive ability). In their study of dyslexic twins, Gayan and Olson (1999) found that genetic factors influenced phonemic awareness, phonemic decoding, and orthographic decoding. It is less clear whether the same genetic factors play a role in all aspects of language. Hohnen and Stevenson's (1999) study of normal 6- and 7-year-old twins found evidence of specific genetic factors for vocabulary/morphosyntax, phonological awareness, and reading. In contrast, the TEDS data from normal two-year-olds suggest that the same genetic factors affect lexical and syntactic development (Dale et al. 2000). The discrepancy in findings between Hohnen & Stevenson 1999 and Dale et al. 2000 could reflect differences in the ages of the children studied, as twin (and adoption) studies of older children often report higher heritability estimates. Alternatively, the discrepancy could reflect that Hohnen and Stevenson's measures of subcomponents of language were much better and more specific than the measures used by Dale and colleagues.

10.4. FUTURE STUDIES. Although there have been a number of reasonably large twin studies of verbal IQ (see note 13), vocabulary, and reading, large twin studies of phonology and morphosyntax are rare. Despite research that shows that the order in which normal children acquire grammatical morphemes and complex syntactic constructions is remarkably similar across children (§1), to date, only one large twin study (Dale et al. 2000) has investigated the heritability of morphosyntax, and this study used a rather crude parent-report measure to assess morphosyntactic development. Similarly, only a handful of studies (Ganger 1998, Koch 1966, Stromswold & Rifkin 1996, Stromswold & Sudhakar 1998) have investigated whether the types or rates of errors made by MZ twin pairs are more similar than those made by DZ twin pairs. The reason for the lack of such studies is quite simple: it is time-consuming to administer morphosyntactic tests to large numbers of twins, and it takes even longer to collect, transcribe, and analyze spontaneous speech samples from large numbers of twins.

One possible solution is to collect all existing transcripts of twins' speech and put them in a central location where they are accessible to all interested researchers. As a group, Chubrich (1971), Fischer (1973), Ganger (1998), Koch (1966), and Mather and Black (1984) collected speech samples from almost 100 pairs of MZ twins and 80 pairs of DZ twins. If transcripts of these samples are made publicly available (perhaps through the CHILDES system maintained by MacWhinney and colleagues), researchers would be able to conduct the same type of analyses of twins' language that have been conducted for singleton children acquiring English. By combining the speech samples collected in different studies, there should be enough statistical power to determine whether there are significant MZ-DZ differences for specific aspects of morphosyntax. (See Stromswold 1996 for a discussion of how to conduct statistical tests on transcript data.)

Another solution to the labor-intensive nature of morphosyntactic twin studies is to design tests or measures that can be administered by the parents of twins or, for older subjects, by the twins themselves. Given the success Saudino and colleagues (1998) have reported for the parent-administered PARCA test of nonverbal cognitive abilities, it seems reasonable to devise parent-administered morphosyntactic tests. Children's comprehension of specific aspects of syntax could be assessed using the forced-choice, picture-pointing methodology employed by standardized language tests such as the Test of Reception of Grammar (Bishop 1983), the Assessment of Children's Language Comprehension test (Foster et al. 1972) and the Northwestern Syntax Screening Test (Lee 1971). Children's comprehension of passives, for example, could be assessed by having children choose the picture that corresponds to semantically reversible passive sentences such as *the lion was chased by the tiger* where the options include pictures of a tiger chasing a lion and a lion chasing a tiger. Morphological competence for real words could be assessed using the methodology employed by the ITPA grammatical closure subtest (Kirk et al. 1968) and the Mehrabian (1970) morphology test. Berko's Wug test could be used to assess productive morphology. For older children and adults, it might be possible to design tests that assess aspects of language for which researchers have detected individual differences among adult speakers (see §1).

Most twin studies that purport to investigate the genetic basis of phonological abilities actually study articulation abilities. Furthermore, it is possible that these studies measure the heritability of general motor skills, rather than the heritability of motor skills specific to language. Future studies could determine if there are genetic factors specific to articulation by administering tests of general gross and fine motor skills, nonlinguistic oral motor skills, and articulation skills. Covariation analyses of these data would determine whether the same genetic factors influence all these skills. Given the studies that exist, it is not possible to determine the extent to which the same genetic factors (if they exist) are involved in phonological production and perception. This question could be addressed by assessing expressive and receptive phonological abilities in the same twins and determining the extent to which these skills covary.

If one wants to investigate the genetics of phonology rather than articulation, twin studies of normal phonological development are desperately needed. No such studies exist because the types of phonological transcriptions and phonological analyses necessary are very labor-intensive. Jennifer Ganger and I are currently analyzing the phonological development of the eight sets of twins from whom Ganger collected bimonthly spontaneous speech samples (Ganger 1998). Preliminary results suggest that MZ twins may be more similar in phonological development than DZ twins. Unfortunately, because Ganger was interested in the genetic basis of lexical and morphosyntactic development, she did not begin recording most of the twin pairs until they were 20 to 24 months old. Hence, it is not possible to use these speech samples to investigate whether heritable factors influence the earliest stages of phonological development.

11. Limitations of twin studies

11.1. Are heritability estimates inflated in twin studies? A frequently voiced concern is that the heritability estimates obtained from twin studies may be inflated by how twins are selected. If twins are located by recruiting volunteers or clinical patients or by doing searches of clinical records or published reports, this may bias the sample in favor of cases that are consistent with there being a genetic component to language: that is, in favor of MZ twin pairs whose language abilities are very similar and DZ twin pairs whose language abilities are quite different. But the finding that twin studies

generally report similar heritability estimates regardless of how twins were selected suggests that ascertainment biases do not affect heritability estimates too much.

A related concern is that heritability estimates may be inflated by the methods used to evaluate twins' language skills. Because researchers often know the zygosity of the twins in their study (through parent report or direct observation), if the same researchers assess both members of twin pairs they may inadvertently rate the language of MZ twin pairs to be more similar than that of DZ twin pairs. Studies that rely on parental report are also subject to this bias. Twin studies in which language disorders are diagnosed through test performance should be less subject to bias than studies in which diagnoses are based on clinicians' subjective judgments. The finding that the difference in MZ and DZ concordance rates for language disorders are similar regardless of the method used to diagnose twins suggests that heritability estimates are not unduly affected by unconscious biases on the part of researchers. Because MZ twins look more like one another than DZ twins, parents of MZ twins may rate the twins' language as being more similar than do parents of DZ twins. In addition, because MZ twins look more alike than DZ twins, parents of MZ twins may be more likely to confuse which twin said what. Analyses performed by Ganger (1998) suggest, however, that parents of MZ twins are no less accurate in their reports of their twins' language abilities than are parents of DZ twins.

Genetic factors seem to account for more of the linguistic variance among language-disabled people than among normal people. For this reason, heritability estimates for the general population might be inflated by the inclusion of a small number of language-impaired twins. One way to reduce this possibility would be to reanalyze the data from existing twin studies, being careful to exclude any twin pairs in which either twin performed below a certain criterion. For example, in Dale et al.'s 1998 study, one could obtain heritability estimates for those twin pairs in which both twins performed above the fifth percentile on the vocabulary measure.

11.2. ENVIRONMENTAL ASSUMPTIONS. Twin studies assume that MZ and DZ twins' environments are equally similar. But about two-thirds of MZ twins share a placenta, while no DZ twins do, so the prenatal environments of many MZ and DZ twins are not equally similar. Sharing a placenta may increase similarity between MZ co-twins because it means that MZ co-twins have exactly the same exposure to toxins and infections. Though sharing a placenta may also cause MZ twins to be less similar than they otherwise would be: for example, twins who share a placenta may undergo fetal-fetal transfusion in which blood is preferentially shunted from one twin to another. Indeed, on average, MZ twins' birth weights differ more than same-sex DZ twins' birth weights, and MZ twins generally suffer from more perinatal complications than DZ twins (Lenneberg 1967). Phillips (1993) has argued that the atypical gestation of MZ twins increases the rate of having one affected MZ co-twin, but Christensen et al. (1995) argue that this is not the case, at least among twins who survive to age 6. If Phillips is correct, group (disordered) heritability estimates will be lowered.[23]

[23] Maternal age and use of fertility drugs are correlated with one another, and with pre- and postnatal environmental factors. Women over 35 are at greater risk of delivering premature or low birth-weight babies. Older mothers are also more likely to use fertility drugs and to be better educated and wealthy. Because advanced maternal age and fertility drugs increase the risk of having DZ twins but not MZ twins, as a group then, MZ and DZ twins may be exposed to different pre- and postnatal environments. But there is no reason to believe that any of these factors result in MZ co-twins having more similar environments than DZ co-twins.

If MZ twins have more similar postnatal environments than DZ twins do (because, for example, people treat twins who look alike more similarly than they do twins who do not look alike), then heritability estimates of language may be inflated. Martin et al. (1997), however, have argued that this effect is generally very small for complex behaviors. And in a clever analysis, Munsinger and Douglass (1976) found that the language performance of MZ twins whose parents erroneously thought they were DZ twins was only slightly less similar than correctly classified MZ twins (r = .78 and .86, respectively), and the language of DZ twins whose parents thought they were MZ was only slightly more similar than correctly classified DZ twins (r = .47 and .40, respectively). For twins reared apart, MZ-DZ differences cannot be the result of MZ co-twins being treated more alike than DZ co-twins. Thus, one can investigate whether heritability estimates are inflated by MZ-DZ differences in postnatal environments by comparing heritability estimates obtained for twins reared together and twins reared apart. Unfortunately, the only study that compares the language abilities of twins reared together and twins reared apart is a study of middle-aged and elderly Swedish twins (mean age 65) who participated in the Swedish Adoption/Twin Study of Aging (SATSA, Pedersen et al. 1994).[24] In this study, 46 MZ twin pairs reared apart, 67 MZ twin pairs reared together, 100 DZ twin pairs reared apart, and 89 DZ twin pairs reared together were given a battery of tests of general and specific cognitive abilities. Three tests taped vocabulary skills (a verbal information test, a synonyms test, and an analogies test). For the verbal information test, 61% of the variance was due to genetic factors, with 37% of the genetic factors being unique to verbal information (i.e. genetic factors that did not influence IQ). For the synonyms test, 64% of the variance was due to genetic factors, with 18% being unique to synonyms. For the analogies test, which taps verbal and reasoning skills, 48% of the variance was due to genetic factors, with 29% being unique to analogies. Consistent with the findings for school-age twins reared together (§6), results of Pedersen et al.'s study indicate that genetic factors account for a little more than half of the variance in vocabulary for elderly twins reared apart. Consistent with Robinson's (1999) and Price et al.'s (2000) findings for 2-year-old twins reared together, the Pedersen study found evidence that vocabulary was influenced by specific-to-language genetic factors. There is no reason to believe that differences in MZ and DZ twins' environments undermine the results of twin studies.

11.3. GENETIC ASSUMPTIONS OF TWIN STUDIES. The assumption underlying all twin-based heritability estimates is that MZ twins share 100% of their genetic material, whereas DZ twins generally share only half of their genetic material (meaning that DZ co-twins are genetically no more similar than nontwin siblings). If some MZ twin pairs are not genetically identical, the role of heritable factors will be underestimated. MZ twins have appreciable rates of nonconcordance for a variety of traits that seem unlikely to be affected by environmental factors—whorls and peaks in finger prints, for example—which suggests that MZ twins may not be genetically identical. The single ovum involved in MZ twinning may split at different times; those that split early will produce MZ twins who are more biologically alike than MZ twins who are the products of late-splitting ovum. MZ twins may differ genetically because of spontaneous mutations that occur after the ova have split or because of different degrees of mosaicism in MZ

[24] Because there exist heritable neurodegenerative disorders (e.g. some forms of Alzheimer's disease, Huntington's disease), behavioral genetic studies of elderly twins may reveal the effects of a second set of genes that affect language. The elderly twins discussed in Pedersen et al. 1994 had no known neurodegenerative conditions.

twins.[25] The role of genetic factors may also be underestimated if DZ twins share more than 50% of their genetic material. The lower rate of graft-rejection between DZ twins than between nontwin siblings is sometimes taken as evidence that DZ twins are more genetically similar than nontwin siblings (Geschwind 1983). However, as Ganger (1998) suggests, this may result from DZ twins having developed more similar antibodies than nontwin siblings because DZ twins have more similar pre- and postnatal environments than nontwin siblings.

11.4. GENERALIZABILITY OF TWIN STUDIES OF LANGUAGE. Day (1932a,b) first compared the language abilities of twins and singletons and found that with respect to vocabulary, grammatical complexity of utterances, and mean length of responses, 2- to 5-year-old twins were linguistically delayed relative to singletons. Day also found that twins' impairment in language (mean language quotation = 68) was greater than their impairment in IQ (mean IQ = 91). Subsequent studies by Koch (1966), Mittler (1969, 1970, 1976), Conway et al. (1980), Rutter and Redshaw (1991), and Dale et al. (2000) have confirmed that twins' language development is slower than singletons'. Even when impaired twins are excluded, twins' language development appears to be about three months delayed relative to that of singletons (Hay & O'Brien 1983). Thus, even correcting for the shorter gestation of twins, twins' language development lags about two months behind that of singletons' language development.

The linguistic delay of twins relative to singletons is usually taken to reflect the fact that twins experience prenatal and postnatal hardships not experienced by singletons. Prenatal hardships of twinning include the fact that the average gestation for twins is 3–4 weeks shorter than that for singletons, and that full-term twins are considerably smaller (in weight, height, and head circumference) than full-term singleton infants. Premature and low birthweight babies are at increased risk for developmental language delays (Breslau et al. 1996, Breslau et al. 2000, Briscoe et al. 1998, Gerry Taylor et al. 1995, Hack et al. 1991, Whitfield et al. 1997). Postnatally, because twins have a sibling of the same age, twins usually receive less social and linguistic input from adults compared to that received by singletons (Stafford 1987, Tomasello et al. 1986). Mothers of twins engage their twins in fewer verbal exchanges than mothers of singletons (Conway et al. 1980, Lytton et al. 1977). Studying normally developing twins, Conway et al. (1980) found that differences in complexity and frequency of maternal speech accounted for more of the variance in twins' language development than differences in gestational age or birthweight. The effects of this type of postnatal deprivation may be seen in the results of Record et al. (1970) who report that twins whose cotwins died at or within a few months of birth have mean IQs that approach that of singleton children, whereas the mean IQ for twin pairs where both twins survived is five points lower than the mean for singletons. (For further discussion of environmental factors that affect twins, see Mohay et al. 1986 and Mogford 1993.)

There may be synergistic effects between genetic and environment factors, with children who are genetically at risk for developing language disorders being particularly sensitive to subtly impoverished linguistic environments. Bishop (1992) found lower rates of familiality of SLI in twin families than in families of singleton probands (22% versus 32%, respectively). She suggests that this difference, although statistically insignificant, may be real and may reflect the additional pre- and postnatal risks that twins experience relative to their singleton peers. Because of these additional environmental

[25] Mosaicism refers to the condition in which a person's cells have different genotypes. For example, some people with Down syndrome have an extra chromosome 21 in only a fraction of their cells.

risk factors, Bishop (1992) suggests that twin SLI populations may be etiologically more heterogeneous than nontwin SLI populations.

Although pre- and postnatal hardships appear to affect twins' language, there is no evidence that either MZ or DZ twins' language is affected differentially. Except as noted (Locke & Mather 1989), in the studies reviewed, the age- and IQ-adjusted language scores for MZ and DZ twins were not appreciably different. As long as environmental hardships affect MZ and DZ co-twins similarly (that is, as long as they do not cause MZ co-twins to be more similar than DZ co-twins, or vice versa), the logic of the twin design is unaffected and heritability estimates should not be affected. However, it may be that, given the same genotype, twins are more likely to be language impaired than singletons; that is, the probability that a genetic language disorder is expressed may be greater for twins than for singletons.

12. Adoption studies

12.1. Overview. Like twin studies, adoption studies can be used to investigate the relative importance of genetic and environmental factors for language development. If language has a large genetic component, adopted children's linguistic abilities should resemble that of their biological relatives more than that of their adopted relatives. If language is primarily due to environmental factors, adopted children's linguistic abilities should resemble those of their adopted relatives more than those of their biological relatives. By far the largest adoption study that has included investigations of language is the twenty-year longitudinal Colorado Adoption Project (CAP). All of the articles reviewed in this section analyze CAP data.

12.2. Normal language development. Thompson et al. (1985) used the Bayley Scales of Infant Development (Bayley 1969) to assess the general and specific cognitive abilities of 182 adopted and 164 nonadopted (control) children. Biological and adopted parents were given a battery of tests of general and specific cognitive abilities. At 12 months of age, infant-parent correlations were minimal for both adopted and biological parents. At 24 months, infant-parent correlations were significant for adopted and biological parents for general cognitive ability, but not specific cognitive abilities (Thompson et al. 1985). These results suggest that, in infancy, genetic and environmental factors affect general cognitive abilities but not specific cognitive abilities. Thompson and Plomin (1988) compared the Sequenced Inventory of Communication Development (SICD, Hedrick et al. 1975) data from 2- and 3-year-old CAP children. Adopted children's SICD scores at age 2 were significantly correlated with their biological siblings' SICD scores ($r = .29$) and IQ scores ($r = .31$). Their SICD scores at age 3, were significantly correlated with their biological siblings' IQ scores ($r = .40$), but not SICD scores. Adopted children's SICD scores were not significantly correlated with their adoptive siblings' SICD or IQs scores at either age. Adopted children's SICD scores at age 2 were significantly correlated with their adopted mothers' and fathers' IQ scores (both r's $= .13$). Adopted children's SICD scores at age 3 were significantly correlated with biological mothers' IQ scores ($r = .22$) and adopted mothers' verbal scores ($r = .20$). Taken together, the sibling and parent SICD results indicate that both genetic and environmental factors influence early language development.

Cyphers et al. (1989) compared the general cognitive abilities and verbal, spatial, perceptual speed, and memory abilities of 163 adopted and 142 nonadopted children at age 3, 4 and 7 with that of their biological and adopted parents. Using multivariate path analyses, they arrived at the most parsimonious model of the data. Over all, the magnitude of genetic influence increased from age 3 to age 7 for verbal abilities and general cognitive abilities, but not for other specific abilities. At age 3, genetic factors

did not play a significant role in general or specific cognitive abilities. At age 4, genetic factors played a significant role in general cognitive performance ($h = .43$), spatial performance ($h = .48$), and perceptual speed ($h = .48$), but not in verbal performance ($h = .16$). At age 7, genetic factors played a significant role for general cognitive ability ($h = .53$), verbal abilities ($h = .50$) and spatial abilities ($h = .50$), but not perceptual speed or memory. Environmental factors shared by parent and child played a significant role only in verbal ability, and even there the effect was small and found only at age 4 ($e = .11$).

Cardon et al. (1992) compared the results of specific and general cognitive tests performed on 196 adopted and 213 nonadopted children, 52 unrelated siblings of adopted probands and 68 natural siblings of the control children. Multivariate analysis revealed that at age 7 heritable factors associated with general intelligence affected verbal ability (.46) and that, in addition, there was a substantial heritable effect of verbal ability independent of IQ (.83), yielding an overall estimate of the heritability of the verbal abilities of .90 ($.90 = .46^2 + .83^2$). Nonshared environmental factors had no effect on verbal ability ($e = .00$), and shared environmental factors had little effect ($c = .10$). The verbal ability score was a composite of the scores on the WISC-R vocabulary test and a verbal fluency task. For WISC-R vocabulary, although genetic factors that also affected IQ had a substantial heritable effect (.69), genetic factors independent of IQ played no role (.00), yielding an overall h of .47. Nonshared environmental factors accounted for most of the remaining variance in vocabulary ($e = .46$). Genetic factors that were common with IQ had a substantial effect on verbal fluency (.54) and genetic factors independent of IQ played a modest role (.20), yielding an overall h of .33. Nonshared environmental factors played a greater role in verbal fluency than shared environmental factors ($e = .46$, $c = .20$).

Rice et al. 1986 used multivariate analyses to determine the genetic correlations among verbal, spatial, perceptual speed, and memory scores at age 4. Genetic correlations between verbal abilities and other cognitive abilities were generally high (verbal-spatial $R_G = .77$, verbal-perceptual $R_G = .57$, verbal-memory $R_G = .55$). Environmental correlations were generally modest (verbal-spatial $E_G = .29$, verbal-perceptual $E_G = .38$, verbal-memory $E_G = .32$). These results suggest that a common genetic factor plays a substantial role in specific cognitive abilities, whereas a common environmental factor plays less of a role in these specific cognitive abilities. Wadsworth et al. 1995 used multivariate analysis and Cholesky modeling techniques to analyze data from 90 pairs of adopted seven year olds and their biologically unrelated siblings and 100 pairs of nonadopted seven year olds and their biologically related siblings. Children were given the Kaufman (1975) verbal comprehension and perceptual organization tests, the PIAT reading recognition test (Dunn & Markwardt 1970) and the CAP mathematics achievement battery. Genetic factors accounted for about a quarter of performance on the language tests (verbal comprehension $h = .20$, reading $h = .30$). Shared environmental factors played a minimal role on these tests (verbal comprehension $c = .08$, reading $c = .13$). Performance on the verbal comprehension test was significantly correlated with performance on the three other tests (all rs between .32 and .40). The genetic correlations between verbal comprehension and the other measures were fairly large (verbal-reading $R_G = .80$, verbal-math $R_G = .57$, verbal-perceptual $R_G = .36$) indicating that much of the covariance between each pair of tests is due to genetic factors that are common to both tests. Although these data could be used to argue that the genetic factors that affect verbal performance are not specific to verbal tasks, Wadsworth et al. argue that the high genetic correlation between verbal comprehension

and mathematics reflects 'the verbal nature of the mathematics tests' (Wadsworth et al. 1995:72).

Alarcón et al. (1998) analyzed the data from 175 adopted twelve-year-olds and their adopted (175 mothers and fathers) and biological parents (175 mothers, 34 fathers). Heritability estimates obtained for the full model were .26 for verbal, .35 for spatial, .38 for perceptual speed, and .53 for memory. In the full model, for verbal and spatial scores, the phenotypic correlation was .45, the genetic correlation was .27, and the bivariate heritability was .18, which indicates that relatively little of the phenotypic similarity between verbal and spatial scores is due to common genetic factors. For verbal and perceptual speed scores, the phenotypic correlation was .41, the genetic correlation was .46, and the bivariate heritability was .35. For verbal and memory scores, the phenotypic correlation was .21, the genetic correlation was .34, and the bivariate heritability was .61. To test whether heritable factors played an equal role in verbal, spatial, perceptual, and memory abilities at 12 years of age, Alarcón et al. (1998) tested the goodness-of-fit of a model in which the same value of h was assigned for all traits. The data fit for the resulting model was not significantly worse than for the full model, which suggests that heritable factors affect all four traits equally. To test whether verbal, spatial, perceptual, and memory traits were genetically differentiated at 12 years of age, Alarcón and colleagues tested a model in which the same value of R_G was fit for all four abilities. The resulting model was not significantly worse than the full model, which suggests that one genetic factor influences all four abilities.

Alarcón et al. 1999 reports similar analyses on the data from 129 adopted 16 year olds and their adopted (129 mothers and fathers) and biological parents (129 mothers, 24 fathers) and 125 nonadopted 16-year-olds and their biological parents. Unlike testing conducted at earlier ages, the children and parents were given the same battery of tests, and scores on these tests were used to create composite verbal, spatial, perceptual speed, and memory scores. Multivariate analyses of these scores yielded h^2 estimates of .64 for verbal, .49 for spatial, .48 for perceptual speed, and .32 for memory. Heritability estimates for the best-fitting full model were somewhat higher (.80, .70, .70, and .56 for verbal, spatial, perceptual speed, and memory, respectively). Alarcón and colleagues argue that the greater heritability estimates at age 16 than at previous ages reflect the greater genetic stability of the measures as a function of age. (It makes sense, intuitively, that heritability estimates will be greatest when children and their parents are evaluated using the same tests.) In the full model, for verbal and spatial scores, the phenotypic correlation was .46, the genetic correlation was .79, and the bivariate heritability was .96, which indicates that most of the phenotypic similarity between verbal and spatial scores is due to common genetic factors. For verbal and perceptual speed scores, the phenotypic correlation was .50, the genetic correlation was .64, and the bivariate heritability was .71. For verbal and memory scores, the phenotypic correlation was .32, the genetic correlation was .43, and the bivariate heritability was .64. As was the case with the data collected when the children were 12 years of age, goodness-of-fit comparisons indicate that heritable factors played an equal role in verbal, spatial, perceptual and memory abilities at 16 years of age, and that one genetic factor influences all four abilities (Alarcón et al. 1999).

12.3. LANGUAGE DISORDERS. Felsenfeld and Plomin (1997) compared the speech of 156 adopted and nonadopted children from the CAP who were at varying risk for speech disorder based on parental history of speech impairment. They compared the speech of 16 adopted children who had an impaired biological parent, 19 adopted

children who had an impaired adoptive parent, 31 nonadopted children who had an impaired parent, and 90 adopted and nonadopted children who had no parental history of speech disorders. Examining the children's speech and language skills at age 7, they found that 25% of the children who had a genetic background of speech impairment had impaired speech or language, as compared to only 9% of children without such a history. Positive genetic background was the best predictor of speech/language disorders, whereas children's full-scale IQ and family environment were not significant predictors of speech/language disorders.

12.4. SUMMARY OF CAP RESULTS. Taken as a whole, the results of the Colorado Adoption Project indicate that for normal and impaired children, genetic factors affect language abilities more than shared environmental factors do. Consistent with the results of twin studies, the results of the CAP study indicate that the importance of genetic factors becomes more apparent as children get older, and that genetic factors influence verbal performance more than other specific cognitive abilities (see Fig. 1, taken from

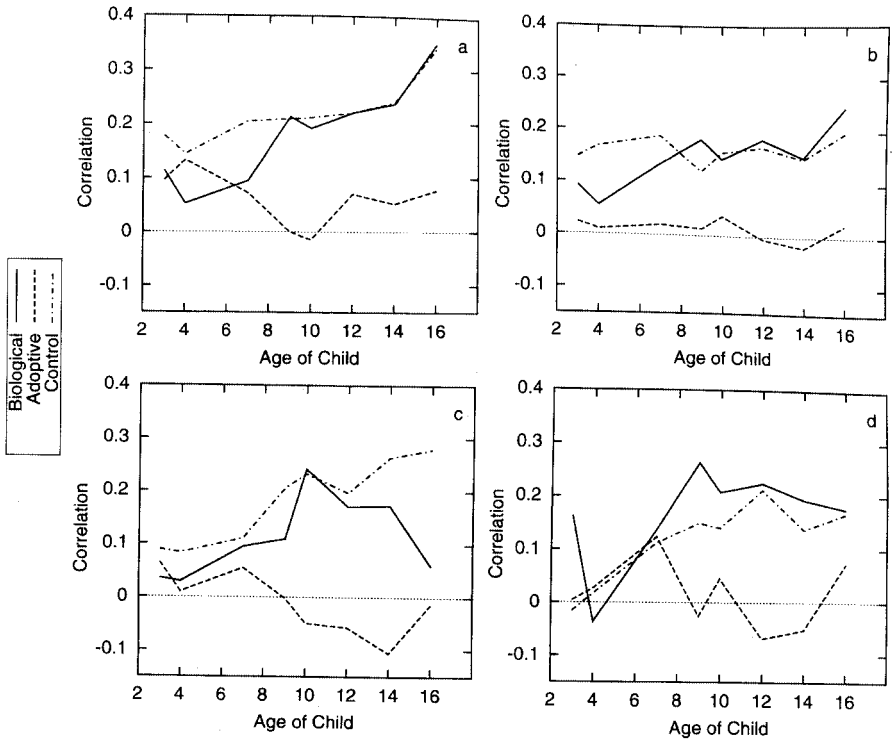

FIGURE 1.
Parent-offspring correlations for factor scores for specific cognitive abilities for adoptive, biological, and control parents and their children at 3, 4, 7, 9, 10, 12, 14, and 16 years. Correlations are shown for verbal ability (a), spatial ability (b), speed of processing (c), and recognition memory (d). Parent-offspring correlations are weighted averages for mothers and fathers. The *N*s range from 33 to 44 for biological fathers, 159 to 180 for biological mothers, 153 to 197 for adoptive parents, and 136 to 217 for control parents.

Plomin et al. 1997). How specific are the genes that affect verbal performance? At age 7, genetic factors that are independent of IQ play a substantial role in verbal performance. However, analyses of CAP data obtained before and after the age of 7 suggest that the same genes that affect verbal ability also affect IQ. What does this mean? Alarcón et

al. (1999) propose that domain-specific genetic variance may have a greater influence during the transition from early to middle childhood than at other developmental stages, although they do not give any reasons for why this would be.

12.5. LIMITATIONS OF CAP RESULTS. Heritability estimates for adoption studies will be skewed if there is selective adoption placement (placing a child with an adoptive family who is similar to the biological family) and assortative mating (mating with someone who is similar to you with respect to a given trait). Selective placement muddies the clean separation of genetic and environmental factors. Fortunately, for IQ and specific cognitive abilities there was little selective placement among the CAP children. Of particular note, the correlation between biological mothers' verbal ability and adopted mothers' verbal ability was .05, and the correlation between biological mother's verbal ability and adopted fathers' verbal abilities was .10 (Plomin & DeFries 1985). Assortative mating inflates parent-offspring correlations and so leads to overestimation of environmental influences (that are based on correlations between adoptive parents and adopted children) and genetic influences (that are based on correlations between biological parents and adopted children). Rice et al. (1986) performed multivariate analyses to determine the extent of assortative mating (mother-father correlations for the same ability) and crossassortative mating (mother-father correlations across different abilities). Results of these analyses indicated assortative mating was greater than crossassortative mating. Assortative mating was greater for verbal ability than spatial ability, perceptual speed or memory (*r*'s .36, .07, .20, and .13, respectively). Estimates of assortative mating for verbal ability were greater for adoptive parents than biological parents when CAP children were 3 years old (.34 +/− .04 and .00 +/− .30, respectively) and 4 years old (.26 +/− .05 and .00 +/− .29, respectively, Bergeman et al. 1988).[26] Subsequent analyses of the CAP data from 3-, 4-, and 7-year-olds revealed that assortative mating for verbal ability was greater for adoptive parents than biological parents at all three ages and the difference was significant at age 4 (Cyphers et al. 1989). The significant difference in biological and adoptive parents' rates of assortative mating might explain why Cyphers and colleagues found that at age 4, genetic factors did not significantly affect verbal performance but environmental factors did.[27] In general, the difference in assortative mating for biological and adoptive CAP parents probably means that heritability estimates of verbal abilities are underestimated and shared environmental effects are overestimated.

Most of the twin studies that looked for specific-to-language genetic factors found them, but the CAP study did not. I suspect that the reason for the discrepancy is simple. The twin studies that found specific-to-language genetic factors typically tested for genetic factors that affect language, but are independent of nonverbal IQ. The CAP study tested for genetic factors that affect language, that are independent of full-scale IQ. Full-scale IQ is a very good predictor of verbal IQ, which in turn is a very good

[26] The large standard error for assortative mating for biological parents is because test data are available from only about a quarter of the biological fathers. Analyses of CAP data obtained at later ages did not report whether the rates of assortative mating for verbal ability were different for adopted and biological parents, but Cardon and colleagues' (1992) analyses of 4-year-olds' CAP data suggest that they are.

[27] Because the difference in assortative mating between biological and adopted parents was not significant for most measures at most ages, Cardon et al. (1992) based their heritability estimates on a model that assumed assortative mating was the same for both types of parents. Models of CAP data obtained when children were 12 (Alarcón et al. 1998) and 16 (Alarcón et al. 1999) also used the same parameter estimate for both biological and adopted parents.

predictor of performance on many language tests (particularly vocabulary tests). It would be relatively simple and quite informative to reanalyze the CAP data and determine what the genetic correlations are for nonverbal IQ and language measures.

Although adoption studies are perhaps the cleanest type of behavioral genetic study, it is a bit worrisome that all of the existing adoption studies analyzed data from the same group of adopted children. If, for whatever reason, this sample of children is not representative of the general population, estimates of the importance of genetic and environmental factors will not generalize. Ideally, a second group of adopted children should be studied, and the heritability and environmental estimates for the two groups could be compared. The need for a second sample of adopted children is particularly pressing given that the results of the Swedish Twin/Adoption Study, unlike those of the CAP study, indicate that specific-to-language genetic factors affect elderly people's performance on verbal tasks (Pedersen et al. 1994).

13. MODES OF TRANSMISSION OF SPOKEN LANGUAGE DISORDERS. Although twin and adoption studies can be used to investigate the extent to which language is the result of heritable factors, other types of studies (e.g. segregation studies, pedigree studies, commingling studies, and linkage studies) are needed to determine the mode of transmission involved in language and language disorders. Language disorders could result from a single major gene that is transmitted according to classic Mendelian patterns (e.g. autosomal dominant, autosomal recessive, X-linked recessive) or from multiple genes interacting in complex ways with each other and the environment. Genetic models may require additional parameters (e.g. rate of spontaneous mutation, probability of ascertainment, penetrance and expressivity of a trait). Even within families, for example, language impairments are often expressed in different ways, with some family members exhibiting severe global deficits in spoken and written language and others exhibiting only moderate deficits in written language. Some family members may have the genotype for speech/language disorders but exhibit no observable sign of a spoken or written language disorder.

To test single major locus transmission models, let us assume that the expressivity and penetrance rates approach 100%, the spontaneous mutation rate approaches zero, and the incidence of language disorder is low (e.g. 5%). If the disorder is autosomal dominant (AD), then most probands will have exactly one affected parent, and on average, half of the probands' siblings will be impaired.[28] If the disorder is autosomal recessive (AR), most probands will have two (unaffected) carriers for parents, and one-quarter of the proband's siblings will be affected. For an X-linked recessive (XLR) trait to be expressed in a woman, the recessive allele must be present on both of a woman's X chromosomes, whereas for a man to express the trait, the recessive allele need be present only on the man's one X chromosome. Thus, if language disorders are simple XLR disorders, the male:female ratio should be $n:n^2$, where n is the frequency of the disordered allele. If the XLR allele is found on only 5% of X chromosomes, the ratio will be 20:1.

In a comprehensive review of studies of familial spoken-language disorders, Stromswold (1998) found that between 19 and 71% of probands had one affected parent

[28] If we assume language-impaired people are equally as likely to mate with impaired people as unimpaired people, when the incidence of an AD language impairment is 5%, the probability of having 2 affected parents is $.05^2 = .0025$. However, the chance of having two impaired parents is probably higher given the rate of assortative mating for language among adoptive CAP parents, and the rates of assortative mating reported by Gilger (1991).

(mean = 31.6%, median = 25.5%). This suggests either genetic heterogeneity (with approximately one-quarter AD transmission and three-quarters AR transmission), or AD transmission with a high rate of spontaneous mutation, or AD transmission with incomplete penetrance or expressivity. A quarter of the studies reported a substantial number of probands (between 7 and 19%) had two affected parents (Stromswold 1998). This surprisingly large number suggests that there is assortative mating for language disability, the incidence of language impairment is much greater than is generally believed, or a simple major-locus model is incorrect. The rate of affectedness among probands' siblings ranged from 15% to 49% (mean rate = 30.3%, median rate = 28.5%). Thus, for most of the studies reviewed, the sibling rates are more consistent with spoken language disorders being genetically heterogeneous or AR than with AD transmission (although the rates are compatible with AD transmission with low penetrance or expressivity). Most of the studies reviewed reported sex ratios of between 2:1 and 3:1 for probands and between 1:1 and 2.5:1 for probands' affected siblings. Even the most extreme sex ratios for probands (6:1) or affected siblings (4:1) are less than would be expected if language disorders were X-linked recessive. Based on these and other results, Stromswold (1998) concluded that spoken language disorders are genetically heterogeneous and that the vast majority of cases are not the result of a single major gene with classic Mendelian transmission.

14. Linkage studies of familial language disorders

14.1. Background. The human genome consists of two sex chromosomes (males have an X and a Y chromosome, females have two X chromosomes) and 22 pairs of autosomal chromosomes. The autosomal chromosomes are numbered from 1 to 22 according to size and band staining patterns, with chromosome 1 being the largest. Each chromosome has a primary constriction (the centromere), which is used to delineate a short arm (conventionally labeled p) and a long arm (conventionally labeled q) of the chromosomes. Locations on these arms are specified based on major staining bands. For example, 15q21 refers to band 21 of the long arm of chromosome 15.

The most definitive method for determining whether there is a genetic basis for language disorders is to determine the genes that are responsible for language disorders. Parametric and nonparametric linkage analysis techniques are used to compare the genomes of language-impaired people and their normal relatives and to determine how the genomes of affected people differ from those of unaffected relatives. The traditional way of doing this is to compare the genetic material of affected and unaffected family members in large multiplex families (multigenerational families in which several family members suffer from the same disorder, and the disorder appears to be AD, AR or XLR). In parametric linkage analyses, the transmission of marker alleles through multiple generations is compared with the transmission of the trait phenotype to determine whether the marker locus and trait locus assort independently, or whether they show decreased recombination (which would indicate that the two loci are close together on the same chromosome). The likelihood of linkage at a given level of recombination is computed, and this likelihood (in the form of an odds ratio) is compared to the likelihood of the data given free recombination. Because individual families are usually too small to yield data sufficient to determine whether a decrease in recombination is statistically significant, the logarithm of the odds ratio is taken so that the results of different families can be added together. This is referred to as the lod score (logarithm of the odds of linkage). A lod score of over 3 is generally taken as evidence of linkage (approximately corresponding to significance at the .05 level), and a lod score of less than -2 is taken as evidence that there is no linkage.

Because, for language disorders, multiplex families are rare (Stromswold 1998), geneticists also compare the DNA of sibling pairs in which one sibling is affected, the other unaffected. In nonparametric sibling-pair analyses, the proportion of marker alleles that are identical in pairs of siblings is compared with the phenotypic similarity between the siblings. For example, siblings share 0, 1, or 2 alleles at a particular locus. If the trait locus is closely linked to a marker allele, similarity between the siblings for the marker alleles should correspond to similarity for the trait phenotype, regardless of mode of transmission or penetrance for a disorder. Sibling-pair linkage analyses have several advantages over multiplex family analyses. First, because sibling-pair analyses are usually nonparametric, they are more likely to reveal associations, particularly with traits with variable expressivity. Second, one does not need to specify the mode of transmission in order to conduct sibling-pair analyses. Third, sibling-pair analyses can reveal linkage even when penetrance is not complete. Fourth, it is much easier to locate sibling pairs and, hence, sample size (and statistical power) is likely to be greater for sibling-pair than for multiplex family analyses. Fifth, because most cases of developmental language disorders do not appear to be transmitted according to simple Mendelian patterns (Stromswold 1998), linkage analyses conducted on multiplex families may implicate genes that CAN cause language disorders but rarely do.

14.2. WRITTEN-LANGUAGE DISORDERS. Linkage analyses of dyslexic multiplex families and sibling pairs suggest that written language disorders are genetically heterogeneous (Bisgaard et al. 1987, Fagerheim et al. 1999, Grigorenko 2001, Smith et al. 1986, Smith et al. 1998). Studies have linked dyslexia to a locus on the short arm of chromosome 1 near the region that codes for the Rh blood factor (1p34–p36, Grigorenko et al. 1998, Rabin et al. 1993, Smith et al. 1991),[29] a locus on the short arm of chromosome 2 (2p15–p16, Fagerheim et al. 1999, Petryshen et al. 2000b), a locus on the short arm of chromosome 6 near the human leucocyte antigen (HLA) region that is associated with immune function (6p21.3–p23, Cardon et al. 1994, 1995, Fisher et al. 1999, Gayan & Olson 1999, Gayan et al. 1995, Gayan et al. 1999, Grigorenko et al. 1997, 2000, Petryshen et al. 2000a, Rabin et al. 1993, Smith et al. 1991), and a locus on the long arm of chromosome 15 (15q21–q23, Grigorenko et al. 1997, Morris et al. 2000, Nopola-Hemmi et al. 2000, Nöthen et al. 1999, Pennington & Smith 1988, Schulte-Körne et al. 1998, Smith et al. 1983). Petryshen et al. (1999c) has linked dyslexia to the region of 11p15.5 near the dopamine D4 receptor gene and Petryshen et al. (1999b, 2001) have linked it to 6q13–16.2. Lubs et al. 1991 reported a case of a family with a translocation and fusion of chromosomes 13 and 14. Six of seven family members with the translocation were dyslexic, which suggests that there may be another dyslexia gene on chromosome 13 or 14. The two studies that have looked specifically at spelling disorders (Nöthen et al. 1999, Schulte-Körne et al. 1998) both link this disorder to the 15q21 locus that has been implicated in dyslexia.

Some researchers suggest that written language disorders associated with deficits in phonemic awareness may be more closely tied to chromosome 6p, whereas disorders

[29] Consistent with 1p36 being an important locus for reading disability, Hussain et al. (2000) reported a case of a balanced reciprocal translation between the short arms of chromosome 1 and 17 [t(1;17)(p36.3; p11.2)] that segregates with psychomotor delay and learning disabilities in eight members in a three-generation family. Two of the affected adults suffer from specific difficulties in reading. Microscopic deletion of 1p36.1 (Chedrawi et al. 1999, Knight-Jones et al. 2000) and monosomy of 1p36.1 (one copy of the 1p36 locus, Slavotinek et al. 1999) are associated with a syndrome the features of which include severe learning disabilities (for both written and spoken language), dysmorphic features, and epilepsy.

associated with deficits in orthographic decoding may be more associated with chromosome 15q (Grigorenko 2001, Grigorenko et al. 1997, Nöthen et al. 1999). Fisher et al. (1999) and Gayan et al. (1999), however, found evidence to suggest that all phenotypes of dyslexia are linked to 6p21.3 and Field and Kaplan (1998, 1999) and Petryshen and colleagues (1999a, 2000a) failed to find linkage of phonologically based dyslexia to 6p21.3. Recent studies have linked phonological dyslexia to loci at 11p15.5 (Petryshen et al. 1999c) and 6q13–q16.2 (Petryshen et al. 1999b, 2001).

There may be a genetic and neurodevelopmental link between dyslexia and other neuropsychiatric disorders that are associated with disturbances of speech or language (see Ramsay 2000). Epidemiologically, the incidence of developmental written- and spoken-language impairments is greater among children and adolescents diagnosed with schizophrenia than among normal children (Beitchman et al. 1996, Cohen et al. 1998a, 1998b, Dryborg & Goldschmidt 1996, Emerson & Enderby 1996, Javorosky 1995, McDonald et al. 1998, Nicolson et al. 2000, Toppelberg & Shapiro 2000). All but two of these studies (Dryborg & Goldschmidt 1996, Javorosky 1995) report that over half of the psychiatrically disturbed children suffer from language disorders. Similarly, the incidence of dyslexia is greater among people with attention-deficit hyperactivity disorder (ADHD) than among people who do not suffer from ADHD (Tannock & Schachar 1996, Tannock & Brown 2000), and 25–40% of children with ADHD are dyslexic (August & Garfinkel 1990, Semrud-Clikeman et al. 1992). Covariation analyses performed on behavioral data from a subset of the dyslexic twins in the Colorado Twin Study of Reading Disorders suggest that the same genes account for 95% of the comorbidity between ADHD and dyslexia (Willcutt et al. 2000). In addition to being linked to dyslexia, the p21–23 region of chromosome 6 has been linked to schizophrenia (Goei et al. 1998, Lopes-Machado and Duarte 2000, Ramsay 2000, Wright et al. 2001) and ADHD (Barr et al. 2000, Odell et al. 1997, Warren et al. 1995a, 1995b). One study found linkage of an ADHD phenotype to 6p21.3 in a population of dyslexics (Smith et al. 2000).

In summary, results of linkage studies strongly suggest that dyslexia is genetically heterogeneous, with dyslexia being linked to six distinct genetic loci. Although some studies suggest that phonologically based dyslexia is preferentially linked to different loci than orthographically based dyslexia, at this point, there doesn't seem to be a one-to-one correspondence between different dyslexia loci and different dyslexic phenotypes. It is notable that at least one of the dyslexia loci (chromosome 6p21–p23) has been linked to other neurodevelopmental and psychiatric disorders that have prominent speech/language symptoms.

14.3. SPOKEN-LANGUAGE DISORDERS. Much less is known about the genes involved in spoken language disorders. Froster et al. 1993 reported a case of familial speech retardation and dyslexia that appears to be caused by a balanced translocation between regions of the short arm of chromosome 1 and the long arm of chromosome 2 (1p22 and 2q31). Elcioglu et al. 1997 reported a case of a boy with a Marfan-like appearance and severe language delay but reasonable nonverbal cognitive skills. Genetic analyses revealed a de novo inverted duplication of the segment (15q13.3→15q21.3) at 15q24.3 (i.e. the duplication includes the 15q21 locus that has been linked to dyslexia). Fisher and colleagues (1998) conducted the first linkage analyses for spoken-language disorders, performing genome-wide analyses of the genetic material of the three-generation KE. The KE family is unusual in that the pattern of affected and unaffected family members suggests that the KE family suffers from an autosomal dominant disorder

with 100% penetrance. Affected members of the KE family suffer from grammatical deficits (Gopnik 1990), oral-facial dyspraxia (difficulty making complex, conscious oral and facial movements) and associated speech disorders (Fisher et al. 1998, Hurst et al. 1990), depressed nonverbal IQ, and developmental learning disorders that do not appear to be verbal in nature (Vargha-Khadem et al. 1995). Fisher et al. 1998 confirmed autosomal dominant transmission with near 100% penetrance, and determined that the impairments exhibited by members of this family are linked to a 5.6 cM long region on the long arm of chromosome 7 (7q31). Lai et al. (2000) more precisely localized the site of the affected allele (7q31.2), and ruled out the possibility that affected KE family members suffer from a microdeletion. In addition, Lai and colleagues performed similar linkage analyses for two people who are unrelated to the KE family, but suffer from similar speech/language impairments. One has a translation in the same region identified in the KE family (7q31.2) and the other has a translation on 7q more than 3.7 mB outside the KE region. Tomblin et al. (1998) conducted linkage analyses to determine whether the genotypic variation at the 7q31 locus was associated with language disability in 434 second graders who are a subsample of the 7,218 children whose language and speech were assessed in the National Institute of Health's Epidemiology of Specific Language Impairment study (Tomblin et al. 1996, 1997a,b). Affected family-based control analysis showed evidence of linkage to 7q31 for language status, phonological memory and reading status, but not speech sound production (Tomblin et al. 1998). Bartlett et al. (2000) conducted linkage analyses on 19 multiplex families with spoken-language disorders. In contrast to the KE family, Bartlett et al.'s (2000) family members did not suffer from oral motor problems, and only those family members with nonverbal IQs greater than 80 were included. Bartlett and colleagues found loci at or near four sites that have previously been linked to dyslexia (a locus 100 cM telomeric from Rabin et al.'s 1p36–p34 locus, a locus 40 cM telomeric from Fagerheim et al's 2p15–p16 locus, a locus at 6p21.3, and a locus at 15q21–q23). Notably, they found no evidence of linkage to 7q31, the region implicated with developmental speech disorders in the KE family. Cholfin et al. (2000) recently presented the linkage results for the MN family, a multigenerational family with an autosomal dominant speech/language disorder. Affected MN family members have language delay, articulatory speech disturbances, and grammatical deficits similar to those exhibited by affected KE family members. Despite the phenotypic similarities between the MN and KE families, linkage analyses revealed that the MN family's language disorder is not linked to 7q31. Cholfin et al. reported that they are in the process of conducting a genomewide scan to determine the locus of the speech/language in the MN family.

The 7q31 region is associated with other neuropsychiatric disorders that affect speech and language. The incidence of speech and language disorders is higher among relatives of autistic people than among the general public (Folstein & Rutter 1988, Kjelgaard & Tager-Flusberg 2001, Konstantareas & Beitchman 1996, Landa et al. 1991, Le Couteur et al. 1996, Maestrini et al. 1998, Mannens & Alders 1999, Palmour 1997, Piven 1999, Silliman et al. 1989), and some researchers suggest that developmental speech disorders like SLI are a milder manifestation of the same genotype that causes autism (Kjelgaard & Tager-Flusberg 2001, Konstantareas & Beitchman 1996, Landa et al. 1991, Le Couteur et al. 1996, Piven 1999, Silliman et al. 1989). Consistent with this, the 7q31 region is strongly linked to autism in families with a high incidence of autism (Ashley-Koch et al. 1999, Barrett et al. 1999, Craddock & Lendon 1999, Folstein & Mankoski 2000, Folstein & Santangelo 2000, Lauritsen et al. 1999, Rutter 2000, Vincent et al. 2000a,b,c, Warburton et al. 2000, Yan et al. 2000). Warburton and colleagues

conducted linkage analyses on a person with autism and a person with a developmental speech/language disorder, and found that both suffered from different, balanced translocations in the 7q.31 region. Ashley-Koch et al. conducted genome-wide linkage analyses on members of a family with autosomal dominant transmission of autism (two brothers are autistic and a sister suffers from a expressive-language disorder). The researchers determined that the siblings inherited from their mother (who is unaffected) a pericentric inversion involving the 7q31 region (inversion from q22-q31.2). They then did linkage analyses of 76 multiplex autistic families, the results of which confirmed a locus for autism in the 7q31 region, and provided some evidence that this locus is paternally expressed. Yan et al. (2000) reported a large multiplex family with a reciprocal translation of the short arm of chromosome 1 and the long arm of chromosome 7 that spanned 7q21. In this family, many members suffer from a variety of neuropsychiatric disorders including autism, schizophrenia, and developmental language disorders.

There may be a neurodevelopmental and genetic link between Tourette syndrome and spoken-language disorders. Tourette syndrome is a complex disorder characterized by vocal and motor tics. People with Tourette syndrome have a higher than normal incidence of written- and spoken-language disorders (Comings 1997, Comings & Comings 1986, 1987, 1991). Several studies have linked Tourette syndrome to the 7q31 locus that has been implicated in spoken-language disorders. Boghosian-Sell et al. (1996) conducted linkage analyses of a multiplex family and determined that affected family members suffered from a translocation of 7q and 18q, and that the translocation affected 7q31. Petek et al. 2001 and Kroisel et al. 2001 recently reported cases of patients with Tourette syndrome who had a de novo duplication and inversion of the long arm of chromosome 7 involving the 7q31 region (the duplication went from q22.1–q31).[30]

Given the linkage results for written-language disorders, a priori, it is extremely likely that familial spoken-language impairments are genetically heterogeneous with many different genes being involved. The few existing linkage studies indicate that this is true: the linkage results for the KE family and the SLI children studied by Tomblin and colleagues (1998) suggest that the 7q31 locus is linked with some developmental spoken-language disorders. But the failure of Bartlett et al. (2000) and Cholfin et al. (2000) to find linkage of spoken-language disorders to 7q31 suggests that other loci are also involved in spoken-language disorders. The finding by Bartlett et al. (2000) that spoken-language disorders are linked to four dyslexia loci provides further evidence that SLI is genetically heterogeneous. In addition, these results are consistent with theories that argue that written- and spoken-language disorders are just phenotypic variants of the same disorder. As is the case with dyslexia, the loci that have been linked to spoken-language impairments have also been linked to other disorders (autism, Tourette syndrome to 7q31; schizophrenia and ADHD to 6p21.3) that affect speech and language. It is possible that in some cases these disorders are different phenotypic expressions of the same genotypes.

14.4. FINDING THE GENE FOR DEVELOPMENTAL LANGUAGE DISORDERS. Knowing the approximate loci for spoken- and written-language disorders does not tell us what genes are involved, nor does it tell us WHY alterations in these genes result in language

[30] Heutink et al.'s (1990) failure to find evidence of linkage of Tourette syndrome to 7q31 or 18q22 in a different multiplex family suggests that Tourette syndrome is genetically heterogeneous.

disorders. There are two routes to understanding the genetic etiology of language disorders. The first is to investigate neural bases of developmental language disorders and then posit a means by which these features might have arisen. (For a comprehensive review of the neural bases of developmental dyslexia, see Grigorenko 2001, Habib 2000) The second route is to determine what proteins are coded for in the loci linked with written- and spoken-language disorders and see whether aberrations in any of these proteins could plausibly cause dyslexia or SLI.

Autopsies of dyslexic brains reveal a number of neuropatholoical findings (Galaburda 1991, Galaburda et al. 1985, Geschwind & Galaburda 1987). Dyslexic brains (particularly male brains) have small (1 mm) clusters of ectopic neurons (brain 'warts') in the outside layer of the cerebral neocortex, a layer that normally lacks neuronal cell bodies. These ectopias are associated with anatomic and biochemical disruption of the six underlying cortical layers and with (smaller) disruptions in homologous areas in the opposite hemisphere. Although ectopias are found throughout the cortex, most are found in the left perisylvian and frontal regions. Some dyslexic brains also have dysplastic microgyri (tiny aberrant infoldings of the cortex). At the macroscopic level, Galaburda and colleagues (1985) found that in dyslexic brains, the right temporal plane is usually the same size or larger than the left temporal plane, whereas in normal controls, the left temporal plane is usually larger. Some studies suggest that dyslexics have selective functional disruption of thalamic magnocellular neurons that are involved in the visual pathway (Livingstone et al. 1991) and the auditory pathway (Galaburda et al. 1994). To date, only one brain of a possible SLI child has come to autopsy. Postmortem examination of this brain revealed atypical symmetry of the temporal planes and a dysplastic micro-gyrus on the inferior surface of the left frontal cortex along the inferior surface of the sylvian fissure (Cohen et al. 1989), findings similar to what Galaburda and colleagues have reported for dyslexic brains.[31]

For the sake of argument, let us assume that dyslexia and (perhaps) SLI are the result of these neuropathological abnormalities. What might cause these neurodevelopmental abnormalities? Noting an apparently higher frequency of immune dysfunction in dyslexic people than in the general population, Geschwind and colleagues suggested that there is a direct link between immune dysfunction and neurodevelopmental disorders (Behan & Geschwind 1985, Crawford et al. 1994, Galaburda 1983, 1991, Galaburda et al. 1985, Geschwind 1983, Geschwind & Behan 1982, Geschwind & Galaburda 1985, 1987, Hugdahl 1994, Pennington et al. 1987, Rosen et al. 1991).[32] Maternal autoantibodies (coded for by the HLA genes) might attack the developing brain prior to the sixth month of gestation and disrupt the normal processes that guide nerve cell migration. They might, for example, act to create a breach in the pial-glial border that normally prevents neurons from migrating too far. Another possibility is that the HLA genes are indirectly responsible because aberrant immune function results in the fetal brain being exposed to neurotoxic substances (e.g. viruses) that prevent normal cell migration. The high incidence of immune disorders among dyslexics (Crawford et al. 1994, Hugdahl 1994, Pennington et al. 1987) is consistent with some version of the immune function hypothesis. The proximity of the 6p21–23 dyslexia locus to the HLA

[31] Because this child had a performance IQ of only 74 (verbal IQ 70), the anomalies noted on autopsy may be related to general cognitive impairment rather than spoken-language impairment.

[32] A number of researchers have suggested that the incidence of autoimmune disorders is also elevated in schizophrenia, ADHD, and autism (Geschwind & Galaburda 1987, Lopes-Machado & Duarte 2000, Odell et al. 1997, Warren et al. 1995a,b). Not all studies are in agreement, however (Jacobsen et al. 1999).

locus and the proximity of the 15q21–23 dyslexia locus to the beta2-microglobin gene (which has been implicated in human autoimmune system, Lazarus & Owen 1994) provides additional support for some version of the dyslexia/immune dysfunction theory. But the epidemiological link between immune dysfunction and dyslexia is not as clear-cut as was once thought. Bryden et al. (1994) found that of 31 studies used to support the GBG theory, only 16 revealed a statistically significant relationship between immune disorders and dyslexia, and there were methodological shortcomings in many of these studies. The failure of a carefully designed twin and family study (Gilger et al. 1998) to reveal an association between dyslexia and immune system dysfunction raises further questions about the dyslexia/immune dysfunction theory.

The 6p21–23 region contains a number of other candidate genes for developmental language disorders. The gene for the gamma-aminobutryic acid (GABA) beta receptor 1 is located at chromosome 6p21.3 (Barr et al. 2000, Goei et al. 1998, Kaupmann et al. 1998). GABA is the major inhibitory neurotransmitter in the central nervous system, modulating neurotransmission by presynaptic inhibition of the release of neurotransmitters and neuropeptides. GABA beta 1 receptor also has an inhibitory effect by increasing $K+$ conductance that results in long-lasting inhibitory postsynaptic potentials. GABA-beta receptor agonists are used to treat some forms of epilepsy, anxiety, and depression (Bittiger et al. 1993). Barr et al. 2000 identified nine variants of the GABA beta receptor 1 gene in 98 ADHD probands, 22 of whom were also dyslexic. They tested whether the three most common polymorphisms of the receptor (each of which occurred in 20% of the ADHD probands) were linked to ADHD in the probands' nuclear families. They concluded that the GABA beta receptor polymorphisms were not linked to ADHD, but because their study included too few dyslexics they could not rule out the possibility of linkage of the GABA beta receptor to dyslexia.

Genes that code for proteins involved in fatty acid and membrane phospholipid metabolism have also been suggested as candidate genes for a wide range of neurodevelopmental disorders, including dyslexia (Bennett & Horrobin 2000, Horrobin & Bennett 1999, Horrobin et al. 1995, Richardson & Ross 2000). Horrobin and Bennett argue that several factors make these genes good candidates for neurodevelopmental disorders. First, recent studies have implicated abnormalities in fatty acid and membrane phospholids metabolism with dyslexia, dyspraxia, ADHD, autism, and schizophrenia, and some studies have found that dietary supplementation with highly unsaturated fatty acids may improve some of these conditions (for a summary, see Richardson & Ross 2000). Phospholipid enzymes are strongly expressed in the brain during the prenatal period, and highly unsaturated fatty acids are critical for normal brain development. In addition, as summarized above, epidemiological studies reveal a high incidence of comorbidity within affected families for many of these disorders. Many are linked to the same chromosomal regions (e.g. 6p21–23 for dyslexia, ADHD and schizophrenia; 7q31 for speech dyspraxia, SLI, Tourette's syndrome, and autism). Although linkage work is still in its infancy, one enzyme involved in phospholipid metabolism (lyso-phospholipid coenzyme A acyl transferase) has already been linked to 6p21–23 (Horrobin & Bennett 1999). Future work is needed to determine whether polymorphisms in lyso-phospholipid coenzyme A acyl transferase are related to written and/or spoken language disorders, and whether any other phospholipid proteins are located at sites linked to dyslexia or SLI.

Jamain and colleagues (2001) recently determined that the human kinesin gene is located at 6p23. In early mouse development, the mouse homologue of the human kinesin gene is expressed almost exclusively in the central nervous system. C. elegans,

mutants that lack the kinesin gene, have behavioral defects, leading Jamain et al. (2001) to suggest that the kinesin gene might be a candidate gene for schizophrenia. Other researchers have suggested that there might be an association between schizophrenia and a particular mutation of the spinocerebellar ataxia type 1 gene that is also located at 6p23 (Joo et al. 1999). Another candidate gene for schizophrenia is a transcription factor activating protein that is also coded at 6p23 (Kawanishi et al. 2000). Given the epidemiological association between schizophrenia and developmental language disorders, these three genes are possible candidates for cases of dyslexia that are linked to 6p21–23.

The linkage of some cases of dyslexia to 2p15 is intriguing because the protein phosphatase calcineurin (which has long been considered a candidate for psychiatric and monogenic brain disorders) is coded for by a gene in the 2p15 region (Stratakis & Taymans 1998). The possible linkage of dyslexia to the region of 11p15.5 near the dopamine D4 receptor gene (Petryshen et al. 1999c) is of interest because dopamine is believed to play a central role in neurodevelopment and in many neuropsychiatric disorders. The linkage of dyslexia to 15q21 is interesting because recent work suggests that neuronal tropomodulins 2 and 3, the major binding protein to brain tropomyosin, are coded for by genes in this locus (Cox & Zoghbi 2000).

Lai and colleagues (2001) recently discovered that all of the affected KE family members (and the unrelated language-impaired person who suffered a translocation involving 7q31.2) have an abnormal form of the gene that codes for a forkhead/winged-helix transcription factor (*FOXP2*), whereas none of the unaffected KE family members or unrelated normal controls have an abnormal *FOXP2* gene. Affected KE family members have a point mutation in FOXP2 gene that results in the amino acid histidine replacing an arginine in the forkhead domain of the transcription factor, and this substitution may disrupt *FOXP2*'s ability to bind DNA (Lai et al. 2001). *FOXP2* is highly expressed in human fetal tissue, and the homologue of *FOXP2* is found in fetal mouse cerebral cortex. Because affected KE family members have only one abnormal *FOXP2* gene, Lai and colleagues (2001) propose that their impairments result from them having not having enough *FOXP2* transcription factor to control some aspect of early neuroembryogenesis. It remains to be seen whether other people with neurodevelopmental disorders linked to 7q31 (e.g., people with autism, Tourette syndrome, the language impaired children in Tomblin et al.'s 1998 study) have abnormalities involving the *FOXP2* gene or some other gene.[33]

15. CONCLUSIONS. A comprehensive review of twin, adoption, and linkage studies of language indicates that genetic factors account for some of the individual differences in linguistic ability for both normal people and people who suffer from developmental language disorders. For both written- and spoken-language impairments, MZ twins are significantly more likely to be concordant for language disorders than are DZ twins. In the studies reviewed, heritable factors typically accounted for over half of the variance in language-impaired people's linguistic abilities. Consistent with this, the one existing adoption study of language disorders found that adopted children who had language-

[33] At least two other candidate genes for neurodevelopmental disorders have been linked to 7q31. One is the gene for a neuronal cell adhesion molecule that is found in the human brain, the homologue of which has been implicated in specific pathfinding roles of axonal growth cones in the developing nervous system of chicks (Lane et al. 1996). Another candidate gene is a G-protein coupled receptor (Donohue et al. 1998, Hellebrand et al. 2000, Marazziti et al. 1997). This receptor is expressed almost exclusively in a few brain regions that have been implicated in some neurolinguistic studies (e.g., the corpus collosum and certain frontal areas).

impaired biological relatives were almost three times more likely to suffer from language disorders than adopted children who did not have language-impaired relatives. Studies that compare normal MZ and DZ twin pairs' performance on language tests generally find that MZ twins are linguistically more similar to one another than DZ twins for all aspects of written and spoken language. Although genetic factors that affect general cognitive ability influence linguistic performance, the greater linguistic similarity of MZ twins is not solely the result of the heritability of general cognitive ability. Similarly, some adoption-study results suggest that the heritability of language is partly the result of specific-to-language genetic factors. Finally, the results of a few twin studies indicate that there may be genetic factors that specifically influence some aspects of language but not others—affecting phonological awareness, for example, but not vocabulary and morphosyntax. Taken together, the results of adoption and twin studies support the hypothesis that language is partly the result of innate predispositions and structures specific to language. If additional studies corroborate that different genetic factors influence different aspects of language, this would provide support for linguistic theories that posit that language results from the interaction of semi-autonomous modules such as phonology and syntax.

In a review of family aggregation, segregation, commingling, pedigree, and sex-ratio studies of spoken-language disorders, Stromswold (1998) concluded that spoken-language disorders are etiologically heterogeneous and that although some may be the result of autosomal recessive or dominant genes that follow classic Mendelian transmission patterns, most are not. The results of the twin studies, adoption studies, and linkage studies reviewed here provide further evidence that specific-to-language genetic factors play a substantial role in the variation observed in linguistic abilities among both people who suffer from language disorders and those who do not. The relationship between the genotype and phenotype (linguistic ability) is not simple, however.

Take the case of frank language disorders. The probable existence of genetic and behavioral heterogeneity for language disorders means at least three distinct relationships could obtain between genotypes and behavioral phenotypes. Given what we now know, it is extremely unlikely that there is a one-to-one relationship between genotypes and phenotypes, with each genotype causing a distinct type of language disorder. Alternatively, there might be a one-to-many mapping between genotypes and phenotypes, with a single genetic disorder resulting in many behaviorally distinct types of language disorders. The disordered 6p21 allele, for example, could cause ADHD and/or dyslexia. One child with a genetically encoded articulation disorder might respond by refusing to talk at all, whereas another child with the same genetic disorder might speak and make many articulation errors. Lastly, there may be a many-to-one mapping between genotypes and phenotypes with many distinctive genotypes resulting in the same type of linguistic disorder. For example, many researchers have noted that English-speaking children with developmental language disorders frequently omit grammatical morphemes such as articles and determiners, auxiliary verbs, and verbal and nominal inflections (see Leonard 1998). Some of these children may have a genetically encoded articulation disorder that causes them to omit these morphemes because they are typically pronounced rapidly. Some may have a genetically encoded disorder affecting processing of rapid auditory input that causes them to have difficulty acquiring unstressed, short-duration grammatical morphemes. Still others might omit them because they have a genetically encoded syntactic deficit, and these morphemes convey grammatical information. In addition, the genotype-phenotype relationship is not necessarily static. Longitudinal studies reveal that, even within an individual, a developmental

language disorder can manifest itself in different ways at different ages (Aram et al. 1984, Silva et al. 1987).

Environmental factors may have different effects on different people depending on their genetic makeup. Although within a fairly wide range, linguistic environment may have little or no effect on language acquisition by normal children (e.g. Heath 1983), there could be synergistic interactions between genes and environment, with children who are genetically at risk for developing language disorders being particularly sensitive to subtly impoverished linguistic environments. Because genetically at-risk children are likely to have relatives who are language impaired, they are likely to be reared in linguistically impoverished environments. Conversely, it is possible that people who have the genetic propensity to succeed at language might benefit more from enriched environments (and better tolerate impoverished environments). Because genetically well-endowed children are more likely to have relatives who are linguistically able, they are likely to be reared in linguistically enriched environments. If environmental factors interact with genetic factors in this way, then standard twin methods for quantifying heritability will overestimate the role of genetic factors.

Plomin and Dale (2000) argue that it is unlikely that a complex trait such as language is the result of a small number of genes acting alone or in combination with the environment. Rather, the variance observed in linguistic abilities is probably the result of many genes, each of which has a relatively small effect, acting together and in combination with environmental factors. If this picture is correct, then language, like height, is a quantitative trait with a distribution that is approximately normal, and the observed heritability of language is the result of quantitative trait loci (QTL). Several factors may make it easier to find the QTLs for language. People tend to choose mates who have similar verbal abilities (the spousal correlation for language is greater than .50), and this type of assortative mating causes the additive genetic variance of a population to increase with each generation (Plomin & Dale 2000). For technical reasons, assortative mating and additive genetic variance make the task of finding QTLs easier. Until recently, molecular geneticists have had the most success finding the genes that affect language by performing linkage analyses on the genetic materials of people who suffer from language disorders. Such techniques are most successful, however, for detecting genes that have major effects (e.g. autosomal dominant or recessive genes). In an article on QTLs associated with general cognitive abilities, Chorney et al. (1998) argued that it may be easier to detect QTLs by performing genetic analyses on people whose abilities fall at the high end of the distribution, rather than by performing such analyses for people who fall at the low end of the distribution. The reason is that at the low end of the distribution, the impact of random mutations and environmental insults may obscure the effects of QTLs that influence normal variation in abilities. If the same argument holds for language, perhaps linguists (particularly second-generation linguists) could aid in the search for the QTLs for language by donating samples of their DNA to their local geneticists.

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