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Why aren't identical twins linguistically identical? Genetic, prenatal and postnatal factors

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Abstract

Results of twin studies clearly demonstrate that genetic factors play an important role in the rate of language acquisition and linguistic proficiency attained by normal and impaired children and adults [see Stromswold, K. (2001). The heritability of language: A review and meta-analysis of twin, adoption and linkage studies. *Language, 77*, 647–723.]. That said, twin-based heritability estimates for language rarely exceed .6 and monozygotic (MZ) twins (who are usually assumed to have identical genetic and environmental endowments) sometimes have very different linguistic profiles. In addition, twins are more likely to suffer linguistic delays and impairments than singletons. Postnatal factors, such as differences in linguistic input twins receive, are usually assumed to be the major reason for these findings. This paper discusses how genetic, epigenetic, and perinatal environmental factors can lower heritability estimates for language, cause MZ twins to be linguistically discordant, and increase the risk of language impairments in twins. We present results from our ongoing Perinatal Environment and Genetic Interaction (PEGI) study that suggest that perinatal environmental factors affect linguistic development more than postnatal factors, and that postnatal factors affect cognitive development more than perinatal factors. Because perinatal factors are overwhelming biological, whereas postnatal factors tend to be psychosocial (e.g., how and how much parents speak to their children), these results support nativist/biological theories of language and language development and call into question empiricist/emergentist theories. These results are also consistent with modularist theories of language. We end by suggesting new methods that can be used to tease apart the effects of prenatal and postnatal environment and to investigate how these factors interact with genetic factors.

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1. Introduction

Results of behavioral and molecular genetic studies clearly demonstrate that genetic factors play an important role in the rate of language acquisition and the linguistic proficiency attained by normal and language-impaired people (see Stromswold, 1998, 2001). Twin studies are the most common type of study used to investigate the impact of genetic factors on language. Roughly speaking, the logic of twin studies is as follows: identical (monozygotic, MZ) twin pairs and fraternal (dizygotic, DZ) twin pairs share essentially the same pre- and postnatal environment, whereas MZ twins share 100% of their alleles and, on average, DZ twins share only 50% of alleles. Therefore, if MZ twin pairs' linguistic abilities are more similar than DZ twin pairs', this suggests that genetic factors play a role in language. One way to determine whether MZ twins are linguistically more similar than DZ twins is to compare the concordance rates for language disorders in MZ and DZ twin pairs. Twin pairs are concordant for a language disorder if both twins are impaired, and discordant if only one twin is impaired. If the concordance rate for developmental 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. Meta-analyses of twin concordance data from 10 studies suggest that heritable factors account for a little more than two-thirds of variance for both written and spoken language impairments (Stromswold, 2001). However, within these studies, MZ concordance rates range from 35% to 100% (mean = 80%), with only one of the 10 studies reporting 100% concordance. If MZ cotwins really have the same genetic and environmental endowments, why are any MZ twin pairs discordant for language impairments?

Perhaps the existence of discordant MZ twins merely reflects that concordance studies take what is almost certainly a continuous variable (linguistic ability) and artificially categorizes twins as either impaired or not impaired. Inevitably, there will be cases in which one MZ twin scores just a few points higher than his or her cotwin, but this small difference is enough to have one cotwin labeled ''normal'' and the other ''impaired.'' In cases where the data obtained are more or less continuous (e.g., scores on language tests) rather than dichotomous (presence or absence of a language disorder), one can sidestep this problem by comparing the degree of similarity of MZ and DZ cotwins. Stromswold (2001) meta-analyzed data from almost 100 twin studies. These analyses indicate that depending on what aspect of language is assessed, heritable factors account for between 1/2 to 2/3's of the variance in language-impaired twins' linguistic abilities and 1/4 to 1/2 of the variance in normal twins' linguistic abilities. These meta-analyses also reveal that for both language-impaired and normal twins, genetic factors play a greater role for phonological

and syntactic abilities than for lexical abilities. For example, heritable factors accounted for about a third of the variance in normal twins' lexical abilities and about a half of the variance in their phonological and syntactic skills. These meta-analytic results are consistent with the initial results from our Perinatal Environment and Genetic Interactions (PEGI) twin study (Stromswold, Schramm, Molnar, Holodak, & Sheffield, 2005).[1] Concordance analyses reveal that genetic factors account for over 80% of the language disorders in the PEGI twins. In addition, genetic factors account for more of the linguistic variance for language-impaired PEGI twins than for normal PEGI twins. For example, dominant genetic factors (D) only play a role in the linguistic abilities of language-impaired twins. Also consistent with Stromswold's (2001) meta-analytic results, for both language-impaired and normal PEGI twins, genetic factors account for more of the variance for phonology (70% for language-impaired and 31% for normal twins) and syntax (100% for language-impaired and 26% for normal twins) than for vocabulary (69% for language-impaired and 5% for normal twins). Collapsing across language-impaired and normal PEGI twins, additive (A) and dominant (D) genetic factors account for 68% of PEGI twins' phonological abilities, 59% of PEGI twins' syntactic abilities, and 40% of PEGI twins' lexical abilities.

Although the heritability estimates obtained in essentially all twin studies indicate that MZ cotwins are more linguistically similar than DZ cotwins, estimates of the role of genetic factors rarely exceed 60% and some MZ cotwins have measurably different linguistic abilities. If MZ cotwins are genetically and environmentally identical, why is this so? Putting aside the possibility that the results are solely due to the misclassification of MZ twins (i.e., calling some DZ twins MZ) or various forms of measurement error, there are at least four types of reasons for this:

(1) MZ cotwins are not genetically identical
(2) MZ cotwins are not epigenetically identical
(3) MZ cotwins have different perinatal environments[2]
(4) MZ cotwins have different postnatal environments

Interactions within and among these four types of factors could accentuate linguistic differences between MZ cotwins. Furthermore, heritability estimates will be

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[1] The PEGI results reported in this paper are for the first 267 same-sex twin pairs who were between the ages of 2 and 6 when they enrolled in our on-going longitudinal PEGI study. Of the twin pairs, 145 were MZ and 122 were DZ. (Zygosity of twins was determined using a questionnaire (Goldsmith, 1991) that asks about the physical and developmental similarity of young twins. This questionnaire has a reliability of over 95%.) Fifty-two percent of the twins were male, and 48% were female. The mean birth weight of the MZ twins was 2294 g (SD = 583 g) and the mean birth weight of the DZ twins was 2309 g (SD = 650 g). The mean gestational age at birth of the MZ twins was 35.1 weeks (SD = 2.75 weeks) and the mean gestational age at birth of the DZ twins was 35.0 weeks (SD = 3.17). For details of the PEGI study, see Stromswold et al. (2005). Links to sample copies of the tests and questionnaires may be found at http://ruccs.rutgers.edu/~karin/PERINATAL/language_perinatal.htm.

[2] We will use the term "perinatal" to refer to the period that begins with the implantation of the embryo and ends at 44 weeks gestation. Postnatal refers to any time after this period.

attenuated if MZ cotwins are not genetically or epigenetically identical or if DZ cotwins are genetically or epigenetically more similar to one another than non-twin siblings. The role of genetic factors will also be attenuated if MZ cotwins have less similar prenatal or postnatal environments than DZ cotwins (and accentuated if MZ twins have more similar environments).

There are reasons to suspect that the genetic makeup and/or environments of twins differ from those of singletons. For example, it is well established that, as a group, twins acquire language more slowly and are more likely to be language impaired than singletons. Day (1932a, 1932b) 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' language impairments tended to be greater than their cognitive impairments, suggesting that twins' linguistic delays may be somewhat selective. Subsequent studies have confirmed Day's observation, and even if we compensate for twins' shorter gestation, twins' language development is 2–3 months delayed relative to that of singletons (e.g., Conway, Lytton, & Pysh, 1980; Dale, Dionne, Eley, & Plomin, 2000; Hay & O'Brien, 1983; Koch, 1966; Mittler, 1969, 1970, 1976; Rutter & Redshaw, 1991; Rutter, Thorpe, Northstone, & Golding, 2003).

In this paper, we will discuss possible genetic, epigenetic and environmental reasons why MZ cotwins are sometimes linguistically discordant, why twins' language development lags behind that of singletons, and how heritability estimates of language may be affected by these factors. In Section 2, we review the basics of twinning, and discuss the genetic and epigenetic similarity of MZ and DZ cotwins. Section 3 discusses differences in the perinatal environments of twins and singletons and MZ and DZ twins, concentrating on perinatal factors that could selectively affect linguistic development. In Section 4, we discuss postnatal factors that may affect twins' language development. Section 5 reports results of analyses of the PEGI twin data designed to determine the relative importance of perinatal and postnatal factors on linguistic and non-linguistic development. Section 6 discusses the complex relationship between genotype and linguistic phenotype, emphasizing ways in which pre- and postnatal environmental and genetic factors may interact with one another to affect language development. Lastly, in Section 7 we suggest ways of teasing apart how genetic, epigenetic, perinatal and postnatal environmental factors influence language development.

2. The genetics of twinning

2.1. Twin types

DZ twins result when two sperm fertilize two ova in the same menstrual cycle. The result is two zygotes, each of which develops its own placenta and amniotic sac

(i.e., dichorionic–diamniotic DZ twins, see Fig. 1).[3] MZ twins, on the other hand, result when a single zygote (that originates from the fertilization of one ovum by a single sperm) divides in two. There are three distinct types of MZ twins, and these differences may have genetic and perinatal environmental implications. MZ twins may be dichorionic–diamniotic (two placentas, two amniotic sacs), monochorionic–diamniotic (one placenta, two amniotic sacs), or monochorionic-monoamniotic (one placenta, one amniotic sac). Dichorionic–diamniotic MZ twins occur when the zygote splits during the first three days following fertilization. This type of MZ twinning accounts for about 20–25% of all MZ twins. When the inner cell mass splits after blastocyst formation but before the formation of the amniotic sac (at 8 days after fertilization), the result is monochorionic–diamniotic MZ twins which account for about 70–75% of MZ twins. When division occurs after the formation of the amniotic sac, but before the establishment of the embryonic axis (at about 15 days after fertilization), the result is monochorionic–monoamniotic MZ twins, the rarest form of twins accounting for only 1–5% of all MZ twins. (For a succinct and accessible summary of twinning, see Redline, 2003.)

2.2. Genotypic similarity of MZ and DZ twins

The validity of heritability estimates obtained from twin studies is predicated on MZ cotwins having identical genotypes. If they do not, heritability estimates will be lowered. A number of mechanisms can cause MZ cotwins to have different genotypes. Although the vast majority of MZ twins are karyotypically identical (i.e., the number and general morphology of the cotwins' chromosomes are the same), if chromosomal non-disjunction occurs just before or at the time of twinning, MZ twins will have different karyotypes and are said to be heterokaryotic (Lejeune, 1963). Reports of heterokaryotic MZ twins in which one twin is normal and the other is affected with Down syndrome (Trisomy 21) or Turner syndrome (X0 females) date back to at least the early 1960s (Lejeune, 1963). A more subtle way that MZ twins may have different genotypes is if a spontaneous mutation occurs either before or after the zygote has split. In most cases, the MZ cotwin would be a genetic mosaic with a subset of the twin's cells exhibiting the mutation. A very early mutation will affect a greater proportion of the cells in the fetus, and will result in generalized mosaicism with most of the tissues affected. A mutation which occurs later (e.g., at the blastocyst stage) will affect a smaller proportion of the cells in the fetus, and the abnormal cells may be restricted to a certain area or tissue type (e.g., the central nervous system). When MZ splitting occurs earlier (i.e., in dichorionic MZ twins), there is a greater chance that MZ cotwins will have different spontaneous mutations.

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[3] The two placentas in dichorionic twins sometimes fuse. Eight percent of DZ twins have two genetically distinct cell lines in their blood, but one cell line in their solid tissues (van Dijk, Boomsma, & de Man, 1996). The high rate of blood chimerism in DZ twins suggests that fused placentas are not that rare, and that some of the special risks thought to affect only MZ twins could affect some DZ twins. It also suggests that twins who share a placenta should not be assumed to be MZ and that, ideally, zygosity testing should be determined by genotyping solid tissue and not blood (Redline, 2003; Souter et al., 2003).
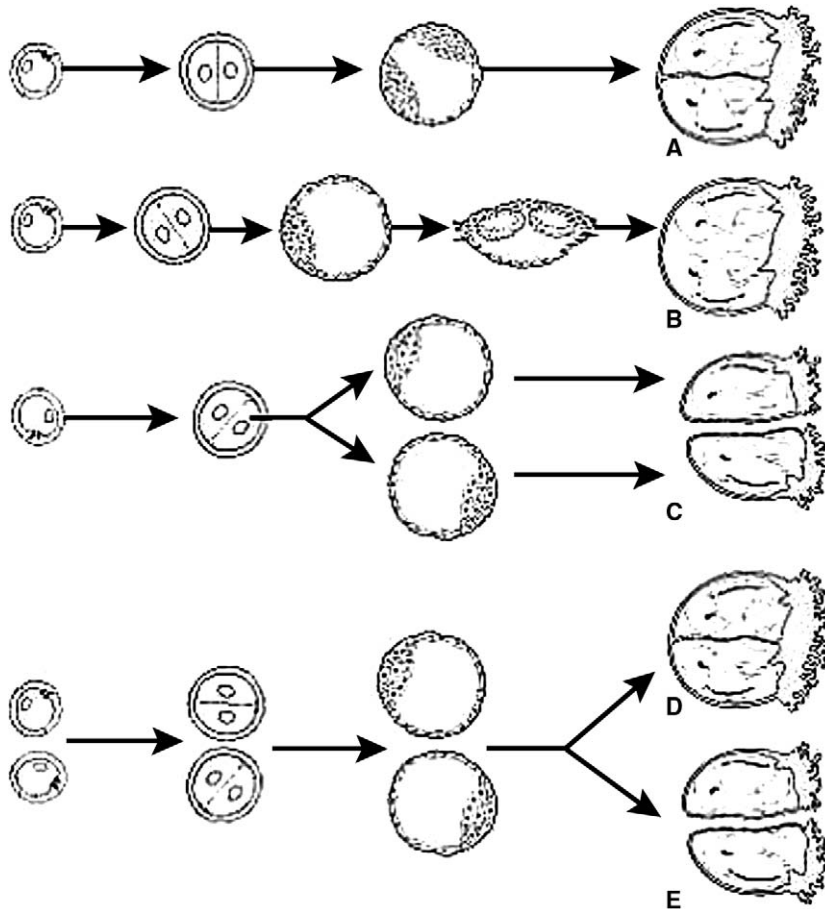
Fig. 1. Types of twins (A) monochorionic, diamniotic MZ twins (1–5% of MZ twins) (B) monochorionic, monoamniotic MZ twins (70–75% of MZ twins) (C) dichorionic, diamniotic MZ twins with separate placentas (20–25% of MZ twins). (D) Dizygotic twins with fused placentas (0–5% of DZ twins) (E) Dizygotic twins with unfused placentas 95–100% of DZ twins.

Thus, dichorionic MZ twins are more likely to differ genetically than monochorionic MZ twins. Very late splitting may mean that the ''handedness'' of the embryo has already been determined prior to twinning, and Sommer, Ramsey, Mandl, and Kahn (2002) have argued that this is associated with MZ cotwins who are discordant for handedness and language lateralization. Nance (1990) has argued that if the inner cell mass splits unequally, there will be different number and types of founder cells for MZ cotwins and this can result in the cotwins being phenotypically different from one another.

Twin-derived heritability estimates will also be skewed if DZ cotwins share more (or less) than 50% of their alleles. Transplant surgeons have known for decades that

the incidence of graft rejection is lower between DZ cotwins than between non-twin full siblings, and this clinical observation has been used to argue that DZ cotwins are genetically more similar to one another than non-twin full siblings (see Geschwind, 1983). Indeed, several studies (Jawaheer, MacGregor, Gregersen, Silman, & Ollier, 1996; Nielsen, Eiberg, Fenger, & Mohr, 1990; Winata, Biegel, Kang, Harmath, & Christian, 1981) have reported that DZ twins are more likely to share HLA haplotypes than non-twin full siblings (cf. Titlestad, Kyvik, Kristensen, & Lillevang, 2002).[4]

2.3. Epigenetic similarity of MZ and DZ twins

In the 1950s, the Central Dogma of molecular biology was that gene expression is a deterministic, unidirectional process whereby a gene encoded in the DNA acts as the template for RNA synthesis (transcription), which is then spliced to produce functional RNA (mRNA) that contains the code for a single protein (translation). By the 1970s, the discovery of reverse transcriptase made it clear that the process was not unidirectional, and the discovery of alternative splicing made it clear that the process was not deterministic because a single gene can code for multiple proteins. By the 1990s, microbiologists had discovered that micro-RNA (which regulates gene expression at the mRNA level by splicing exons, silencing genes, and editing proteins) and other epigenetic processes (e.g., methylation, phosphorylation, glycosylation, acetylation, imprinting, X chromosome inactivation) could alter gene expression without changing the underlying genomic sequence. In addition to playing a crucial role in early development, these epigenetic processes can occur at any point in development as random events or in response to environmental factors (Jaenisch & Bird, 2003), and Fraga et al. (2005) recently reported that epigenetic differences between MZ cotwins increase with age.

Epigenetic processes appear to be responsible for some cases of neuropsychologically discordant MZ twins. For example, Tsujita et al. (1998) compared the DNA of schizophrenia-discordant MZ twins and found two loci that were clearly different. These differences are consistent either with the cotwins having different methylation patterns or with one of the cotwins having a very small mutation. Recently, Petronis et al. (2003) compared the 5′ regulatory region of the dopamine 2 receptor gene in one pair of MZ twins who were concordant for schizophrenia and one pair of MZ twins who were discordant for schizophrenia. They found that the methylation patterns were more similar in the concordant twin pair than in the discordant twin pair, suggesting that the differences in methylation patterns are (at least partially) responsible for the phenotypic differences in MZ twins. X chromosome inactivation is the process in females whereby every second X chromosome in a cell is randomly inactivated

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[4] Because HLA testing was done on blood and DZ twins sometimes exchange blood cells in utero (van Dijk et al., 1996), it is possible that the increased HLA similarity of DZ twins is spurious.

to create cellular mosaics of paternal and maternal X chromosomes. Due to its stochastic nature, differences in X chromosome inactivation patterns are another source of phenotypic discordance in female MZ cotwins. In one study, 19% of female MZ twins had skewed X chromosome inactivation pattern that differed from that of their cotwin (Fraga et al., 2005). Although in the Fraga et al. study, the difference in X chromosome inactivation pattern was not associated with obvious phenotypic discordance, there have been cases of MZ female twins who are carriers for X-linked red-green color blindness (Jorgensen et al., 1992), Duchenne muscular dystrophy (Lupski, Garcia, Zoghbi, Hoffman, & Fenwick, 1991; Richards et al., 1990) and Fragile X syndrome (Kruyer et al., 1994; Willemsen, Olmer, De Diego Otero, & Oostra, 2000), in which one twin is affected and the other is not. In each case, the affected twin had more inactivation of paternal than maternal X chromosomes. Some studies have reported that non-random or skewed X chromosome inactivation patterns are more frequent in MZ twins than in DZ twins or singletons (Goodship, Carter, & Burn, 1996; Trejo et al., 1994). Furthermore, monochorionic twins have much more similar X chromosome inactivation patterns than dichorionic MZ twins (Monteiro et al., 1998). Thus, to the extent that genes on the X chromosome contribute to linguistic ability, we would predict that monochorionic MZ twins would be more similar than dichorionic MZ twins.[5] Loat, Asbury, Galsworthy, Plomin, and Craig (2004) have shown that male MZ twins are more linguistically similar to one another than female MZ twins, a finding that they interpret as reflecting differences in patterns of X inactivation in females MZ twin pairs. They also report that female DZ cotwins are more similar to one another than male DZ cotwins, a finding also consistent with X inactivation. Genetic imprinting, in which the expression of a gene depends upon the parent who passed on the gene, could also account for discordance among MZ cotwins. One known case is Beckwith–Wiedemann syndrome, a genetic syndrome that almost always includes developmental speech impairments secondary to macroglossia (see Van Borsel, Morlion, Van Snick, & Leroy, 2000; Weksberg et al., 2002). In MZ cotwins who are discordant for Beckwith–Wiedemann syndrome, the affected twin has an imprinting defect at KCNQ1OT1 on 11p15, whereas the unaffected twin does not (Weksberg et al., 2002; Weksberg, Shuman, & Smith, 2005).

To the extent that MZ cotwins are epigenetically different from one another, or DZ cotwins are epigenetically more similar to one another than non-twin siblings, heritability estimates for language will underestimate the importance of genetic factors on linguistic abilities. Having already discussed how MZ cotwins can vary epigenetically, let us now turn to the question of whether DZ cotwins are epigenetically more similar to one another than non-twin siblings. Although comprehensive searches of Medline and PsychLit electronic databases (and less

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[5] Fisk, Howard, Ware, and Bennett's (1999) findings suggest that X-inactivation may occur after monochorionic twinning, which means that even monochorionic cotwins can be discordant for X-linked traits.

comprehensive Google searches) failed to reveal any studies that directly address this question, the lower incidence of graft rejection for DZ twins than for non-twin siblings could reflect the greater epigenetic similarity of DZ cotwins than non-twin siblings.

3. Perinatal environmental factors and linguistic development

Researchers who study language acquisition often implicitly assume that when one refers to the role of environmental factors on language development, one is primarily referring to postnatal, psychosocial factors such as the quantity or quality of adult linguistic input that children receive. If psychosocial factors have a large impact on language development, this would support theories that argue that language development is largely the result of children's social and language environments (empiricist/emergentist theories). It would also call into question nativist/biological theories that argue that language acquisition is largely the result of children's innate, biological endowment (nativist/biological theories). If, on the other hand, the environmental factors that affect children's language are predominantly perinatal (and, hence, unlikely to be psychosocial) or clearly biological (e.g., pre- or postnatal accidents, illnesses or toxins that affect the brain), this would support nativist/neurobiological theories, and call into questions empiricist/emergentist theories. A wide range of perinatal complications are associated with adverse neuropsychological outcomes (Bryan, 1993), and women carrying twins suffer more perinatal complications than women carrying a single baby (Creasy, Resnik, & Iams, 2004). In this section, we review some of the perinatal complications associated with twinning, concentrating on those that have been linked to neurodevelopmental delays.

3.1. Premature birth

A large body of research has documented that, as a group, children who are born prematurely (before 37 weeks gestation) reach speech and language milestones later, perform more poorly on a wide range of speech and language tests, and are more likely to be diagnosed with speech and language disorders than their full-term peers (see, for example, Briscoe, Gathercole, & Marlow, 1998; Jennische & Sedin, 1999; Luoma, Herrgard, Martikainen, & Ahonen, 1998; Stevenson, Roach, Leavitt, Miller, & Chapman, 1988; Taylor, Klein, & Hack, 2000a; Taylor, Klein, Minich, & Hack, 2000c, and references therein). These studies reveal that the more premature the child, the greater the risk of poor linguistic skills, but that even children born between 32 and 36 weeks gestation do more poorly than children born full term (Hediger, Overpeck, Ruan, & Troendle, 2002; Huddy, Johnson, & Hope, 2001). Even preterm children with normal cognitive function and no major neurodevelopmental disability are 2–3 times more likely to suffer from written and spoken language disorders than

full-term children. This suggests that language may be particularly vulnerable to factors associated with premature birth.[6] In the United States, over half of all twins are born prematurely, whereas only about 10% of singletons are born prematurely (Guyer et al., 1999). In addition, twins are more likely to be born very prematurely (before 32 weeks gestation) than singletons (Holmgren & Hogberg, 2001). The greater incidence of prematurity among twins than singletons probably is part of the reason why twins are more likely to be language impaired than singletons and why even normal twins' language development lags behind that of singletons.

3.2. Low birth weight

Low birth weight is a risk factor for linguistic delays and impairments independent of prematurity.[7] For example, even low birth weight children who are full term are more likely to suffer from language and learning disorders than normal birth weight full-term children (Low et al., 1992; Walther, 1988). Multivariate regression analyses reveal a significant positive relationship between birth weight and performance on a variety of linguistic tasks even at birth weights above 3000 g (Breslau, Chilcoat, DelDotto, & Andreski, 1996; Breslau, Chilcoat, Johnson, Andreski, & Lucia, 2000). Even low birth weight full term children who are apparently neurologically intact do worse on language tasks than normal birth weight children (Breslau et al., 1996; Breslau et al., 2000; Low et al., 1992; Walther, 1988), suggesting that language may be particularly vulnerable to factors associated with low birth weight.

In the US, twins are 10 times more likely to be born at low birth weights than singletons (Guyer et al., 1999), and low birth weight adversely affects linguistic outcome in twins as well as singletons. For example, Rooney, Hay, and Levy (2003) has shown that twins with low birth weights have lower speech and reading scores than other twins, and that the negative impact of low birth weight is particularly pronounced in male twins. Given that low birth weight has a detrimental affect on language development, the greater incident of low birth weight in twins than singletons is probably partially responsible for twin-singleton language lag. In addition, differences in MZ cotwins' birth weights could account for some cases of linguistically discordant MZ twin pairs.

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[6] Premature birth per se is unlikely to be the cause of linguistic delays because, in theory, development in the neonatal intensive care unit (NICU) could be identical to development in the womb. Rather, the linguistic delays are likely due to the conditions that result in premature delivery (e.g., intrauterine infection, placental dysfunction, thrombophilila, gestational diabetes, pregnancy-induced hypertension, etc.) and/or the conditions and treatments that are secondary to premature delivery (e.g., hypoxic brain injuries, hyperbilirubinemia, postnatal infection, respiratory distress syndrome, treatments and medications received in the NICU, developmentally inappropriate sensory stimulation in the NICU, etc.). Some of the most likely candidates are discussed in Sections 3.2–3.9.

[7] The linguistic delays could be the direct result of being low birth weight (i.e., the effect of having a smaller brain) or it could be the result of perinatal conditions associated with being low birth weight.

3.3. Placental and amniotic complications

Placental abruption refers to the condition in which the placenta separates from the uterus prior to delivery. This can result in maternal hemorrhage and placental dysfunction, both of which cause decreased blood flow to the fetus. Diminished fetal blood flow is a major risk factor for prenatal brain injuries. Consistent with this, recent studies have shown that placental abruption is associated with neuropsychiatric disorders (Jablensky, Morgan, Zubrick, Bower, & Yellachich, 2005) and neurodevelopmental disorders such as cerebral palsy (O'Shea & Dammann, 2000). Premature rupture of membranes (PROM) refers to the condition in which the amniotic sac ruptures prior to delivery. There are three reasons why twins' higher incidence of PROM has important ramifications for twin studies of language. First, PROM is associated with intrauterine infection (Simhan & Canavan, 2005),[8] and intrauterine infection is a major causal factor for developmental disabilities (see Section 3.4). Second, umbilical cord prolapse (when the cord enters the birth canal before the baby) is a frequent complication of PROM (Simhan & Canavan, 2005). When this occurs, the cord is compressed and blood flow to the baby is diminished, thereby causing hypoxic/ischemic brain injuries. Lastly, PROM is a predictor of perinatal mortality and morbidity in twins independent of infection or cord prolapse (Vergani et al., 2004). Because the incidence of both PROM (Mercer, Crocker, Pierce, & Sibai, 1993) and placental abruption (Ananth, Smulian, Demissie, Vintzileos, & Knuppel, 2001; Salihu et al., 2005) is twice as high in twins than in singletons, these complications are likely partially responsible for twins' linguistic problems. In addition, for MZ cotwins who do not share a placenta (diachorinic twins) or amniotic sac (diamniotic twins), amniotic and placental complications can affect one cotwin and not the other and this could cause some MZ cotwins to be linguistically discordant.

3.4. Intrauterine infection

There is growing evidence that pro-inflammatory cytokines produced in response to perinatal infection damage the developing brain (Rezaie & Dean, 2002; Volpe, 2001). Indeed, recent studies suggest that intrauterine infection is the major risk factor for major neurodevelopmental disabilities such as cerebral palsy (O'Shea & Dammann, 2000; O'Shea, Klinepeter, Meis, & Dillard, 1998; Wilson-Costello et al., 1998; Wu & Colford, 2000) and an important risk factor for more subtle impairments such as linguistic and cognitive delays (Dammann, Drescher, & Veelken, 2003; Schendel, 2001). The incidence of intrauterine infection is greater in twins than singletons because twins are at greater risk for several conditions that increase the chances of infection. First, the incidence of incompetent cervices and PROM is higher in twins, increasing the risk of vaginal pathogens ascending and coming into contact with the

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[8] There is a synergistic effect between intrauterine infection and PROM. PROM can be caused by intrauterine infection and mothers with PROM are at greatly increased risk for intrauterine infection (Simhan & Canavan, 2005).

developing fetus. Second, women carrying twins usually have longer labors, so even when the membranes do not rupture prior to the onset of labor, twins (particularly the second born twin) are exposed to vaginal pathogens longer than singletons. Third, mothers of twins are more likely to have frequent and invasive vaginal examinations and intrapartum monitoring, either of which may introduce new pathogens or allow existing pathogens to ascend more easily. That intrauterine infection is a major risk factor for major and minor developmental disabilities and that this complication is seen more frequently in twins than singletons may partially account for the higher rates of linguistic delays and impairments in twins than singletons. Furthermore, some diamniotic MZ cotwins could be linguistically discordant because one twin was infected in utero and the other was not.

3.5. Intrapartum complications

Intrapartum (labor and delivery) complications are also associated with adverse neurodevelopmental outcome (Bryan, 1993). The main worry with intrapartum complications is that the neonate will become hypoxic during labor and delivery. Vaginal deliveries are most straightforward when the head of the baby is facing the birth canal (vertex presentation). Perinatal morbidity and mortality rates are higher with non-vertex deliveries than vertex deliveries (Cetrolo, 1986; Chervenak, Johnson, Youch, Hobbins, & Berkowitz, 1985; Piekarski, Czajkowski, Maj, & Milewczyk, 1996), and non-vertex presentation is a risk factor for neurodevelopmental disorders such as cerebral palsy (Bryan, 1993).[9] Between one quarter to one third of twin fetuses are non-vertex at the time of delivery as compared to less than 5% of singleton fetuses (Chervenak et al., 1985; Piekarski et al., 1996). Another intrapartum complication associated with hypoxic/ischemic brain injuries and very poor neurodevelopmental outcome is velamentous umbilical cord insertion (Benirschke & Kim, 1973). In velamentous umbilical cord insertion, fetal vessels may tear when the amniotic membranes rupture, and this can result in the baby losing massive amounts of blood. Fetal vessels can also be compressed during labor, resulting in diminished or absent blood flow to the baby (Chervenak et al., 1985; Piekarski et al., 1996). The incidence of velamentous cord insertion is much higher in twin than singleton gestations (Loos, Deroma, Deroma, & Vlietincka, 2001). Umbilical cord prolapse during labor and delivery is also more common in twin pregnancies than in singleton pregnancies (Usta, Mercer, & Sibai, 1999). Because essentially all intrapartum complications are more common in twin pregnancies (Creasy et al., 2004) and many intrapartum complication are associated with major and minor developmental disabilities, some of twins' linguistic lags are likely due to intrapartum complications. In addition, discordant intrapartum experiences could cause some MZ cotwins to differ linguistically.

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[9] Some researchers have suggested that fetuses with neurodevelopmental abnormalities are less able to turn in the womb and, thus, breech babies are at greater risk for neurodevelopmental disabilities in part because breech presentation is a marker for these abnormalities (Kuban & Leviton, 1994).

3.6. Neonatal hyperbilirubinemia

Bilirubin that is not bound to albumin is a potent neurotoxin, and neonatal hyperbilirubinemia (jaundice) is a major cause of neurodevelopmental disability (Dennery, Seidman, & Stevenson, 2001). Although hyperbilirubinemia can affect any part of the central nervous system, the auditory pathways are particularly vulnerable (Shapiro, 2002; Spencer, Shaia, Gleason, Sismanis, & Shapiro, 2002), and sensorineural hearing loss and auditory dysfunction are the most common major sequelae of neonatal hyperbilirubinemia (Amin et al., 2001; Hack et al., 2000). Neonatal hyperbilirubinemia may be a contributing factor for some cases of language impairments because even very minimal hearing losses (hearing thresholds of between 16 and 25 dBs) put children at increased risk for language delays and impairments (Bess, 1999; Finitzo-Hiever, 1981; Gravel, Wallace, & Ruben, 1996; Loutonen, Uhari, & Aitola, 1996; Yoshinaga-Itano, Sedey, Coulter, & Mehl, 1998).

Premature and low birth weight infants are at greatly increased risk of being hyperbilirubinemic because their small, immature livers have difficulty clearing bilirubin from the bloodstream (Dennery et al., 2001). For a given level of bilirubin, the neurotoxicity depends on factors such as blood brain barrier permeability (which is increased in premature and low birth weight babies, and in the presence of hypoxia or infection), albumin levels (which are lower in premature and low birth weight babies), and blood pH (the acidosis that accompanies hypoxia and infection impairs bilirubin conjugation). Because twins are more likely than singletons to be premature, low birth weight, hypoxic, and infected, neonatal jaundice affects a higher percentage of twins than singletons. The differential incidence of jaundice in twins than singletons probably accounts for some portion of the twin-singleton language lag. Additionally, some cases of linguistically discordant MZ cotwins could be the result of the cotwins having different levels of bilirubin during the first weeks of life.

3.7. Perinatal glucocorticosteroids

Many studies have investigated the adverse effects of excess glucocorticosteroids (GCs) on the developing nervous system (for a review, see Matthews, 2000). Women who seem likely to deliver prematurely are often given synthetic GCs because prenatal GCs lessen the risk of neonatal respiratory distress syndrome and neonatal hypoxia (Liggins & Howie, 1972), thereby decreasing the rate of short- and long-term morbidity. Adults and children who were born prematurely and received a single course of prenatal steroids do better on verbal tests than those who received no steroids (Dessens, Haas, & Koppe, 2000; Stromswold, Sheffield, & Eisenband, 2003). However, recent studies have found that multiple courses of prenatal GCs are associated with greater infant morbidity than a single course of prenatal GCs. For example, babies exposed to weekly prenatal GCs are 2.5 times more likely to have head circumferences below the 10th percentile (Praveen & Roopa, 2005) and 4 times more likely to have severe intraventricular hemorrhages (Guinn et al., 2001) than babies exposed to one course of GCs. Consistent with these results, Stromswold et al. (2003) found that children exposed to multiple courses of prenatal GCs have worse

language outcomes than children exposed to a single course of prenatal GCs, and that multiple courses of GCs hurt language development more than gross motor, fine motor, social or cognitive development.[10] Neonatal steroids also adversely affect neurodevelopmental outcome. Compared to preterm infants who receive no GCs in the neonatal intensive care unit (NICU), preterm infants who receive GCs in the NICU have smaller head circumferences and brain volumes (Murphy et al., 2001; Stark et al., 2001), and are 3 times as likely to have cerebral palsy and twice as likely to have a major neurodevelopmental impairment (Barrington, 2001a, 2001b). Particularly relevant, children who receive neonatal GCs have worse linguistic outcomes than children who don't (Short et al., 2003; Stromswold & Sheffield, 2003). Twins are more likely than singletons to be exposed to excess perinatal GCs for several reasons. First, mothers carrying twins are more likely to deliver prematurely, and therefore, they are more likely to be given exogenous GCs. Second, twins are more likely to be born prematurely and/or small and, hence, they are more likely to receive GCs in the NICU. Third, twins are more likely to suffer intrauterine growth restriction, and cortisol levels are higher in growth restricted fetuses (Goland, Conwell, & Jozak, 1995; Goland et al., 1993). Given the probable association between excess perinatal GCs and poorer developmental outcome, their greater use in twins than singletons could partially explain why twins have more linguistic problems than singletons. It is also possible that differential exposure to postnatal GCs causes some cotwins to be linguistically discordant.

3.8. Neonatal sensory environments

Children who are born early are exposed to visual stimuli and high frequency sounds prematurely, and some studies have demonstrated negative effects of this type of early sensory stimulation. For example, quails that receive visual stimulation prior to hatching fail to respond appropriately to maternal visual cues, continue to respond to maternal auditory cues into later stages of postnatal development and fail to learn prenatally their mother's unique call (Lickliter, 1990; Sleigh & Lickliter, 1995). Because their mother's body attenuates sounds above 250 Hz (Lecanuet, 1998), while in the womb, human fetuses are preferentially exposed to the low frequency sounds used to convey prosody and only after birth are they exposed to the high frequency sounds used to convey phonemes, words and sentences. We know that training simple recurrent neural networks on input filtered in such as way as to

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[10] Although we do not know why language is particularly vulnerable to excess perinatal GCs, results of animal studies provide some intriguing possibilities. These studies have shown that the developing hippocampus is exquisitely vulnerable to excess GCs (Takahashi, 1998; Uno et al., 1994; Weinstock, 1997). Although not traditionally considered a language area, the hippocampus has been implicated in normal language processing (Brockway, 1999) and injuries to the hippocampus are associated with language disorders (Brockway, 1999; DeLong, 1992; Mackay, Stewart, & Burke, 1998). Animal studies have also revealed that the developing cerebellum is especially sensitive to the deleterious effects of GCs (Ferguson & Holson, 1999; Gramsbergen & Mulder, 1998; Howard, 1968). Again, although the cerebellum is not a language area per se, the cerebellum has been implicated in language and language disorders (Justus, 2004; Leiner, Leiner, & Dow, 1993; Silveri & Misciagna, 2000).

simulate the auditory experience of fetuses in the womb enhances the networks' later ability to perform syntactic tasks (Christiansen & Dale, 2001). Not only do preterm twins have less opportunity for this type of phased linguistic learning, many preterm and low birth twins spends weeks or months in NICUs where average background noise levels range from 60 to 90 dB. In their review of the literature, the American Academy of Pediatrics Committee on Environmental Health (1997) concluded that exposure to excessive noise in the NICU can cause cochlear damage, poor growth, and poor development in neonates. (For further discussion of the effects of NICU noise on language, see Stromswold & Sheffield, 2004.)

Because twins are more likely to be born preterm, low birth weight and suffer from many prenatal, intrapartum and postnatal complications than singletons, they are more likely to spend time in the NICU. Thus, if exposure to developmentally inappropriate sensory stimuli adversely affects development, this may be part of the reason why twins' language tends to lags behind that of singletons. Some cases of linguistically discordant MZ twins could also be the result of MZ cotwins having different neonatal sensory experiences.

3.9. Brain injuries to language areas

Perinatal complications are worrisome because they tend to cause hypoxic/ischemic brain injuries (Volpe, 2001). Because perisylvian language areas are in a vascular watershed, they are particularly vulnerable to hypoxic/ischemic injury. Perinatal hypoxia can also damage the central nervous system auditory pathways and the cochlea (Jiang, Wang, Brosi, Shao, & Wilkinson, 2004a, 2004b), and hypoxia potentiates noise-induced hearing loss (Chen, 2002). Oligodendroglia that are in the process of myelination are most vulnerable to hypoxic injury (Perlman, 1998; Volpe, 2001), and the late myelination of the temporal poles (Inder & Huppi, 2000) means that many of the brain regions that subserve language are in a vulnerable state for an extended period.

Because their brain vasculature is less mature, the incidence of perinatal hypoxic/ischemic brain injuries is much higher for premature neonates than full-term neonates (Perlman, 1998). It is well established that preterm children with severe brain injuries (periventricular leukomalacia and high-grade intraventricular hemorrhages, IVHs) have worse neurodevelopmental outcomes compared with preterm children without severe brain injuries. More recently, MRI studies of preterm infants have shown that even low-grade IVHs (which are traditionally said to have no clinical significance) are associated with marked reductions in cortical volumes (Vasileiadis et al., 2004). Furthermore, even if their neonatal ultrasounds were perfectly normal (i.e., no evidence of even low-grade IVHs), preterm children who suffered intrapartum hypoxia have worse cognitive and linguistic outcomes than preterm children who were not hypoxic (Hopkins-Golightly, Raz, & Sander, 2003).

Volumetric MRIs reveal that preterm children have significantly smaller cortices (particularly sensorimotor, premotor, and midtemporal cortices), basal ganglia, amygdala, hippocampus, and cerebellums than full-term children (Peterson et al., 2000). Furthermore, sensorimotor and midtemporal cortical volumes (i.e., cortical

regions that include language areas) are positively associated with full scale, performance, and verbal IQs in preterm children (Peterson et al., 2000). A recent study confirms that the cortical areas that subserve written and spoken language are smaller in preterm children than full-term children, and shows that the reduction in size is particularly pronounced in preterm boys (Reiss et al., 2004).[11] This is particularly interesting in light of the fact that preterm boys are more likely to be language-delayed than girls born at the same gestational age and weight (Brothwood, Wolke, Gamsu, Benson, & Cooper, 1986; Hindmarsh, O'Callaghan, Mohay, & Rogers, 2000; Ornstein, Ohlsson, Edmonds, & Asztalos, 1991). In addition to having smaller perisylvian language areas than full term children, premature children's brains process language differently than full term children. For example, an fRMI study has shown that preterm children process spoken words in the same way that term children process strings of meaningless sounds, and fMRI signal during semantic processing correlates with verbal IQ scores in preterm but not full-term children (Peterson et al., 2002). The higher incidence of prematurity and frank neurodevelopmental disability in twins than singletons almost certainly means that twins suffer (sometimes subtle) hypoxic brain injuries to language and auditory pathways more frequently than singletons. Furthermore, if one MZ cotwin suffers a brain injury and the other does not, the cotwins will likely differ linguistically.

3.10. Differences in perinatal hardships for MZ and DZ twins

Although the perinatal hardships discussed above are risk factors for language delays and disorders, the linguistic abilities of children who experience these hardships vary tremendously, with the variance increasing as the severity of the perinatal hardship increases. At one end of the spectrum, children may have perfectly normal linguistic development, whereas at the other end of the spectrum children may suffer from devastating linguistic disorders. Consider, for example, birth weight. About a third of children born at moderately low birth weights (between 1500 and 2500 g) are linguistically normal and two-thirds are linguistically impaired (Huddy et al., 2001) and about half of children born at very low birth weights (1000–1500 g) are linguistically normal and half are linguistically impaired (Vohr & Msall, 1997). Even a quarter of children born weighing less than 800 appear to be linguistically normal (Whitfield, Grunau, & Holsti, 1997). As these statistics reveal, children born at lower birth weights are clearly at greater risk for language impairments than higher birth weight children. That said, children's linguistic abilities vary tremendously even within birth weight levels, with the variance being greater the lower the birth weight (Grunau, Kearney, & Whitfield, 1985). To the extent that MZ twins experience more severe perinatal hardships than DZ twins, MZ cotwins will tend to be more linguistically different from one another than DZ cotwins and this will depress twin-based estimates of the role of genetic factors in language development.

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[11] Reiss et al. (2004) suggest that the sex difference could reflect the protective effect of either female sex hormones or genes located on the X chromosome.

It has been recognized for a long time that MZ are at greater risk than DZ twins for many perinatal complications (e.g., Lenneberg, 1967). Sharing a placenta is a major risk factor for neuromorbidity in twins, with the rate of neurodevelopmental disability being 5–7 times greater for monochorionic twins (80% of MZ twins, and ~0% of DZ twins) than for dichorionic twins (Adegbite, Castille, Ward, & Bajoria, 2004). In addition, monochorionic twins are born very preterm twice as often as dichorionic twins (Sebire, Snijders, Jighers, Sepulveda, & Nicolaides, 1997). Male MZ twins are more likely to be preterm than female MZ twins, whereas there doesn't appear to be a male/female difference for same-sex DZ twins (Bryan, 1993). Thus, male MZ twins are at greatest risk for language impairments associated with prematurity. As a group, MZ twins also weigh less than DZ twins born at the same gestational age (Ananth, Vintzileos, Shen-Schwartz, Smulian, & Lai, 1998; Senoo et al., 2000; Victoria, Mora, & Arias, 2001). Monochorionic MZ twins weigh less than dichorionic MZ twins (Ananth et al., 1998; Senoo et al., 2000; Victoria et al., 2001) who in turn weigh less than DZ twins (Corney, Robson, & Strong, 1972). Given the inverse relationship between linguistic ability and both gestational age and birth weight, we would predict poorer and more variable performance for MZ twins than DZ twins. This will result in twin studies underestimating the importance of heritable factors on language, and some MZ twins being linguistically discordant.

3.11. Discordance of perinatal environments within MZ and DZ twin pairs

Many researchers have pointed out that the role of genetic factors will be overestimated if MZ cotwins have more similar environments than DZ cotwins. Researchers have generally assumed that MZ cotwins either have equally similar or more similar environments than DZ cotwins. With respect to perinatal environment, MZ cotwins may have *less* similar environments than DZ cotwins. If perinatal hardships can affect one MZ cotwin and not the other or if MZ cotwins experience perinatal hardships to different degrees, this could explain why some MZ twins are linguistically discordant and why heritability estimates are usually less than .60. In this section, we review some of the perinatal hardships that may affect one member of a twin pair more than another.

As discussed in the previous section, MZ twins typically weight less than DZ twins. In addition, the intra-twin pair differences in birth weight are greater for monochorionic twins (i.e., most MZ twins) than dichorionic twins (essentially all DZ twins), and the difference remains significant even when the sex of twins is factored in (Vlietinck et al., 1989). This is critical because, even among twins who are full term and appropriate size for their gestational age, having discordant birth weights is an independent risk factor for poor neonatal outcome (Amaru, Bush, Berkowit, Lapinsk, & Gaddipat, 2004, 2001), perhaps because birth weight discrepant twins tend to have higher rates of intraventricular hemorrhage than non-BW discrepant twin (Amaru et al., 2004). Because 95% of monochorionic MZ twins have placental vascular anastomoses (Machin, 2004), with respect to intrauterine exposure to infection and toxins, monochorionic MZ cotwins are more likely to be more similar than dichorionic MZ or DZ cotwins (Davis & Phelps, 1995; Phung et al., 2002). There are

also intrauterine environmental reasons why placental vascular anastomoses might cause monochorionic MZ twins to be *less* similar than dichorionic MZ or DZ twins (Machin, 2004). Because of placental vascular anastomoses, 15–20% of monochorionic MZ twins suffer from twin–twin transfusion syndrome (TTTS) in which one twin acts as the donor and the other acts as the recipient of fetal blood (Machin, 2001, 2004). Diminished blood flow to the donor twin can further increase the risk of hypoxic/anoxic injuries, whereas the increased blood flow to the recipient twin can result in congestive heart failure that secondarily can cause neural injuries (Benirschke, 1995; Machin, 2001; Maier et al., 1995; Pharaoh, 2002). In addition, donor twins usually are anemia which can lead to fetal growth restriction, cellular malnutrition and hypoxic/ischemic injuries (particularly of the brain), whereas recipient twins are usually polycythemic (they have too many red blood cells), and this can result in sluggish blood flow which increases the chance of thrombotic injuries (particularly of the brain) and hyperbilirubinemia (from the breakdown of excess red blood cells). Gratacos et al. (2004) report that twins with TTTS are 4 times more likely to suffer brain damage than twins without TTTS, and that among twins with TTTS, the recipient twin is 7 times more likely to suffer brain damage than the donor twins.

Empirically, the relationship between chorionicity and phenotypic similarity is complex. On the one hand, monochorionic MZ cotwins look less like one another (Forget-Dubois et al., 2003) and are more discordant for birth weight and congenital anomalies (Machin, 2004) than dichorionic MZ cotwins. On the other hand, some studies suggest that monochorionic MZ twins have more similar personalities (Sokol et al., 1995) and verbal and non-verbal cognitive abilities (Gutknecht, Spitz, & Carlier, 1999; Jacobs et al., 2001) than dichorionic MZ twins. Using differences in fingerprint patterns as an index of chorionicity, Reed, Pfefferbaum, Sullivan, and Carmelli (2002) have argued that monochorionic MZ twins have more similar corpus callosa than dichorionic MZ twins and Davis, Phelps, and Bracha (1995) have argued that monochorionic MZ twins are more likely to be concordant for schizophrenia than dichorionic MZ twins. It is possible that the effects of chorionicity depend on whether one is studying somatic or neuropsychological similarity. Because monochorionic twins are genetically and epigenetically more similar than dichorionic MZ twins (see Sections 2.2 and 2.3), if monochorionic MZ twins are neuropsychologically more similar to one another, this could be taken as evidence that genetic factors are more important for neuropsychological development, and prenatal environmental factors are more important for somatic development. A second possible explanation for the apparent differential effects of chorionicity is that extremely discordant twin pairs are more likely to be excluded from studies of neuropsychological traits than studies of somatic traits. The reason is that studies of neuropsychological similarity only include MZ twin pairs in which both twins survive into adulthood and are testable, whereas studies of somatic similarity typically study infants and do not involve testing the twins. Because twins in studies of neuropsychological traits are older than the twins in studies of somatic traits, a third possible explanation is that monochorionic MZ twins become neuropsychologically more similar as they age.

About 95% of MZ twins are diamniotic, and diamniotic twins may have different perinatal experiences by virtue of the fact that each twin has his or her own amniotic sac. As was the case for chorionicity, the relationship between amnionicity and phenotypic similarity is complex. When twins have separate amniotic sacs, sometimes only one cotwin will have too little amniotic fluid, a condition which is associated with fetal deformities (Hatkar & Bhide, 1999; Luke & Keith, 1990; MacGillivray, 1986; Machin, 2001; Sherer, 2001). Furthermore, twins who have separate amniotic sacs may be exposed to different toxins and infections, whereas twins who share an amniotic sac are exposed to the same toxins and infections (e.g., Arai et al., 2002). Because perinatal infection is a major risk factor for developmental disorders (see Section 3.4), some cases of the neurodevelopmental discordance in MZ twins could be the result of the MZ twins having two amniotic sacs. On the other hand, MZ twins who share an amniotic sac are likely to become interlocked or entangled in one another's umbilical cords, and half of all monoamniotic pregnancies end with the death of one or both twins (Benirschke, 1995). Even when both monoamniotic MZ twins are born alive, the chance that at least one of the twins has an ischemic/hypoxic brain injuries is very high (Benirschke, 1995).

To the extent that the prenatal and intrapartum hardships of MZ cotwins differ, the cotwins will have different neonatal experiences. The MZ cotwin who experiences more hardships will almost certainly have a rockier neonatal period, suffering more of the neonatal complications that are associated with poor neurodevelopmental outcome such as chronic lung disease (Vohr et al., 2000), jaundice (Hack et al., 2000), sepsis (Wilson-Costello et al., 1998), and necrotizing enterocolitis (Vohr et al., 2000). Consequently, he is likely to receive drugs (e.g., postnatal steroids, Vohr et al., 2000) and treatments (e.g., ventilator use, Wilson-Costello et al., 1998) that are themselves independent risk factors for neurodevelopmental disability. Differences in the neonatal experiences of some MZ cotwins almost certainly cause the cotwins to be neurologically discordant, and this could explain why some MZ cotwins have very different linguistic abilities. In addition, the greater intra-twin pair differences in perinatal environment for MZ cotwins than DZ cotwins is likely to result in the role of genetic factors in language being underestimated in twin studies.

3.12. Summary: Perinatal hardships, discordant MZ cotwins, and heritability estimates

The perinatal environmental hardships associated with twinning result in twins being much more likely than singletons to be born premature and/or low birth weight, to suffer from intrauterine and neonatal infections, premature rupture of membranes, umbilical cord prolapse, exsanguination (due to vasa previa or placental abruption), neonatal hyperbilirubinemia, and to be exposed to perinatal corticosteroids and developmentally inappropriate sensory stimuli. The result is that perinatal mortality in twins is 2–4 times higher than in singletons (Machin, Bramforth, Innes, & Minichul, 1995, 1997). In addition, twins are more likely than singletons to have congenital malformations (Little & Bryan, 1988; Luke & Keith, 1990; Pharaoh, 2002) and neurodevelopmental disabilities (including language and learning disabilities) than singletons (Bryan, 1993; Pharaoh, 2002). Taken as a group, the perinatal

hardships associated with twinning result in some twins suffering overt or covert damage to the central and peripheral neural structures involved in language. Thus, perinatal hardships are almost certainly (part of) the reason why twins' language development lags behind that of singletons. In addition, because cotwins can differ in the perinatal complications they experience (or the degree to which they suffer these complications), perinatal complications almost certainly account for some cases of linguistically discordant MZ cotwins. Lastly, because MZ cotwins are more likely to have discordant perinatal environments than DZ cotwins, twin studies probably underestimate of the role of genetic factors in language development.

4. Postnatal environment and language development

4.1. Linguistic input and language development

Some studies have shown that postnatal environment affects language development. For example, some researchers (Hoff & Naigles, 2002; Huttenlocher, Haight, Bryk, Seltzer, & Lyons, 1991) report that children who receive more linguistic input have larger vocabulary and acquire new vocabulary items more easily than children who receive less adult linguistic input. Other studies have shown that children who receive syntactically more complex adult input develop syntax faster than those who receive less syntactically complex input (Huttenlocher, Vasilyeva, Cymerman, & Levine, 2002). Some studies (Fenson et al., 1994; Hoff-Ginsberg, 1998; Pine, 1995) have found that first born children have slightly larger vocabularies than later-born children (but see Bornstein, Leach, & Haynes, 2004), a finding usually attributed to first-born children receiving more adult linguistic input than later-born children. Some studies have also found that socio-economic status (SES) affects children's lexical development, with high SES being associated with more advanced language development (Hoff, 2003; Landry, Smith, & Swank, 2002; Largo, Pfister, Molinari, & Kundu, 1989; Vohr, Garcia-Coll, & Oh, 1989). Lastly, mothers with more education may talk to their children more than those with less education (Hoff-Ginsberg, 1994), and some studies have shown that children with more educated mothers acquire language faster than those with less educated mothers (Fenson et al., 1994; Hoff-Ginsberg, 1998). It should be noted, however, that studies of perinatally at-risk children have generally shown that neonatal factors play as great a role on language development as postnatal factors (see Hershberger, 1996; Largo et al., 1989; Resnick et al., 1998; Landry et al., 2002; Vohr et al., 1989).

4.2. Twins' linguistic input and language delays

Postnatally, because twins have a sibling of the same age, twins usually receive less adult linguistic input than singletons (Reznick, 1997; Stafford, 1987; Tomasello, Mannle, & Kruger, 1986), and mothers of twins engage their twins in fewer verbal exchanges than mothers of singletons (Conway et al., 1980; Lytton, Conway, & Suave, 1977; Thorpe, Rutter, & Greenwood, 2003). For example, Conway et al.

(1980) found that differences in complexity and frequency of maternal speech accounted for almost twice as much of the variance in children's language development as differences in APGAR scores, gestational age at birth, or birth weight. Although Conway et al.'s (1980) results are frequently cited as showing that perinatal factors play little role in twins' language development, there are several reasons to question this conclusion. First, the study had only 12 twin pairs. Second, the twins had very benign perinatal histories (they were born an average of 2 weeks later and 400 g heavier than the mean for U.S.-born twins). Third, there was less variance for neonatal variables than maternal variables, and this may have decreased the predictive power of the neonatal variables. Lastly, because the study did not distinguish between MZ and DZ twins, and twin and singleton data were collapsed in the regression analyses, these data cannot be used to evaluate the relative importance of neonatal versus maternal variables on twins' language.

Two recent papers have explored the extent to which perinatal and postnatal factors account for the linguistic lag of twins relative to singletons (Rutter et al., 2003; Thorpe et al., 2003). Thorpe et al. (2003) have argued that maternal communicative variables (such as the extent to which mothers talk to their child, read to their child, elaborate their speech, etc.) account for much of the twin-singleton lag in neurologically intact twins born at 33 weeks or above. Although generally sound, there are several worrisome features of the study. First, because they looked at many potential maternal/familial factors, selected those 6 factors that were most predictive of language development, and then summed these factors to create a composite factor, Thorpe et al. (2003) likely inflated the importance of maternal communicative behaviors in explaining the twins' linguistic lag. Furthermore, because the intent of the study was to determine which factors account for the twin-singleton language lag, they eliminated from consideration factors that occurred at equal rates for twins and singletons. Thus, it is likely that they eliminated factors that have an impact on language development, but affect both twins and singletons.

The companion paper (Rutter et al., 2003) argues that prenatal and obstetric complications play no role in the twin-singleton language lag. In light of the many studies that have shown that perinatal factors have a profound effect on language development, how can this be? A careful reading of Rutter et al.'s (2003) paper reveals that the study suffers from a host of serious problems, and these problems are probably why perinatal factors appear to play no role in language development. For example, by eliminating any twins who were born before 33 weeks gestation, had clinical signs of neurological damage, or had evidence of brain damage on neural ultrasounds or MRIs, Rutter et al. (2003) removed exactly those twins for whom perinatal factors are most likely to play a large role. The second problem is that the authors provide very little information about the perinatal characteristics of the children in the sample. For example, we are not told what the means or standard deviations are for twins' or singletons' birth weights (BWs), gestational ages at birth (GAs), composite perinatal indices, or any of the measures that goes into the composite perinatal indices. It is possible that lack of variability for these factors preclude them from having a statistically significant effect on language. Third, in their analyses, the authors used BWs and GAs that were normalized on the BWs and GAs of the children in the

study. However, because Rutter et al. (2003) normalized the twins' and singletons' data separately (i.e., twins' values were normalized using only twin data, singletons' values were normalized using only singleton data) and twins are born an average of 4 weeks earlier and 500 g lighter than singletons, Rutter et al.'s (2003) normalization scheme assumes their conclusion (that BW and GA do not account for the twin-singleton language lag), and probably obscures any effects of BW and GA. Fourth, the formula that Rutter et al. (2003) use to quantify the degree of BW discordance is not the standard one, precisely because it has the effect of treating cotwins who weighed 1500 and 2000 g and cotwins who weighed 3000 and 3500 g the same, even though in the former case the smaller twin's weight is 75% of the larger twin's weight, whereas in the latter case the smaller twin's weight is 86% of the larger twin's. Doing this almost certainly obscures the effect of discordant BWs. Fifth, the authors use three composite indices of perinatal risk factors, each of which includes many perinatal factors that have been shown to have little or no impact on neurodevelopment (e.g., 1 min APGAR score, perineal lacerations, labor induction) and excludes many perinatal factors that have been shown to affect neurodevelopment (e.g., infection, hypoxia, BW, and hyperbilirubinemia). In addition, the composite measures include measures that are subjective, composite measures that cover an unspecified set of factors that could vary greatly in clinical severity (e.g., ''complications during labour''). In short, the adequacy of the composite measures of perinatal risk contrasts sharply with the adequacy of the composite measure of psychosocial risk, in which the most predictive factors of language for this sample were selected and then combined to form a composite measure.

4.3. Twins' linguistic input, heritability estimates and discordant MZ twins

Even if the way mothers speak to twins (partially) accounts for twins' linguistic delays, this does not necessarily mean that linguistic input affects heritability estimates. Many researchers have suggested that MZ cotwins may have more similar postnatal environments than DZ cotwins because people may treat cotwins who look, act or sound alike (i.e., MZ twins) more similarly than twins who look, act or sound different from another (i.e., DZ twins). If MZ cotwins do have more similar linguistic environments than DZ cotwins, heritability estimates will be inflated. Heritability estimates will be further accentuated if people treat MZ cotwins more similarly than DZ cotwins *and* there are gene-environment interactions (see Section 6). It is also possible that the linguistic environment of MZ cotwins is *less* similar than that of DZ cotwins. If mothers prefer one twin to the other, they may provide more or better linguistic input to the preferred twin. Minde, Corter, Goldberg, and Jeffers (1990) reported that the majority of the mothers in their study of premature twins admitted to preferring one twin within 2 weeks after birth. In general, mothers preferred the healthier, heavier infant, and this preference was stable for at least 4 years (Minde et al., 1990). Another larger study found that differences in MZ cotwins' health affected mothers' feelings for the twins (Caspi et al., 2004). It is possible that differential maternal treatment accentuates differences between sick versus healthy twins and contributes to some cases of linguistic discordance in MZ twins.

Furthermore, as discussed in Section 3.11, intra-twin pair differences in neonatal health and size are generally greater for MZ twins than DZ twins. This could result in mothers of MZ twins treating their twins more differently than mothers of DZ twins, thereby reducing heritability estimates. Notice, however, that these intra-twin pair differences in postnatal environment are secondary to perinatal environmental differences and, hence, ultimately perinatal environment is what affects heritability estimates.

There are several strands of evidence that suggest that, from a linguistic standpoint, people do not treat MZ cotwins any more or less similarly than DZ cotwins. MZ twins whose parents erroneously thought they were DZ are only slightly less linguistically similar than correctly classified MZ twins, and DZ twins whose parents thought they were MZ twins are only slightly more linguistically similar than correctly classified DZ twins (Munsinger & Douglass, 1976). For twins that are reared apart, MZ-DZ differences cannot be the result of MZ cotwins being treated more similarly than DZ cotwins. 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. Very few studies of twins reared apart versus together exist, and only one study has examined the linguistic abilities of twins (Pedersen, Plomin, & McClearn, 1994). In this study, linguistic heritability estimates were virtually identical to those obtained from similar studies that only include twins reared together (see Stromswold, 2001). Based on these and other results, Martin, Boomsma, and Machin (1997) have argued that the effect of greater similarity of postnatal environment for MZ than DZ cotwins is generally very small for complex behaviors.

5. Teasing apart the role of biological and psychosocial environmental factors on language

5.1. Genetic studies of the impact of environmental factors on language

As reviewed in Section 1, twin studies clearly show that genetic factors play a substantial role in children's language development. Twin studies can also be used to investigate the role of environmental factors on language development (For a discussion of problems associated with using twin studies to estimate the effects of shared environmental factors, see Turkheimer, D'Onofrio, Maes, and Eaves, 2005). Indeed, twin studies have shown that environmental factors play at least as important role as genetic factors in normal twins' language development. For example, the flip side of Stromswold's (2001) meta-analytic results is that environmental factors account for two-thirds of the variance in normal twin' lexical abilities and one-half of the variance in their phonological and syntactic abilities. Similarly, the results of the PEGI twin study indicate that environmental factors account for essentially all of the variance in normal twins' lexical abilities, three-quarters of the variance in their syntactic abilities, and two-third of the variance in their phonological abilities (Stromswold et al., 2005). Twin studies distinguish between environmental factors

that both twins experience and those that only one twin experiences. Shared environmental factors (C) include the linguistic input twins receive (assuming parents speaks similarly to their twins), and non-shared environmental factors (E) include illnesses or accidents that only occur to one twin. Shared environmental factors contribute to twins' similarity and non-shared environmental factors contribute to their dissimilarity.

How we interpret the finding that environmental factors account for most of the variance in twins' linguistic abilities depends on whether these environmental factors are biological or psychosocial. If the environmental factors are biological, this supports nativist theories of language acquisition, whereas if the factors are psychosocial, this supports empiricist theories of language acquisition. Unfortunately, although classic twin studies distinguish between the role of shared and non-shared environment, these studies *conflate* the role of perinatal and postnatal environment. The fact that twin studies conflate perinatal and postnatal environmental factors is critical for theories of language acquisition because, as discussed in Section 3, perinatal environmental factors are predominately biological, whereas postnatal environmental factors are predominantly psychosocial.[12]

Even though classic twin studies conflate the role of perinatal/biological and postnatal/sociolinguistic environment, twin studies can be used to investigate the relative impact of biological and psychosocial factors. One way to do this is to use a measure of perinatal environment (e.g., GA, BW, etc.) as a proxy for biological environment and a measure of postnatal environment (e.g., SES, maternal education, etc.) as a proxy for psychosocial environment. If MZ and DZ twin pairs have equally similar environments and a shared environmental factor affects linguistic development, it should increase the linguistic similarity of MZ and DZ twins equally. Thus, to the extent that a shared environmental factor affects linguistic development, estimates of the impact of genetic factors on linguistic abilities (A) will be lowered and estimates of the impact of shared environment (C) will be raised. So, for example, if twins' linguistic delays are (partially) due to adverse perinatal environment, we would expect low GA twins (i.e., twins at high biological risk) to have higher shared environment estimates (C) and lower genetic estimates (A) than high GA twins (i.e., twins at low biological risk). If twins' linguistic delays are (partially) due to adverse postnatal environment, low SES twins (i.e., twins at high psychosocial risk) should have higher C's and lower A's than high SES twins (i.e., twins at low psychosocial risk). The greater the impact of an environmental factor on language development, the greater the discrepancy in environmental and genetic estimates for language for twins who are at high- versus low- risk for that environmental factor.

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[12] Examples of shared perinatal environmental factors include such things as gestational age at birth (GA), drugs that the twins were exposed to in utero and intrauterine infection, and examples of shared postnatal environmental factors include the quantity and quality of twins' linguistic input and the SES of the family. Examples of non-shared perinatal environmental factors include cases in which twins are discrepant in birth weight (BW), perinatal brain injury, or perinatal drugs received, and examples of non-shared postnatal environmental factors include cases in which only one twin experiences a postnatal illness or injury.

Koeppen-Schomerus, Eley, Wolke, Gringras, and Plomin (2000) conducted analyses of data from the Twins Early Development Study (TEDS) that address the question of how prematurity affects heritability estimates for verbal and non-verbal development. They found that genetic factors played a significant role in the verbal and non-verbal development of moderately preterm and full-term twins, accounting for between 18% and 32% of the variance, but that for very premature twins, the effect of shared environment completely overshadowed the effect of genetics. A study by Turkheimer, Haley, Waldron, D'Onofrio, and Gottesman (2003) suggests that impoverished postnatal environment can also diminish the impact of heritability factors on IQ. In this study, for twins living at or near the poverty level (low SES twins), 58% of the variance in full scale IQ was accounted for by shared environment (C) and 10% of the variance was explained by heritable factors (A). For high SES twins, the proportions were almost exactly reversed (A = .72, C = .15).

5.2. The role of genetics in perinatally and postnatally high-risk twins

5.2.1. Method
Unfortunately, because different groups of twins were studied by Koeppen-Schomerus et al. (2000) and Turkheimer et al. (2003), we cannot determine the relative importance of perinatal (biological) and postnatal (psychosocial) environment. By studying how perinatal and postnatal environment affects heritability estimates for linguistic and non-linguistic development in our initial cohort of PEGI twins (see footnote 2 for a description of these twins), we sought to determine the relative importance of biological and psychosocial environment on children's linguistic and non-linguistic development. For the purposes of these analyses, twins' linguistic and non-linguistic development were assessed using the parent-administered Ages and Stages (AS) communication (language), problem solving (cognitive), gross motor, fine motor, and social-personal (social) tests (Bricker & Squires, 1999). These tests were normed on 10,000 at-risk and normal children, and results have been shown to be valid and reliable. We used Koeppen-Schomerus et al.'s (2000) and Turkheimer et al.'s (2003) procedures to divide twins into high and low perinatal and postnatal risk groups. High perinatal risk twins were born at GAs of 32 weeks or less and low perinatal risk twins were born at GAs of 33 weeks or more. Twins were divided into high and low postnatal risk groups based on SES. SES was calculated by summing the mother's education score (on a 4 point scale), the father's education score (on a 4 point scale) and the family income score (on a 5 point scale). High postnatal risk twins had an SES score of 9 or less and low postnatal risk twins had an SES of 10 or greater.

5.2.2. Results
Genetic factors (A) accounted for only 13% of the variance in low GA twins' language scores (see Fig. 2), but 60% of the variance in high GA twins' language scores (see Fig. 3). Shared environmental factors (C) accounted for 79% of the variance in low GA twins' language scores and only 29% of the variance in high GA twins' language scores. Results were similar for gross motor scores (High GA A = .72,
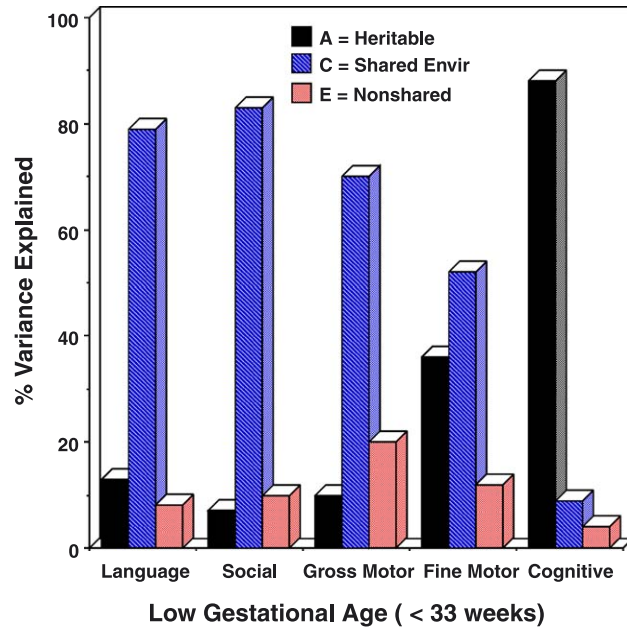
Fig. 2. The role of heritable and environmental factors on the linguistic and non-linguistic development of low gestational age twins.
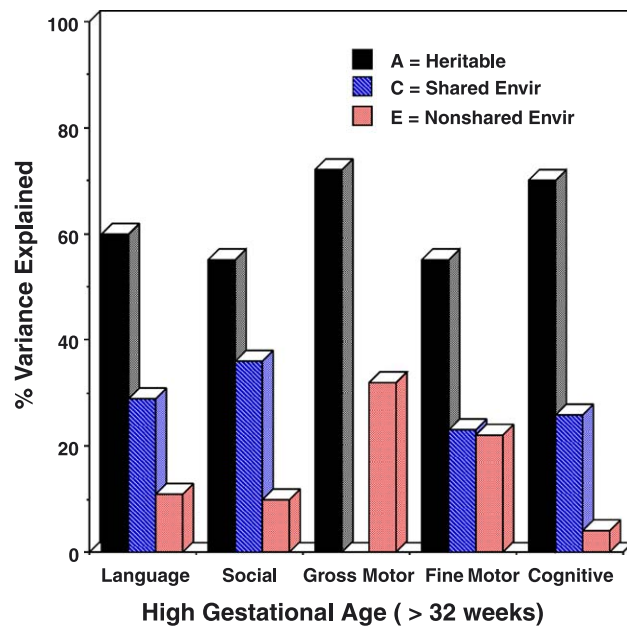

Fig. 3. The role of heritable and environmental factors on the linguistic and non-linguistic development of high gestational age twins.
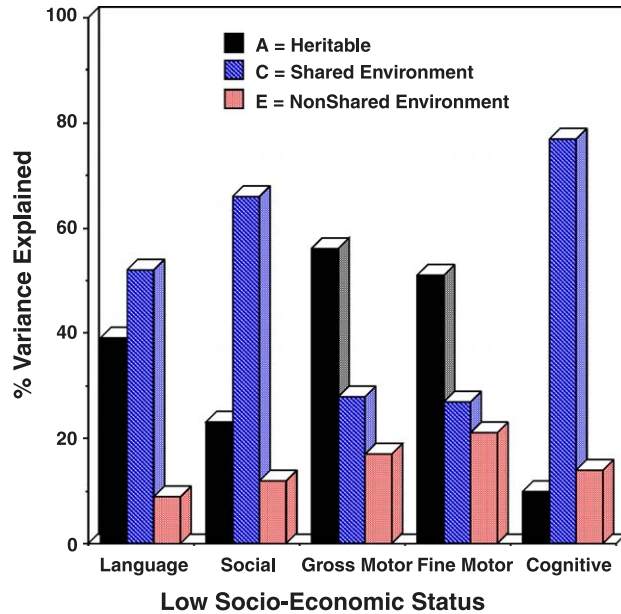
Fig. 4. The role of heritable and environmental factors on the linguistic and non-linguistic development of low SES twins.

C = .00; Low GA A = .10, C = .70) and social scores (High GA A = .55, C = .36; Low GA A = .07, C = .83), but not for cognitive scores where heritable factors affected both GA groups similarly (High GA A = .70 C = .26; Low GA A = .88, C = .09;).[13] In other words, perinatal hardship decreased estimates of the role of genetic factors for language, gross motor and social development but not cognitive development, and perinatal hardship increased estimates of the role of shared environmental factors for language, gross motor and social development but not cognitive development.

Heritability and shared environment estimates of language scores were virtually identical for the low SES (A = .39, C = .52, see Fig. 4) and high SES groups (A = .38, C = .52, see Fig. 5). Results were similar for gross motor scores (High SES A = .74, C = .05; Low SES A = .56, C = .28) and social scores (High SES A = .28, C = .63; Low SES A = .23, C = .66). For cognitive scores, however, shared environment accounted for only 1.5 times more variance than genetic factors for the high SES group (A = .32, C = .49), whereas shared environment accounted for about 8 times more variance than genetic factors for the low SES group (A = .10, C = .77).

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[13] For fine motor development, genetic and shared environment estimates were affected by both GA (High GA A = .55, C = .23; Low GA A = .36, C = .52), and SES (High SES A = .29, C = .53; Low SES A = .51, C = .27).
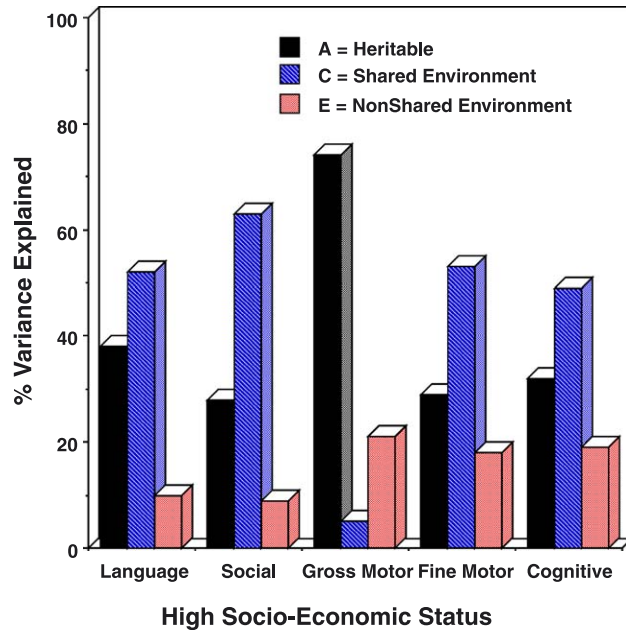
Fig. 5. The role of heritable and environmental factors on the linguistic and non-linguistic development of high SES twins.

5.2.3. Implications

Perinatal hardship decrease estimates of the role of genetic factors for language, gross motor and social development but not cognitive development, and postnatal hardship decrease estimates of the role of genetic factors for cognitive development but not language, gross motor or social development. Perinatal hardship increase estimates of the role of shared environment for language, gross motor and social development but not cognitive development, and postnatal hardship increase estimates of the role of shared environmental factors for cognitive development but not language, gross motor or social development. In short, perinatal environment affects language, gross motor and social development and postnatal environment affects cognitive development. Put another way, when it comes to language development, children's neural status is more important than their psychosocial environment, and when it comes to cognitive development, children's postnatal psychosocial environment is more important than their neural status. These results support nativist theories of language acquisition, and call into question empiricist/emergentist theories. Because the language and cognitive results are so different from one another, our results also provide support for modularist theories of language that argue that language is distinct or dissociable from general cognitive ability.

Gross motor development is generally believed to be largely the result of innate, biological factors, with postnatal environmental factors playing a relatively minor role. Thus, the finding that gross motor development patterns with language

development (i.e., perinatal environment affects both gross motor and language development whereas postnatal environment affects neither) provides further support for nativist/biological theories of language acquisition. One might expect that psychosocial/postnatal factors would play a greater role on social development than biological/perinatal factors. Thus, at first blush, it may seem puzzling that social development patterns with linguistic development. This finding could merely reflect that a few of the social test items have a linguistic component (e.g., ''does your child tell you the names of two or more playmates, not including brothers and sisters?''). This seems unlikely to be the explanation because some of the cognitive test items also have a linguistic component (e.g., ''when you point to the figure [of a snowman] and ask your child, 'What is this?' does your child say a word that means a person?''), and opposite patterns were found for cognitive and language development. Furthermore, the factors that affect social development don't appear to be that different from those that affect language development. For example, autistic spectrum disorders appear to be largely the result of genetic and biological environmental factors, with perinatal environment playing an important role (see Larsson et al., 2005 and references therein). Perinatal environment also appears to play an important role in the social development of children who do not fall in the autistic spectrum. For example, low BW has been linked to poor social function (see Hack et al., 2004 and references therein). In summary, our results provide further support that within the normal range of psychosocial environments, biological factors are most important for language, gross motor and social development. For cognitive development, on the other hand, psychosocial factors play an important role, even within the normal range of psychosocial environments.

6. Interactions among genetic and environmental factors

Environmental factors almost certainly affect twins differently according to their genetic makeup. For example, perinatal hardships that result in a minor brain injury to language areas or a mild sensorineural hearing loss might have devastating effects on the linguistic abilities of a twin genetically at risk for language impairment, yet have no discernible adverse effect on a twin who is not genetically at risk. Similarly, children who are genetically at risk for developing language disorders may be particularly vulnerable to subtly impoverished linguistic environments. Genetic-postnatal environmental interactions do not necessarily have to involve psychosocial environmental factors. Children who are genetically at risk for language impairments may be more susceptible to the adverse effects of malnutrition, environmental toxins, or postnatal head injuries, whereas children who are not genetically at risk may be more resilient to these insults.

There may be synergistic interactions between genetic and environmental factors. For example, because genetically at-risk children probably have relatives who are language impaired, they are likely to be raised in (somewhat) impoverished linguistic environments. Furthermore, children who are linguistically less adept may respond less to linguistic input. Their parents may unconsciously respond by providing less

or less complex linguistic input, which could further slow their children's rate of language acquisition. Less linguistically adept children might also unconsciously choose activities and friends that make less linguistic demands of them, thereby further impeding their linguistic development. At the other end of the scale, if there are synergistic interactions between genetic and postnatal environmental factors, people who have the genetic propensity to succeed at language might benefit more from linguistically rich environments. Because genetically well-endowed children probably have relatives who are also linguistically adept, they are likely to be reared in linguistically rich environments. In addition, parents may respond to children's linguistic precociousness by providing more or richer linguistic input, and linguistically gifted children may seek out environments that are linguistically challenging. As discussed above, twins probably receive less adult linguistic input than singletons. Therefore, if there are synergistic interactions between genetic and postnatal environmental factors, we would predict that twins would be more likely to be language-impaired than singletons with the same genetic makeup. Consistent with this prediction, the rate of positive family history for language impairment is lower for twin SLI probands than for singleton SLI probands (Bishop, 1992).

Environmental factors may be correlated (e.g., prenatal and postnatal malnutrition in poor families; hypoxia and hyperbilirubinemia in preterm neonates) or interact with one other in ways that accentuate the effects of the environment on language development. Consider how perinatal hardships can interact to damage hearing. Not only is the preterm neonate more likely to be hyperbilirubinemic than the full-term child, but because his blood–brain barrier is more permeable and his blood pH and albumin levels are lower, the same amount of bilirubin is more likely to damage his auditory pathways than those of a full-term neonate. Furthermore, hypoxic neonates typically spend time in noisy NICUs and hypoxia potentiates noise-induced hearing loss (Chen, 2002). Perinatal and postnatal environment may also interact synergistically to cause greater linguistic impairments. For example, children who have sustained damage to auditory pathways because of perinatal hardships may be more vulnerable to slightly impoverished linguistic input than other children (Nelson & Soli, 2000). Studies that have followed preterm children longitudinally generally find that the discrepancy in linguistic performance between full-term and preterm children becomes more apparent as children get older (e.g., Botting, Powls, Cooke, & Marlow, 1998; Saigal, Hoult, Streiner, Stoskopf, & Rosenbaum, 2000; Taylor, Klein, Minich, & Hack, 2000b). One interpretation of this finding is that prenatal and postnatal environmental factors interact.

Gene–gene interactions (epistasis) may also occur. For example, a child with *either* a genetic propensity for attention deficit disorder *or* specific language impairment might be phenotypically normal, whereas a child with *both* genetic risk factors might be clinically impaired. Epistasis can also occur at the other end of the scale: a child who is genetically predisposed to be gifted in both language and non-verbal cognitive abilities might exhibit linguistic abilities that exceed those of a child who is only genetically gifted for language.

Genetic–environmental interactions make it difficult to map genotypes to linguistic phenotypes. The probable existence of language-related phenocopy and

pleiotropy make the mapping even more complex. Phenocopy is the term used to describe the situation when different genotypes can result in the same phenotype. The fact that at least 9 distinct loci have been linked to dyslexia and at least 7 loci have been linked to spoken language impairments suggests that different genotypes can cause at least broadly defined phenotypes such as written and spoken language impairments (for a review, see Stromswold, 2005). Even rather specific language impairment phenotypes may have different causes, and hence may be due to different genotypes. Consider a phenotype that is characterized by the selective omission of grammatical morphemes (e.g., the – *ed* in *kicked*, the – *s* in *cats*). This phenotype could be the result of a genetic disorder that selectively impairs syntax, a genetic disorder that specifically impairs control of rapid, complex oral motor movements necessary for language (speech dyspraxia), a genetic disorder that specifically impacts some component of auditory processing (e.g., auditory short term memory, auditory sequencing, rapid auditory processing), or a genetic disorder that affects multiple aspects of language but not non-verbal cognition (see Stromswold, 1997).

Pleiotropy is the term that is used when the same genotype results in different phenotypes. A particularly clear example of pleiotropy is incomplete penetrance, where family members share a mutation for a disorder but only some of these family members are clinically affected. Another type of pleiotropy is when all family members who have a mutation are affected, but the nature of the disorder varies among family members. Consider again a genetic mutation that affects people's abilities to coordinate complex oral motor movements (oral motor apraxia). A person with such a genotype could present as someone who is unwilling or unable to speak in any situation (mutism) or in selective situations (selective mutism), as someone with speech dyspraxia, as someone who has a dysfluency or stutter, or as someone who omits phonologically unstressed elements (i.e., grammatical morphemes) and, hence, appears to have a grammatical deficit. The KE family provides a particularly vivid example of pleotropy: all KE family members with the FOXP2 mutation are affected, but the nature of the disorder varies among family members.

Geneticists must also grapple with the problem that a genotype may be expressed phenotypically in different ways at different points of development. Returning again to the oral motor apraxia mutation, an infant with such a mutation might have difficulty coordinating suck and swallow, and might present as having a feeding disorder or failing to grow adequately. As a toddler, the child might have outgrown his feeding disorder, but be unwilling to speak. By the time he is school-aged, he might speak but selectively omit phonologically unstressed elements. As an adult, his impairment might not be readily apparent, but he might nonetheless avoid linguistically taxing social or professional settings, and hence might seem shy. In a similar fashion, a child who starts out with a fairly language-specific deficit might over time begin to show additional secondary deficits. For example, because he has difficulty understanding what is said to him, he might appear to have attention deficit disorder. Eventually, the child's difficulty understanding spoken language is likely to result in poor school performance, and perhaps even lowered non-verbal IQ.

Recently Becker (2004) has proposed the Common Variant/Multiple Disease (CV/MD) hypothesis to account for pleiotropy and phenocopy in autoimmune

disorders, metabolic disorders (type 2 diabetes and obesity) and schizoid disorders (schizophrenia and bipolar disorders), According to the CV/MD hypothesis, common alleles that contribute to a particular disease under particular genetic and environmental conditions may result in a different disease under other genetic and environmental conditions. For a group of related disorders (e.g., autoimmune disorders such as thyroiditis, systemic lupus erythematosus, and multiple sclerosis), there are some genetic and environmental factors that are unique to a particular disease and other genetic and environmental factors that are shared by several diseases. The CV/MD hypothesis could explain why *most* of the loci that have been linked to written and spoken language disorders have also been linked to other neurodevelopmental disorders, why most cases of familial language disorders do not have simple Mendelian patterns of transmission, why different people with the same genetic mutation have different clinical pictures, and why linkage analyses of people with familial language disorders often fail to identify susceptibility loci, including loci that have been previously identified. The CV/MD hypothesis could also explain why some MZ twins are discordant for language. By adopting the CV/MD framework that developmental language disorders belong to a larger class of neurodevelopmental disorders, we will be in a better position to explore and understand how genetic and environmental factors interacting with one another to affect language.

7. Future directions: Teasing apart the role of genetic and environmental factors

7.1. Molecular genetic studies

One way to study the role of genetic and epigenetic factors on language development is to perform fine-grained molecular analyses to determine whether linguistically discordant MZ twins differ more genetically (e.g., in terms of frequency of mutations) or epigenetically (e.g., in terms of methylation patterns) than linguistically concordant MZ twin pairs. This type of molecular genetic study of MZ twins is beginning to bear fruit in the study of other diseases that are believed to have multi-factorial polygenic origins (e.g., schizophrenia, Petronis et al., 2003). Molecular genetic studies of discordant MZ twins are particularly valuable because by comparing twins with virtually identical DNA sequences, one can detect subtle genetic differences (e.g., micromutations, microdeletions, and microduplications) and epigenetic differences that would be missed in other molecular genetic studies. Thus, it provides a novel way to discover language loci or genes that would otherwise remain unknown. Because there is growing evidence that language is the result of quantitative trail loci, and that most cases of clinical impairments represent the extreme end of the normal spectrum (Stromswold, 2005), we believe it may be equally, if not more, fruitful to compare MZ cotwins whose language outcome measures are the most discrepant, even if the MZ cotwins are not clinically discrepant.

One could also uncover new loci for language through linkage analyses of linguistically similar and dissimilar DZ twins (and their first degree relatives). Although DZ twins are no more genetically similar than full siblings, there are clear advantages of performing linkage analyses on DZ twins as opposed to non-twin siblings. First, the environments of DZ twins are almost certainly more similar than the environments of non-twin siblings. Thus, phenotypic differences between DZ twins are less likely to reflect environmental factors than are phenotypic differences between non-twin siblings. Second, because DZ twins are the same age, one can use the same tests and measures to evaluate their linguistic function, thereby eliminating a huge source of noise in linkage studies. Third, because twins are the same age, one does not have to worry about the possibility that the language-disordered genotype may be expressed phenotypically in different ways at different ages. Eliminating the developmental problem greatly reduces another source of noise and uncertainty in linkage analyses. Despite the clear advantages of performing molecular genetic studies on twins, to date no such studies of spoken language have been performed, although TEDS researchers have begun to collect DNA samples from a subset of their twins (Trouton, Spinath, & Plomin, 2002).

7.2. Genetic load and family history

Perhaps for multifactorial disorders such as language impairments, MZ twins that are concordant for a disorder have a higher genetic load than MZ twins that are not discordant. This possibility can be explored empirically by comparing the family histories of MZ twins where both twins are impaired, one twin is impaired, and where neither twin is impaired. If the genetic load hypothesis is correct, concordant language-impaired MZ twins should have higher rates of family history of language impairment and a higher percentage of impaired relatives than discordant MZ twins who, in turn, should have higher rates than MZ twins where neither twin is impaired. However, as is the case with family aggregation studies, it is possible that what appears to be the result of having a higher genetic load for language impairment is at least partially the result of having a higher environmental load for language impairment. Put another way, it could be that the reason both MZ twins are language impaired is because they have received more impoverished or deviant linguistic input from their language impaired relatives, not because they received more of the wrong genes from these relatives (Stromswold, 1998).

7.3. Broad-sense versus narrow-sense heritability

Geneticists often distinguish between broad-sense heritability and narrow-sense heritability. Broad-sense heritability refers to the variance accounted for by all genetic factors, whereas narrow-sense heritability is the variance accounted for by additive genetic factors. Broad-sense heritability includes the influence of gene dominance, epistasis and interactions between genes and environment, whereas narrow-sense heritability is the amount of genetic influence that is likely to be passed on to offspring. One way of distinguishing between broad- and narrow-sense heritability is

to study the offspring of discordant MZ twins. If an impaired MZ twin's disorder is purely due to environmental factors, then offspring of the non-impaired cotwin should not have an increased risk of being language impaired.[14] If the language-impaired and non-impaired MZ cotwins share a genetic predisposition for being language-impaired, but the non-impaired twin has not experienced the environmental insults that precipitate or contribute to being language disordered, then the non-impaired cotwin will transmit this genetic predisposition to his or her offspring, and these offspring will have higher-than-normal rates of language impairment.

The strategy of looking at the affectedness rate among offspring of impaired and non-impaired MZ twins to contrast broad- and narrow-sense heritability was first used in schizophrenia (Fischer, 1971; Gottesman & Bertelsen, 1989). These studies revealed that schizophrenic MZ cotwins' offspring and well cotwins' offspring were equally at risk for schizophrenia, suggesting that the discordance between MZ cotwins is largely due to differences in the cotwins' environments. Although this type of study *does* provide insights about whether a phenotype is the result of genetic or environmental factors, unfortunately, it does not allow one to distinguish between the effects of prenatal and postnatal environments, nor does it allow one to distinguish between the effects of biological environmental factors (e.g., perinatal brain injuries) and social environmental factors (e.g., amount of linguistic input).

7.4. Birth weight discrepancies and linguistic development

As discussed in Section 5, traditional methods of analyzing behavioral twin data do not distinguish between the effects of prenatal and postnatal environment. Because DZ twins share only 50% of their alleles, birth weight differences in DZ twin pairs reflect differences in the genetic endowment of twin pairs (one twin might be genetically predisposed to be bigger than his cotwin) *and* differences in the prenatal environment. In contrast, because MZ twins share 100% of their alleles, differences in MZ twin pairs' birth weights solely reflect differences in the cotwins' prenatal environments. By comparing MZ cotwins that have very similar birth weights with MZ cotwins that have very dissimilar birth weights, we can obtain an estimate of the effect of intrauterine environment on later development. To the extent that MZ cotwins with very similar birth weights are linguistically more similar to one another than MZ cotwins with very different birth weights, this is a measure of the effect of intrauterine environment on language development. Estimates of the effect of intrauterine environment can be calculated using slight variants of methods traditionally used to calculated heritability estimates. However, instead of contrasting the linguistic similarity of MZ and DZ cotwins, we compare the linguistic similarity of MZ cotwins with similar and dissimilar birth weights. The effect of

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[14] Traditionally, one way of distinguishing genetic from epigenetic or environmental factors is to assume that genetic factors can be directly transmitted to offspring, whereas epigenetic and environmental factors cannot. However, in mice (Waterland & Jirtle, 2003) and men (Kaati, Bygren, & Edvinsson, 2002; Lumey, 1992), diet changes (an environmental factor) can result in phenotypic changes that are heritable. In mice, this is due to changes in DNA methylation, an epigenetic process (Waterland & Jirtle, 2003).

interactions between genetics and intrauterine environmental factors can be estimated by comparing how great an effect having very different birth weights has for MZ and DZ twins (in essence calculating a difference of a difference score).

7.5. Effects of early versus late prenatal environment on language development

Some birth weight discrepant MZ twin pairs remain weight-discrepant postnatally. It is generally argued that MZ twins who catch up to their cotwin in weight were only malnourished in the latter part of the pregnancy (after they had already acquired their full cellular complement), whereas MZ twins who remain smaller than their cotwins were malnourished throughout pregnancy (Bryan, 1993). Thus, one way to investigate how early versus late fetal growth restriction affects linguistic and non-linguistic development is to compare the similarity of MZ twins whose weights remain discrepant compared to MZ twins whose weights equalize over time. Factors that inhibit intrauterine growth early in pregnancy (e.g., viral infection that affects mitosis) cause symmetric growth-retardation, whereas factors that inhibit later intrauterine growth (e.g., placental insufficiency) cause asymmetrical growth retardation. A symmetrically growth-restricted neonate has a normal ponderal index (weight/length^3), but is less than 10th percentile in length, weight, head and abdominal circumferences. In an asymmetrically growth retarded neonate, length and head circumference are relatively preserved, but weight is reduced (Hack, Breslau, Rivers, & Fanaroff, 1989; Hack et al., 1991). Hence, another way to compare the effects of early versus late prenatal growth restriction is to compare the linguistic similarity of height discrepant MZ twins (who had different early prenatal experiences) versus weight discrepant MZ twins (whose prenatal experiences began to deviate later).

Twins' fingerprints may also provide insight into how prenatal environment affects language development.[15] Normally, MZ cotwins have very similar fingerprints (Bouchard, Lykken, McGue, Segal, & Tellegan, 1990; Nylander, 1971), but MZ cotwins who are exposed to markedly different prenatal environments may have very different fingerprints (Babler, 1991a; Bracha et al., 1992; Chakraborty, 1991). For example, intrauterine growth retardation tends to decrease ridge count, whereas fetal edema tends to increase ridge count (Babler, 1991a; Bracha et al., 1992; Chakraborty, 1991). Dermatoglyphic (fingerprint) traits develop between the 12th and 19th gestational weeks and a–b ridges develop between the 15th and 17th gestational week (Babler, 1991a, 1991b), Hence, dermatoglyphic discordance in MZ twins can be used to pinpoint fairly precisely when MZ twins experienced different environments. If dermatoglyphic discordance is associated with linguistic discordance in MZ twin pairs, this would suggest that second trimester prenatal

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[15] At first blush, suggesting that one study fingerprints to investigate anything as complex as language sounds akin to palm-reading and phrenology. It might not be as crazy as it sounds because researchers have had a fair degree of success using dermatoglyphic analyses to investigate the role of prenatal factors in the development of schizophrenia (Bracha, Torrey, Gottesman, Bigelow, & Cunniff, 1992; Davis & Bracha, 1996; Kelly et al., 2004), fetal alcohol syndrome (Wilber, Newell-Morris, & Streissguth, 1993) and sexual orientation (Hall & Kimura, 1994; Hall, 2000).

environmental factors are (partially) responsible for the MZ cotwins' linguistic discordance. One might even argue that the degree of correlation between dermatoglyphic and linguistic similarity in MZ cotwins is a rough measure of the extent to which second trimester *in utero* factors affect language development. In summary, by studying the differences in MZ cotwins' birth weights, ponderal indices, head circumferences and fingerprints, we may gain fresh insights into how intrauterine hardships that occur during different gestational periods affect language development. In addition, such analyses may prove to be a new way of investigating neurolinguistic development and how the developing brain recovers structurally and functionally from insults to brain regions that subserve language.

7.6. Effects of specific perinatal complications

Although several twin studies have investigated the effects of premature birth or low birth weight on later language development, few studies have investigated the effects of specific prenatal complications on later language development. One exception is Bishop's (1997) study of 124 twin pairs. In this study, although language-impaired twin pairs did not differ from non-language impaired twins in terms of birth weight, gestational age at birth, APGAR scores or a composite measure of perinatal hazards, mothers of language-impaired twins were more likely to suffer from toxemia (preeclampsia) than mothers of non-impaired twins. A second exception is a moderate-sized case-control study by Tomblin, Smith, and Zhang (1997), which failed to find evidence that maternal disease or prenatal exposure to occupational chemicals were related to language impairment in offspring.

Clearly, what is needed is large scale studies in which enough information is collected about a large range of perinatal complications so that we can tease apart the effects of specific prenatal, intrapartum and early postnatal complications and the treatments received for these complications. Given the studies reviewed in Section 3, a good place to start would be to look at the effects of perinatal hyperbilirubinemia, GCs, hypoxia (or a proxy such as the need for supplemental oxygen, or assisted ventilation), infection and brain injuries. Mothers of twins are very good at remembering whether they experienced pregnancy and delivery complications (Reich, Todd, Joyner, Neuman, & Heath, 2003). For example, over 95% of the mothers of twins in our Perinatal Risk Factor Study are able to report what prenatal complications they experienced, when the complications arose, and what treatments were received. They are also able to tell us whether each of their twins was breech, how long they were in labor, what drugs they received during labor, how much time passed between the delivery of the first twin and the second twin, whether forceps or vacuum extraction was used for each twin, and whether there were any cord complications. Lastly, they are able report what neonatal complications (e.g., hyperbilirubinemia, brain injuries, infection, etc.) and treatments each twin experienced.[16] Data such as these could be used to test whether linguistically discordant MZ twins

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[16] The two notable exceptions were Apgar scores and chorionicity of twins.

experience more obstetrical complications than linguistically concordant MZ twins, as has been done for schizophrenia-concordant and -discordant MZ twins (McNeil et al., 1994).

7.7. Perinatal brain injuries

The most consistent predictors of poor later mental development and behavior in prematurely born and intrauterine growth restricted children are hypoxic-ischemic brain injuries and subnormal brain growth (Berg, 1989; Harvey, Prince, Burton, Parkinson, & Campbell, 1982; Ounsted, Moar, & Scott, 1988; Parkinson, Wallis, & Harvey, 1981; Westwood, Kramer, Munz, Lovett, & Watters, 1983). Brain growth is initially spared in intrauterine growth restriction, and when this protective mechanism fails, neurodevelopmental outcome is adversely affected (Kramer, McLean, Olivier, Willis, & Usher, 1989). This is especially true when brain growth (head size) fails to catch up during infancy and childhood (Hack et al., 1989; Hack et al., 1991). Neonatologists and pediatricians routinely measure and record infants' head circumferences, and it is trivial to obtain this measurement on older children and adults. One could easily test whether discrepancy in head circumference in MZ twins is associated with linguistic discordance. Because there are well-normed growth curves for head circumference, one could also use MZ twins to investigate whether MZ twins whose head circumferences are persistently discrepant are more likely to be linguistically discordant than twins whose head circumferences become more similar with time.

The twins most at risk for perinatal brain injuries usually spend time in NICUs, where neural ultrasounds are routinely obtained to detect and determine the severity and location of IVHs. Despite the widespread availability of IVH data, we are only aware of one study that has examined cognitive abilities in twins discordant for IVH (Dennis & Barnes, 1994), and this study only included 2 sets of twins. One way of determining whether linguistic discordance in MZ twins is the result of perinatal brain injuries is to conduct a large-scale study to determine whether MZ twin pairs whose neural ultrasounds are more discrepant are more likely to be linguistically discordant. Interactions between genetic and intrauterine factors could be estimated by comparing how great an effect being discrepant in neonatal neurological scores (or head circumference) has for MZ and DZ twins.

7.8. Neuroimaging studies of older twins

Functional and volumetric neuroimaging studies of older twins have begun to elucidate how genetic factors affect the neural bases of language. For example, in a volumetric MRI study of twins, Thompson et al. (2001) found the highest heritability estimates for cortical regions involved in language, with heritable factors accounting for 95–100% of the variance in gray matter density in Broca's and Wernicke's areas. Posthuma et al. (2003, 2002) have performed studies comparing the similarity of twins' test performance and twins' brain structures. In one study, they found that genetic factors completely accounted for the correlation of grey and white matter

volume with full scale IQ and working memory (Posthuma et al., 2002). However, more recently they have shown that, whereas the processing speed subcomponent of full scale IQ was genetically related to white matter volume and the perceptual organization subcomponent was genetically and environmentally related to cerebellar volume, the verbal comprehension subcomponent was not related to grey matter, white matter, or cerebellar volume either genetically or environmentally (Posthuma et al., 2003). This result is surprising because, in their study, verbal comprehension showed the greatest degree of heritability. As they suggest, their failure to find genetic or environmental effects for verbal scores most likely reflects that they used overall brain volumes rather than regional brain volumes as was done in Thompson et al.'s (2001) study.

Imaging studies of MZ twins may be particularly edifying. Results of a recent MRI study that compared the similarity of healthy adult MZ twins' brains suggest that genetic factors are largely responsible for the overall shape of the brain, but both genetic and non-genetic factors affect sulcal and gyral patterns (Mohr, Weisbrod, Schellinger, & Knauth, 2004). It is well established that people who are right-handed are more likely to have language lateralized in the left hemisphere than people who are left handed. It is also well established that 10–25% of MZ twins are discordant for handedness. (For a discussion of how environmental and genetic factors affect handedness, see Medland, Wright, Geffen, Hay, & Levy, 2003.) In a recent fMRI study, MZ twins, who were either concordant for handedness (with both twins being right handed) or discordant for handedness, performed verb generation and semantic decision tasks (Sommer et al., 2002). All of the twins were at least 35 weeks gestation and 2000 g at birth, and all had normal language. There was a significant intra-twin pair correlation for language lateralization scores in the handedness-concordant twins ($r = .74$, $p < .01$), but not in the handedness-discordant twins. Sommer et al. (2002) argue that the significant correlation for the handedness-concordant MZ twins suggests that genetic factors play a role in language lateralization, and that discordant language lateralization and hand dominance results when MZ splitting occurs after the establishment of the embryo's left-right axis.[17]

Neuroimaging studies of MZ twins who are discordant for neurodevelopmental disorders can be used to study the neural bases of these disorders. Examples of how such work might proceed can be seen in recent studies of other neurodevelopmental disorders such as attention deficit hyperactivity disorder (Castellanos et al., 2003), Alzheimer's disease (Jarvenpaa et al., 2004; Jarvenpaa et al., 2003a; Jarvenpaa et al., 2003b; Lipton et al., 2003), schizophrenia (Reveley, Reveley, & Baldy, 1987; Sommer, Ramsey, Mandl, Van Oel, & Kahn, 2004; Spaniel et al., 2003; Suddath, Christison, Torrey, Casanova, & Weinberger, 1990; Weinberger, Berman, Suddath,

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[17] Left-handedness is more prevalent in people with perinatal risk factors such as premature birth, low birth weight, perinatal brain injuries, RH incompatibility, breech delivery and prolonged labor (Medland et al., 2003). It is possible that lateralization findings for handed-discordant MZ reflect intra-twin pair differences in the perinatal environments of twins who are discordant for handedness. Unfortunately, because Sommer et al. (2002) do not report birth weights or gestational ages for handedness-concordant and -discordant MZ twins, we cannot rule out this possibility.

& Torrey, 1992) and epilepsy (Briellmann, Jackson, Torn-Broers, & Berkovic, 2001; Brodtkorb, Myhr, & Gimse, 2000). For example, Briellmann et al. (2001) compared the MRIs of 12 pairs of epilepsy-discordant MZ twins, and found MRI differences in 10 of the 12 cases. In 4 cases, there were postnatal risk factors for epilepsy and evidence of an acquired lesion. There were also 4 cases of a lesion identifiable on MRI, but no postnatal risk factor. Of these, there were 2 cases of unilateral cortical dysgenesis, 1 case of bilateral periventricular heterotopia, and 1 case of focal atrophy. Finally, there were two cases of MZ twins with large differences in brain size, but no MRI lesions. The authors argue that the 6 cases of epilepsy-discordant, MRI-discordant MZ twins without postnatal insults probably represent cases of subtle prenatal hypoxic injuries. In another illustrative study of the possible effects of prenatal insults, Kunugi, Urushibara, Murray, Nanko, and Hirose (2003) describe a case of schizophrenia-discordant MZ twins, in which the schizophrenic twin weighed 30% less than the normal twin at birth. Whereas the unaffected twin had an unremarkable MRI, the schizophrenic twin's MRI revealed high intensity signal in the white matter and enlarged ventricles, consistent with a prenatal hypoxic injury that could have been partially responsible for the twin's schizophrenia.

Neuroimaging studies of MZ twins could help elucidate what the neural substrates of language are, and how environmental factors affect these regions. By comparing which brain regions are and are not concordant for lesions in MZ twins who are discordant for different types of language impairments, we may gain further insights into the neural circuitry involved in different aspects of language. By correlating the types of perinatal (biological) hardships and postnatal (psychosocial) hardships that cotwins experienced (especially those that only one cotwin experienced) with the linguistic abilities of cotwins, we can learn more about the ways in which environmental factors affect neural circuitry involved in linguistic development. Given the perinatal (i.e., neurobiological) risk factors associated with twinning, it would not be at all surprising to find that even for twins who are linguistically concordant, one (or both) have brain lesions. The mere presence of discordant lesions in linguistically discordant MZ twins could be irrelevant from the standpoint of language. In other words, non-discordant MZ twins serve as a control group in brain imaging studies, and it is critical that *both* linguistically concordant and linguistically discordant MZ twins are included in all such studies. Results of imaging studies of linguistically concordant MZ twins will be informative for another reasons: any brain regions that are discordant for lesions in MZ twins who are concordant for linguistic ability are not likely to be critical in language development. (Imaging studies of DZ twins can also be informative. However, the genetic differences between DZ cotwins make it more difficult to interpret the import of differences between DZ cotwins' brains.)

8. Implications of genetic, epigenetic, and perinatal environmental factors

The validity of twin studies of heritability rest on the validity of two assumptions. The first assumption is that MZ cotwins are genetically and epigenetically identical and that DZ cotwins are genetically and epigenetically no more similar

than non-twin siblings. The second assumption is that the environments of MZ cotwins and DZ cotwins are equally similar. In this paper, we present evidence that puts into question both assumptions. MZ cotwins differ genetically and epigenetically in subtle and not-so-subtle ways and DZ cotwins may be genetically and epigenetically more similar to one another than non-twin siblings. To the extent that researchers address the issue of the environmental similarity of MZ and DZ cotwins' environments, researchers generally assume that MZ cotwins' environments are more similar than DZ cotwins' environments and, thus, twin studies overestimate the role of genetic factors on language development. However, perinatal environmental factors that affect MZ twins more than DZ twins may mean that MZ cotwins' environments are *less* similar than DZ cotwins' environments during critical stages of neurodevelopment. We argue, therefore, that twin-based heritability estimates understate the role of genetic factors on language. Indeed, the results of the experiment presented in Section 5 suggest that perinatal factors selectively reduce heritability estimates of language development (and not cognitive development). Given the relationship between GA and language development, even studies that exclude premature twins probably underestimate the role of genetic factors in language development because full-term twins are born an average of 3–4 weeks before full-term singletons. Similarly, given the relationship between BW and language development, even twin studies that exclude low BW twins are likely to underestimate the role of genetic factors because even normal BW twins weigh an average of 400–500 g less than normal BW singletons. In addition to lowering heritability estimates for language, genetic, epigenetic, and perinatal environmental differences between MZ cotwins almost certainly result in some MZ cotwins being linguistically discordant from one another. Our finding that the environmental factors that affect language development are predominantly biological supports nativist theories of language acquisition. In addition, our finding that biological factors affect language development, whereas social factors affect cognitive development, is consistent with modularist theories of language that argue that language is (at least partially) dissociable or distinct from cognition.

In summary, we have learned much about how environmental and genetic factors shape language development by comparing the similarity of MZ twins and DZ twins. We can learn even more by carefully studying what makes members of twin pairs linguistically different from one another. By studying differences in the perinatal environments of linguistically discordant and concordant MZ twins, we can learn how intrauterine environment affects neural development, and how the resulting differences in neural structures can affect language development. We can also use linguistically concordant and discordant MZ twins to explore whether and how postnatal psychosocial and biological insults affect language development. We can simplify the task of identifying loci and genes that affect language by analyzing the DNA of MZ and DZ twins. By applying recently developed techniques and advances from molecular and behavioral genetics, neuroimaging, perinatology, and typical and atypical language development to the study of twins, we can gain new insights into the genetic and environmental forces that enable people to acquire and use language.

Acknowledgements

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