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Genetic and Environmental Influences on Sex-Typed Behavior During the Preschool Years

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The genetic and environmental etiologies of sex-typed behavior were examined during the preschool years in a sample of 3,990 three- to four-year-old twin and nontwin sibling pairs. Results showed moderate genetic and significant shared environmental influence for boys and substantial genetic and moderate shared environmental influence for girls. For both boys and girls, twin-specific shared environmental effects contributed to twins' similarity in gender role behavior and accounted for approximately 22% of the shared environmental variance. These findings extend previous research conducted with older samples by showing not only important genetic contributions to gender role behavior but also an important role for shared environment. The inclusion of nontwin siblings showed that some of the shared environmental influence is specific to twins.

Boys and girls differ, on average, in their preferences for toys, games, and activities from a young age. Boys generally prefer cars and trains to dolls and jewelry, engage in more rough-and-tumble play than they do in caretaking role play, and prefer pretend play that involves adventure and fighting to nurturing and caring (Golombok & Fivush, 1994; Golombok & Hines, 2002; Ruble & Martin, 1998). These sex-typed behaviors are stable across development and increase substantially from early to middle childhood (Golombok & Fivush, 1994; Golombok & Hines, 2002; Ruble & Martin, 1998). In explaining the etiology of such average sex differences, socialization (Bandura, 1977; Mischel, 1966, 1970), cognitive (Martin & Halverson, 1981), and biological hypotheses (Berenbaum & Hines, 1992; Collaer & Hines, 1995) have been proposed. Although a framework involving all three types of hypotheses has seldom been employed in empirical research, there is now consensus that social, cognitive, and biological factors interactively contribute to sex-typed behavior (e.g., Bussey & Bandura, 1999; Ruble & Martin, 1998).

Most research on the etiological basis of sex-typed behavior has focused on average differences between the sexes despite the considerable individual differences that exist within each sex (Golombok & Rust, 1993a; Golombok & Rust, 1993b). A large body of research supports the role of genetic and environmental factors in explaining individual differences in many aspects of biological and behavioral development (Plomin, DeFries, McClearn, & McGuffin, 2001). However, little is known about the genetic contribution to individual differences in sex-typed behavior, especially during the preschool years. The current study examined genetic and environmental sources of individual variation in childhood sex-typed behavior in a large population-based sample of 3- and 4-year-old twin and nontwin sibling pairs.

Individual Differences in Gender Role Behavior

Although large differences in gender role behavior are observed between the sexes, there are substantial individual differences within the sexes in the extent to which boys and girls adopt masculine-typical and feminine-typical behaviors. That is, although some boys are extremely masculine in their preferences for toys, games, and activities, others are less so (Golombok & Rust, 1993a, 1993b; Lippa,

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2002). The causes of individual differences within groups can differ from the causes of average differences between groups. Nonetheless, theories that attempt to explain average differences between the sexes have been assumed to apply as well to individual differences within the sexes.

For socialization theories, variation in parental sex typing could create individual differences within the sexes. Socialization theories suggest that parental attitudes toward gender roles contribute to variability in parental sex typing. For example, parents who hold traditional views should be more likely to foster sex-typed children than should parents who hold egalitarian views (Huston, 1983). Consistent with this, several studies have found that families that espouse gender-egalitarian roles have children with more flexible gender stereotypes and preferences. For example, father's involvement in nontraditional household tasks has been linked to lower indexes of sex role knowledge (e.g., Serbin, Powlishta, & Gulko, 1993; Turner & Gervai, 1995; Weinraub et al., 1984) and sex typing (Baruch & Barnett, 1986) in both preschoolers and elementary school children. Likewise, maternal involvement in nontraditional gender activities has been related to lower levels of same-sex peer preferences and sex-typed occupational (Serbin et al., 1993) and activity preferences (Turner & Gervai, 1995). In contrast, parental traditionality has been associated with early development of gender-related knowledge as well as preferences for sex-typical toys and activities (e.g., Fagot & Leinbach, 1989; Fagot, Leinbach, & O'Boyle, 1992; McHale, Crouter, & Tucker, 1999; Weinraub et al., 1984). Negative findings have been reported (see Ruble & Martin, 1998, for a review) in this area as well, but overall this research suggests that socialization agents may be associated with individual differences both in the acquisition of gender knowledge and in the development of sex-typed preferences.

Individual differences in sex-typed behavior may also arise from within-sex variability in gonadal hormone exposure prenatally (see Collaer & Hines, 1995; Hines, Golombok et al., 2002). For example, studies of intrauterine positioning of female rodents suggest a relationship between variability in exposure to testosterone and within-sex variability in sexually dimorphic behaviors. In a variety of rodent species, female fetuses located near male fetuses in utero, and thus exposed to testicular hormones, display more masculine-typed behaviors than do other females (Clark & Galef, 1998). The diffusion of testosterone across amniotic membranes is thought to constitute a source of within-sex hormonal variability. In human populations, comparing opposite-sex and same-sex female twins might provide a method to examine whether within-sex testosterone fluctuations are associated with individual differences in sex-typed behaviors, although this method has seldom been employed (Miller, 1994).

One study tested this hypothesis by comparing opposite-sex female twins with same-sex female twins on aspects of sensation seeking that are more characteristic of males as compared with females (Resnick, Gottesman, & McGue, 1993). In accordance with animal research, results indicated greater sensation seeking in opposite-sex female twins as compared with same-sex female twins. These findings are compatible with the hypothesis that within-sex variation in hormonal exposure may be associated with sex-typed behavior variability. However, in a twin study of childhood sex-typed play, females with a male cotwin did not engage in more play with masculine-typical toys than did females with a female cotwin, suggesting that normal variation in testosterone levels may not be consequential to individual differences in sex-typed play independent of sharing a postnatal environment with a sibling of the opposite sex (Henderson & Berenbaum, 1997).

Although hormonal and social-cognitive theories propose plausible mechanisms for the emergence of within-sex variability in gender role behaviors, one inherent limitation of the studies reviewed is that social-cognitive and biological mechanisms are difficult to separate. That is, although increased masculine type behavior in opposite-sex female twins could result from hormonal mechanisms, it could also result from sharing a postnatal environment with a male, as opposed to a female, cotwin (Henderson & Berenbaum, 1997; Rust, Golombok, Hines, Johnston, & Golding, 2000). Similarly, because children share their genes as well as their social environment with their parents, the association between parental traditionality and children's sex typing could be explained by either environmental or genetic influences. Moreover, it can be difficult to ascertain the direction of influence in associations of parenting and children's sex typing (Bell, 1968). That is, parents may be responding to rather than creating individual differences in their children's propensities toward sex-typed behavior, and these individual differences may have genetic as well as social origins.

Quantitative Genetic Research on Gender Role Development

Quantitative genetic methods such as the twin design that compares identical (monozygotic [MZ]) and fraternal (dizygotic [DZ]) twins can be used to

investigate genetic and environmental contributions to individual differences in gender role development. These methods are just as useful for testing the net effect of shared environmental influence as for identifying genetic effects. That is, socialization theories that posit that parental attitudes toward gender roles are an important factor in the development of sex-typed behavior would predict that two children growing up in the same family would be similar in their sex-typed behavior regardless of the level of their genetic resemblance.

In addition to disentangling genetic from environmental influences, behavioral genetic methods can distinguish shared and nonshared environmental influences. Shared environmental influences make children growing up in a family similar whereas nonshared environment refers to all other environmental factors (Plomin & Daniels, 1987). It should be emphasized that the word *environment*, whether shared or nonshared, is broadly construed in quantitative genetics to include all nongenetic factors. In other words, environmental influence could be biological (e.g., hormones) as well as social (e.g., parents, peers, and television). That is, because twins share the same womb as well as their home environment, the shared environment can include prenatal hormonal influences, or other aspects of the physical environment, as well as social influences.

Quantitative genetic research on sex-typed behavior has focused almost exclusively on sex-dimorphic personality traits in samples of adolescent and adult twins. Although results from these studies have been inconsistent (Rose, 1988; Rowe, 1982), the majority have challenged the role of shared environmental effects in sex typing and have emphasized the importance of genetic and nonshared environmental influences (Cleveland, Udry, & Chantala, 2001; Horn, Plomin, & Rosenman, 1976; Lippa & Hershberger, 1999; Loehlin, 1985; Loehlin & Martin, 2000; Mitchell, Baker, & Jacklin, 1989). That is, consistent with behavioral genetic research on other aspects of personality, these studies have reported significant genetic (30%–50%) and nonshared environmental influences (50%–70%) but negligible shared environmental effects.

Despite the pervasive evidence that points to the importance of early sex-typed behaviors (Block, 1983; Leaper, 2000; Leaper & Gleason, 1996; Liss, 1983; Sprafkin, Serbin, Denier, & Connor, 1983), only two studies have examined genetic and environmental influences on aspects of gender role behavior typical of early and middle childhood. A study of 158 Australian male twin pairs ages 19 to 40 years examined genetic and environmental influences on sex-dimorphic behavior assessed concurrently in adulthood and retrospectively in childhood. Although shared environmental factors did not contribute to adult sex-typed behaviors, modest genetic (10%) and substantial shared environmental (46%) factors contributed to individual differences in recalled childhood sex-typed behavior (Buhrich, Bailey, & Martin, 1991). These findings lend support to social as well as genetic transmission of childhood sex-typed behavior and suggest that shared environmental factors may be more important in explaining individual differences in sex typing in childhood as compared with adulthood. The small sample size, the wide age range of the participants, the use of retrospective reports of childhood sex-dimorphic behavior, and the lack of information on female twins, however, argue for additional research.

The only other twin study of childhood sex-typed behavior (702 twin pairs, ages 4 to 12 years) compared MZ and DZ twins' correlations on a 16-item parent-rated male–female scale, which focused on gender nonconformity and measured each twins' preference for sex-typical toys, games, and activities (Elizabeth & Green, 1984). For boys, greater similarity between MZ as compared with DZ twins suggested significant genetic (~58%) and shared environmental (~30%) influences. However, for girls, MZ and DZ twins' correlations were similar, suggesting substantial shared environmental influences (~46%) and weak genetic effects (~6%). Within this study, sex typing was also assessed on a 10-point male–female scale, ranging from high femininity to high masculinity. Although results converged with earlier findings for boys (i.e., A= ~54%, C= ~16%), for girls MZ correlations suggested substantial genetic (~60%) but negligible shared environmental effects (0%). The findings from this study provide additional support for social as well as genetic influences on childhood sex-typed behavior. However, the inconsistent pattern of results across measures, the relatively small sample size, the wide age range of the participants, and the lack of formal model-fitting analyses also argue for further research on the genetic and environmental basis of childhood sex-dimorphic behavior.

The main objective of the present study, therefore, was to overcome the methodological limitations of previous research and to examine genetic and environmental influences on gender role behavior in a large sample of preschool male and female twins ages 3 to 4 years—an age group previously neglected—using a standardized parent-rated instrument of sex-typed behavior specifically designed to asses within-sex variation in children of this age (Go-

lombok & Rust, 1993a, 1993b). On the basis of previous research (i.e., Buhrich et al., 1991; Elizabeth & Green, 1984), we hypothesized that both genetic and shared environmental factors would play an important role in the etiology of gender role behavior in the preschool years. Because preschool twins, who are the same age, may be more likely than nontwin siblings to play predominantly with one another and to share the same toys, the current study also for the first time employed data from the twins' younger siblings to examine the extent to which shared environmental effects that are specific to twins influence variation in sex-typed behavior. As this issue has not previously been examined in relation to sex-typed behavior, no more specific a priori hypotheses were formulated.

Method

Participants

The analyses presented in this study are based on data from the Twin Early Development Study (TEDS), an ongoing longitudinal study that sampled from all twins born in England and Wales in 1994, 1995, and 1996 (Trouton, Spinath, & Plomin, 2002). Families of twins were identified by the Office for National Statistics from their children's birth records and were contacted after checking for infant mortality when the children were 1 year old. The project was explained briefly through the mail and parents were asked to indicate whether they would like to learn more about the project. Families that did not reply were sent up to two reminders, following which it was assumed that the families were not interested in receiving further information on TEDS. Around the time the twins were 18 months old, families that expressed an interest in the study ($N = 16{,}810$) were sent a booklet explaining the project in detail and asking for background information about the twins and their families. Booklets were then sent shortly before the twins' second, third, and fourth birthdays as part of subsequent waves of data collection. A parent-rated instrument adapted from Goldsmith (1991) was employed to assign twin zygosity when the twins were 18 months old and again when the twins were 3 and 4 years old. This resulted in unambiguously identifying 95% of the twin pairs as MZ or DZ (Price et al., 2000). The potential 5% inaccuracy in zygosity assignment is unlikely to have an impact on the current results as twins with uncertain zygosity assignment were excluded from our analyses.

In addition to the twin data, families participating in TEDS were also asked whether the twins had any younger brothers and sisters and, if so, whether they would like them to participate in the study as well. Parents who consented were sent a questionnaire to complete for the younger siblings at the same ages as they had also completed a questionnaire for the twins (i.e., 2, 3, and 4 years). This allowed an age-matched comparison between the twins and their younger siblings.

In this study, we report 3- and 4-year-old data from the 1994 and 1995 cohorts. For these cohorts combined ($N = 10{,}932$), 9,442 families returned a completed first-contact booklet, a participation rate of 86.4%. Of these families, 584 were excluded from our sample: 208 pairs whose zygosity was uncertain; 201 pairs in which at least one twin had a specific medical syndrome; and 175 pairs who were extreme outliers for birth weight, time spent in hospital, special care after birth, gestational age, or maternal alcohol consumption during pregnancy. Of the families that returned the first-contact booklet, 64% also returned the 3-year booklet, 54% also returned the 4-year booklet, and 70% returned either the 3- or the 4-year booklet. The families that completed the 3- and 4-year booklets were similar in background (i.e., ethnicity, education, and employment) to families that completed the first contact booklet but did not complete the other booklets, and parents of twins participating in TEDS have been shown to be representative of the general U.K. population with respect to ethnic background, education, and employment status (Iervolino, 2003).

For the analyses presented in this paper, we used data from families of same-sex twins that returned either the 3- or the 4-year booklet ($N = 4{,}447$). We excluded 417 of these families: 276 in which one or both twins had a severe medical condition or information on sex and zygosity was missing, 74 for whom Pre-School Activities Inventory (PSAI, Golombok & Rust, 1993a, 1993b) data were missing for both members of the twin pair, and 67 pairs who were extreme outliers (i.e., their PSAI score was 2.5 *SD* above or below the mean) on the PSAI. (Note that including these outliers did not change the pattern of results.) Of the remaining 4,030 twin pairs, 213 had a same-sex younger sibling who had also returned the 3- or the 4-year booklet. We excluded 17 younger siblings: 6 who did not share the same parents as the twins, 8 for whom age was missing, and 3 for whom PSAI data were missing.

The total number of twin–twin and twin–younger-sibling (T–YS) pairs in the present study, therefore, was 3,990 and included 906 male (MZM) and 1,022 female (MZF) identical twin pairs, 963 male (DZM) and 903 female (DZF) fraternal twin

pairs, and 90 male and 106 female T–YS pairs. For the T–YS pairs, one member of the twin pair with a younger sibling of the same sex was selected at random and paired with his or her brother or sister. To create independent groups, the MZ and DZ groups included only twins who did not have any younger siblings.

Measure

Sex-typed behavior was measured at ages 3 and 4 years using the PSAI (Golombok & Rust, 1993a, 1993b). The PSAI is a psychometrically constructed instrument assessing gender role behavior, which has been standardized on more than 2,000 participants in countries including the United Kingdom, the Netherlands, and the United States. The PSAI consists of 24 items, 12 feminine type items and 12 masculine type items, that fall into three content categories: toys (e.g., guns, jewelry, dolls), activities (e.g., playing house, soldier, dressing up), and characteristics (e.g., enjoys rough-and-tumble play, likes pretty things). Parents complete the inventory to indicate how often their child plays with particular toys and engages in particular activities and games on a scale ranging from 1 (*never*) to 5 (*very often*).

A pseudo *T* score (i.e., a *T* score with $M = 40$ and $SD = 10$ for girls, and $M = 60$ and $SD = 10$ for boys) is computed to make the mean score close to 50 for boys and girls combined and the standard deviation close to 10. A high score on the PSAI represents male-type behavior and a low score represents female-type behavior. Acquiescence—a form of response bias that arises from variation in the extent to which respondents agree (or disagree) with an item in the questionnaire regardless of its content—and response bias effects have been eliminated through a careful balance between masculine-type and feminine-type items (see Golombok & Rust, 1993a, 1993b).

The PSAI has good validity and reliability. Parents' ratings on the PSAI correlate with teachers' ratings ($r = .48$, $p < .001$ for girls, and $r = .37$, $p < .001$ for boys). Results for 1-year test–retest reliability include correlations of .62 for boys and .66 for girls, and results for split-half reliability include correlations of .66 for boys and .80 for girls (Golombok & Rust, 1993a). Scores on the PSAI correlate with prenatal testosterone exposure in normal girls (Hines, Golombok et al., 2002). Also, females with congenital adrenal hyperplasia who are exposed to extremely high levels of testosterone prenatally have elevated (more masculine-type) scores on the PSAI (Hines, Brook, & Conway, 2004; Hines et al., 2003).

Because the PSAI has been constructed to discriminate both within and between the sexes, variation can be assessed among as well as between boys and girls (Golombok & Rust, 1993a, 1993b). In other words, the PSAI can differentiate between "masculine" and "feminine" boys and girls within normal population samples of preschool children. For this reason, the PSAI was well suited for the purpose of this study, which was to examine genetic and environmental influences on individual differences in gender role behavior.

In our sample of preschool twins, reliability for the PSAI was high across sex ($\alpha = .71$ at 3, $\alpha = .65$ at 4) and for girls (αs = .77 at 3 and 4) and boys ($\alpha = .75$ at 3, $\alpha = .74$ at 4) separately. The PSAI showed substantial longitudinal correlations from ages 3 to 4 ($r = .87$, $p < .001$). Although analyses were conducted separately using the 3- and 4-year data, the results were similar at 3 and 4 years, as expected from the correlation of .87 from 3 to 4 years. For this reason and to facilitate presentation of the results, we report data for the 3- and 4-year aggregate. For this aggregate, alpha reliabilities were high and ranged from .76 to .91.

Both animal (Beach, 1975) and human (Bem, 1977) research suggest that the factors influencing masculinity and femininity can differ, making it useful to study these dimensions separately. Therefore, in addition to conducting twin correlations and model-fitting analyses on the PSAI, quantitative genetic analyses were also conducted separately on the masculine-type and the feminine-type items. Analyzing these items separately yielded the same pattern of results except that the variance attributable to shared environmental influences was greater, a finding that likely reflects acquiescence effects (for a more in-depth discussion, see Iervolino, 2003). For this reason, and in the interest of space, these analyses are not presented but may be requested from the first author.

Analyses

Intraclass correlations. The differing levels of genetic resemblance between MZ and DZ twins were employed to disentangle genetic and environmental influences on sex-typed behavior (Plomin et al., 2001). Because MZ twins share all of their genetic makeup, and DZ twins are only about 50% similar genetically, MZ twins are expected to be more similar than DZ twins if genetic influence is important. Shared environmental influence is seen as the extent to which genetic factors do not account for twin resemblance. If shared environment is primarily

responsible for variation in sex-typed behavior, twin correlations will be large and similar for both MZ and DZ twins. Nonshared environmental influence (and error of measurement) is implicated to the degree that genetic and shared environmental sources of variance cannot account for all of the observed variation.

As well as disentangling genetic from environmental influences on gender role behavior, data from the twins' younger siblings were used to separate shared environmental influences that are common to all children growing up in the same family from shared environmental influences that are specific to twins. As twins and their younger siblings share half of their genetic heritage, as do DZ twins, but differ in age and do not share the same womb at the same time as do other siblings, comparing the degree of resemblance of DZ twins with that of a twin and their younger sibling provides an estimate of twin-specific shared environmental effects. Thus, twin-specific shared environmental effects on sex-typed behavior are implied to the extent that DZ twins' correlations are greater than T–YS correlations. Conversely, twin-specific shared environmental effects are not implied if DZ twins' correlations are comparable to those of twins and their younger siblings.

Model-fitting genetic analyses. Maximum-likelihood model-fitting analysis offers a more sophisticated method to estimate the contribution of additive genetic (A), shared (C), twin-specific shared (T) and nonshared environmental (E) factors by partitioning the variance of a phenotype into these four components simultaneously and providing confidence intervals for these parameter estimates. Model-fitting analysis also provides estimates of fit between the assumed model and the observed data, and thus allows hypothesis testing with alternative models (Neale & Cardon, 1992). Univariate maximum-likelihood model-fitting analyses of raw data were conducted to test the ACTE model shown in Figure 1 using the structural equation modeling package MX (Neale, 1997), which has been designed especially for analyzing genetically sensitive data.

As can be seen in Figure 1, the ACTE model assumes that the variance of a particular phenotype is due to the four latent factors: A (additive genetic variance), C (shared environmental variance common to all children), T (shared environmental variance specific to twins), and E (nonshared environmental variance plus measurement error; Koeppen-Schomerus, Spinath, & Plomin, 2003). The within-pair phenotypic correlation for A is fixed according to the genetic relatedness of the sibling pairs: 1.0 for MZ twins and 0.5 for DZ twins and T–YS pairs. The within-pair phenotypic correlation for C is set to 1.0 for all sibling types because, by definition, such shared environmental factors contribute to sibling similarity for all children growing up together in the same family. The within-pair phenotypic correlation for T is set to 1.0 for MZ and DZ twins and to 0 for T–YS pairs because, by definition, T contributes to the covariance of twin–twin siblings but not to that of nontwin siblings. The E parameter is uncorrelated within pairs because, by definition, nonshared environmental factors account for differences between siblings beyond those resulting from genetic differences between siblings. In T–YS pairs, T is subsumed by nonshared environmental variance.

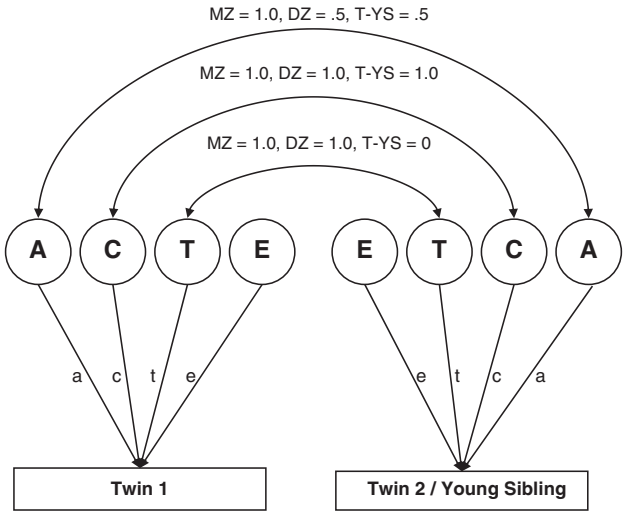

Figure 1. The ACTE model. The ACTE model decomposes the variance of the Pre-School Activities Inventory (PSAI) into additive genetic (A), shared environment (C), nonshared environment (E), and twin-specific environment (T). The within-pair phenotypic correlation for A is fixed to 1.0 for monozygotic (MZ) twins and 0.5 for dyzogotic (DZ) twins and twin–young sibling (T–YS) pairs. The within-pair phenotypic correlation for C is set to 1.0 for all sibling types, although the within-pair phenotypic correlation for T is set to 1.0 for MZ and DZ twins and to 0 for T–YS pairs. The E parameter is uncorrelated within pairs. In T–YS pairs, T is subsumed by nonshared environmental variance.

As is standard procedure with analysis of raw data, the data were first fit to a saturated model (see Neal & Cardon, 1992, for further details), and goodness of fit, in terms of a log-likelihood chi-square statistic (a small and nonsignificant chi-square indicates a good fit) and Akaike information criterion (AIC; a large and negative AIC value indicates a good fit), was obtained by comparing the -2 log-likelihood statistic (-2Ll) of the more parsimonious genetic model (i.e., the ACTE model) with that of the saturated model.

To establish the best fit for the data, alternative models were tested by systematically dropping

paths from the full model. That is, the data were fit to nested submodels in which the phenotypic variance is accounted for by A, C, and E, or by C, T, and E, as compared with the full model that includes A, C, T, and E. The nested and full models were compared using the likelihood ratio test (Neale & Cardon, 1992), and the relative improvement or worsening of the fit was assessed on the basis of a change in chi-square relative to the change in degrees of freedom (e.g., $\chi^2_{\mathrm{ACTE}} - \chi^2_{\mathrm{ACE}}$, $df_{\text{full model}} - df_{\text{submodel}}$).

Quantitative sex differences in parameter estimates were also explored. The hypothesis that genetic and environmental factors contribute equally to sex-typed behavior in boys and girls was tested by comparing the fit of the ACTE model, which allows sex differences, with more parsimonious submodels that constrain A, C, T, or E to be equal for boys and girls. These models were compared using the likelihood ratio test. A good fit for the more parsimonious submodels indicates that the relative contribution of A, C, T, and E to sex-typed behavior does not differ for males and females. On the other hand, an improvement in fit from the more parsimonious submodels to the ACTE model indicates that there are significant differences in parameter estimates for males and females.

Results

Descriptive Statistics

Means and standard deviations for the PSAI are presented in Table 1 for male and female MZ and DZ twins and for their younger siblings. For MZ and DZ twins, PSAI scores ranged from 37 to 91 for males and from 9 to 60 for females, indicating substantial variability within as well as across sex and considerable overlap between boys and girls. For the twins' younger siblings, PSAI scores were similar and ranged from 46 to 83 for males and from 10 to 51 for females. PSAI means and standard deviations are comparable to those reported by Golombok and Rust (1993a, 1993b) in a large sample of boys and girls ages 3 to 5 years and to those reported by Hines, Johnston et al. (2002) in a population sample of more than 9,000 three-and-a-half-year-olds.

To assess the effects of sex and zygosity, a 2 × 2 between-subjects analysis of variance (ANOVA) was conducted independently for twins (using data from one randomly selected twin from each pair) and their younger siblings. As indicated by their greater PSAI mean, male twins engaged in more masculine-typical behavior and in less feminine-typical behavior than female twins, $F(1, 3793) = 10232.37$, $p < .001$, $\eta_p = .73$, sex accounting for 73% of the variance. Twins' younger brothers also engaged in more masculine-typical behavior and in less feminine-typical behavior than twins' younger sisters, $F(1, 195) = 878.01$, $p < .001$, $\eta_p = .82$, sex accounting for 82% of the variance. There were no significant main effects of zygosity; PSAI means were similar for MZ and DZ twins, $F(1, 3793) = 1.62$, *ns*, and for MZ and DZ twins' younger siblings, $F(1, 195) = .99$, *ns*, and no significant Sex × Zygosity interactions were found. As Table 1 shows, PSAI means were greater for the twins' younger brothers and lower for the twins' younger sisters than they were for male and female twins, respectively, indicating more sex-typed behavior for younger siblings than for twins: Sex × Sibling Status interaction $F(1, 3945) = 26.97$, $p < .001$, $\eta_p = .07$. This finding is consistent with previous research indicating greater sex-typed behavior in children with older siblings of the same sex, as compared with singleton children and children with older siblings of the other sex (Rust et al., 2000).

Table 1
Pre-School Activities Inventory: Means and Standard Deviations for Male and Female Monozygotic (MZ) and Dizygotic (DZ) Twins and for the Twins' Younger Siblings

	Boys			Girls		
Sibling type	*N*	*M*	*SD*	*N*	*M*	*SD*
Twins	1869	64.14	8.71	1925	35.92	8.46
MZ	906	64.09	8.30	1022	36.30	8.40
DZ	963	64.19	9.08	903	35.50	8.52
Twins' younger siblings	90	65.72	8.14	106	31.20	8.11

Twin Intraclass Correlations

Twin intraclass correlations were calculated to provide a rough index of genetic and environmental contributions to sex typing in both boys and girls and are presented in Table 2. As Table 2 shows, MZ correlations are consistently greater than DZ correlations, indicating genetic influence. However, greater heritability is suggested for girls than for boys, and greater shared environmental influence is suggested for boys than for girls. Although the twin correlations suggest genetic influence, they indicate even stronger influence of shared environmental factors, as seen in the generally high level of correlations for both MZ and DZ twins.

Thus, intraclass correlations were also calculated for T–YS pairs to explore the extent to which shared

Table 2
Pre-School Activities Inventory: Intraclass Correlations and Confidence Intervals (CI) for Male and Female Monozygotic (MZ) and Dizygotic (DZ) Twins and for Twins and Their Younger Siblings (T – YS)

	Boys		Girls	
Sibling type	*N*	*r*(CI)	*N*	*r*(CI)
MZ	898	.83** (.81 – .85)	1,020	.78**(.75 – .80)
DZ	956	.69** (.66 – .72)	893	.50** (.45 – .55)
T – YS	90	.41** (.23 – .57)	106	.33** (.10 – .51)

**$p < .01$.

environmental factors that are specific to twins contribute to twin similarity on the PSAI. As Table 2 shows, T – YS correlations are consistently lower than DZ correlations, suggesting greater similarity for twins than for nontwin siblings, and thus a twin-specific shared environmental effect on PSAI scores. This pattern of results is confirmed by Fisher's *Z* comparisons (see Cohen, Cohen, West, & Aiken, 2003), which indicate that T – YS correlations are significantly lower than DZ twin correlations for both boys ($z = 3.77$, $p < .01$) and girls ($z = 2.20$, $p < .05$).

Model-Fitting Analyses

Model-fitting genetic analyses of raw data were conducted to confirm correlational analyses and are shown in Tables 3 and 4. Comparisons of the ACTE model with the saturated model, presented in Table 3, show that the ACTE model fit the data well, as indicated by a small and nonsignificant chi-square and a negative AIC value.

Exploring the significance of twin-specific shared environmental effects. Twin-specific shared environmental effects on the PSAI were examined by comparing the fit of the ACTE model with the fit of a more parsimonious submodel in which the twin-specific shared environmental parameter (T) is fixed to zero (shown in Table 4). Consistent with correlational analyses, dropping T from the model (Models 2 – 3) provided a significantly worse fit to the data for both boys and girls, indicating that twin-specific shared environmental effects contribute to twin similarity on the PSAI.

Table 3
Pre-School Activities Inventory: Model-Fitting Results for the Saturated and the Free Genetic Model

	Model fit		Change in fit			
Model	– 2Ll	*df*	χ^2	*df*	*p*	AIC
Saturated model	17,313.37	7964				
Free genetic model	17,325.71	7974	12.34	10	.26	– 7.66

Note. – 2Ll = Minus twice log likelihood; AIC = Akaike information criterion.

Exploring quantitative sex differences in genetic, shared, nonshared, and twin-specific shared environmental parameter estimates. Comparing the fit of the ACTE model with more parsimonious submodels in which A, C, T, or E are constrained to be the same for boys and girls (Models 4 – 7) provided a test for quantitative sex differences. As Table 4 shows, twin-specific shared environmental effects were similar in magnitude for boys and girls; that is, constraining T to be the same across the sexes did not worsen the fit of the model. The contribution of genetic, shared, and nonshared environmental factors to the PSAI, however, differed for boys and girls. Thus, requiring A, C, or E to be the same across the sexes provided a significantly poorer fit to the data, indicating quantitative sex differences in the etiology of sex-typed behavior.

Exploring the significance of genetic and environmental parameter estimates. Models 8 to 11, presented in Table 4, examined the significance of additive genetic and shared environmental influence on the PSAI for boys and girls independently. As Table 4 shows, dropping A and fitting the data to a model that assumes the variance of the PSAI to be due solely to shared, twin-specific shared and nonshared environmental factors provided a significantly worse fit to the data for both boys and girls, indicating that genetic influences are important in explaining individual variation in sex-typed behavior. Although, for girls, the data could be fit to a model that assumes no shared environmental influence on the PSAI without any loss in fit; for boys, dropping C yielded a significantly worse fit to the data. This suggests that whereas shared environmental factors that are common to all children growing up together in the same family are important in explaining variation in sex-typed behavior in boys, they are not important for girls.

Parameter estimates for the full ACTE model, and their 95% confidence intervals, are summarized in Table 5 for both boys and girls. Consistent with correlational analyses, shared environmental effects that are specific to twins contributed to twins' similarity on sex-typed behavior and accounted for approximately 20% of the PSAI variance. Although twin-specific environmental effects were similar for boys and girls, additive genetic influences were greater for girls (57%) than they were for boys (34%), and shared environmental influences were greater for boys (29%) than they were for girls (0%). Nonshared environmental influences were also greater

Table 4
Pre-School Activities Inventory: Model-Fitting Results Examining Quantitative Sex Differences and Twin-Specific Environmental Effects

Model		Model fit		Change in fit				
		χ^2	*df*	Compare to:	χ^2	*df*	*p*	AIC
1	Free genetic model	12.34	10	-	-	-	-	−7.66
	Testing significance of specific twin-environment effects							
2	Drop T boys	24.82	11	1	12.48	1	.01	2.82
3	Drop T girls	22.57	11	1	10.32	1	.01	.057
	Testing significance of quantitative sex differences							
4	Equate A	27.34	11	1	15.00	1	.01	5.34
5	Equate C	17.80	11	1	5.46	1	.05	−4.19
6	Equate T	12.34	11	1	0.00	1	.34	−9.66
7	Equate E	32.11	12	6	19.77	1	.01	8.11
	Testing significance of specific parameter estimates							
8	Drop A boys	142.17	12	6	129.83	1	.01	118.17
9	Drop A girls	189.31	12	6	176.97	1	.01	165.31
10	Drop C boys	44.10	12	6	31.76	1	.01	20.10
11	Drop C girls[†]	12.34	13	6	0.00	1	.42	−11.66

Note. AIC = Akaike information criterion; A = additive genetic; C = shared environment; E = nonshared environment; T = twin specific environment.
[†]Best fitting model.

for girls (21%) than they were for boys (15%), although the difference was small.

Discussion

The present study extended behavioral genetic research on sex-typed behavior in three important ways. First, it investigated genetic and environmental influences on gender role behavior in an age group (i.e., preschoolers) previously neglected. Second, it avoided some of the methodological shortcomings of previous studies, such as small sample sizes, retrospective reports, wide age range, or focus on only one sex. Third, by including data on nontwin siblings, it examined, for the first time, the extent to which shared environmental factors that are specific to twins influence variation in sex-typed behavior. Consistent with previous research, both genetic and environmental factors were found to be important in explaining individual differences in gender role behavior. However, contrary to research conducted with older samples (Cleveland et al., 2001; Horn et al., 1976; Lippa & Hershberger, 1999; Loehlin, 1985; Loehlin & Martin, 2000; Mitchell et al., 1989), shared environmental factors were found to contribute significantly to within-sex variability in gender role behavior. Furthermore, in comparisons with nontwin siblings, we found that much of this shared environmental influence for boys and all of it for girls is specific to twins.

Limitations

The findings of this study should be interpreted in view of several limitations, some of which apply to twin studies in general (Hoffman, 1991; Maccoby, 2000; Rutter, 2002; Rutter, Pickles, Murray, & Eaves, 2001), and others that are specific to the current

Table 5
Pre-School Activities Inventory: Parameter Estimates and Confidence Intervals (CI) for Additive Genetic (A), Shared (C), Nonshared (E), and Twin-Specific Environment (T) Effects for Boys and Girls

Boys				Girls			
A	C	T	E	A	C	T	E
.34 (.28 – .41)	.29 (.09 – .42)	.22 (.09 – .41)	.15 (.13 – .17)	.57 (.47 – .65)	0[a] (0 – .001)	.22 (.09 – .30)	.21 (.19 – .23)

[a]Parameter estimate could be dropped without any loss in fit.

study. First, a core assumption of the twin method is the generalizability of findings from twin to nontwin populations. By including data on the twin's younger siblings, we were able to address this issue and to tease apart shared environmental factors unique to twins from those common to all siblings. However, because of our study design, which examined data for the twin's younger siblings, it was impossible to disentangle the effects of birth order from those of sibling status. Future studies employing data on the twin's older as well as younger siblings may be able to resolve this issue. A second limitation of this study is that data on parental sex typing were not collected; thus, it was not possible to measure the extent to which assortative mating (i.e., the nonrandom mating of spouses) inflated shared environmental variance (see Eaves, D'Onofrio, & Russell, 1999). However, as shared environmental factors were negligible for girls in our study, it is unlikely that assortative mating affected our estimates. A final limitation of this study, like other twin studies, is that it could not pinpoint the exact nature of the environmental and genetic factors that contribute to variability in gender role behavior. One important direction for future research is to identify these underlying genetic and environmental factors and to specify the processes by which they come to influence individual differences in gender role behavior (see Bronfenbrenner & Ceci, 1994). Once these proximal processes begin to be identified, a next step would be to examine how environmental factors mitigate or exaggerate genetic influences through potential Gene $\times$ Environment interaction effects (see Scarr & McCartney, 1983).

Genetic and Environmental Influences in the Etiology of Gender Role Behavior

The findings from this study are consistent with those reported by Buhrich et al. (1991) and Elizabeth and Green (1984) involving children but are inconsistent with those reported by most studies of older twin samples (e.g., Loehlin & Martin, 2000), which found negligible shared environmental influences on gender role behavior. Differences in age, rater, and types of measures offer three potential explanations for this discrepancy in findings.

Concerning age, behavioral genetic research has shown that the influence of shared environment for some traits, especially cognitive abilities, decreases with age from infancy and early childhood to adolescence and adulthood (McGue, Bouchard, Iacono, & Lykken, 1993; Plomin et al., 2001). Because of this, differences in age across the samples (i.e., preschool children vs. adolescents and young adults) could have contributed to finding substantial shared environmental influence in younger, but not older, samples. That is, shared environmental influence may be more important in explaining variation in sex-typed behavior during the preschool years, as compared with adolescence and early adulthood when children distance themselves from the relatively controlled environment of the family home and experience a more varied environmental context. It is also possible that shared environmental influences become less important following puberty, when dramatic biological and social changes take place. In accordance with this age explanation, prior twin studies of childhood sex-typed behavior (Buhrich et al., 1991; Elizabeth & Green, 1984) have reported significant shared environmental estimates, comparable in magnitude to those reported in the current study. Moreover, in a study of male adult twins, shared environmental effects were more important in explaining childhood sex-dimorphic behavior than in explaining adult sex-dimorphic behavior (Buhrich et al., 1991).

A second possible explanation for the discrepancy in findings may be that rater bias inflated shared environmental estimates because the same parent rated sex-typed behavior for both twins and their younger siblings, whereas the other two twin studies used self-report questionnaires. However, the balancing of positively scored (masculine type) and negatively scored (feminine type) items within the PSAI counterbalances and thus eliminates response bias from acquiescence, the response style that is of most concern where participants share the same rater and a particular difficulty where the rater is the mother of twins. Furthermore, rater bias could not account for the greater shared environmental estimates in boys (29%), than in girls (0%); we would expect to observe the same pattern of shared environmental influence across sex if these effects were an artifact of rater bias alone. That is, to explain the results, a rater-bias explanation of the shared environment results would have to explain why the bias exists for boys more than girls. One such possible explanation may be that parents are more reluctant to report sex-atypical behaviors in sons than they are in daughters. However, the significant shared environmental effects observed for retrospective self-reports of childhood sex-typed behavior in a study of male adult twins (Buhrich et al., 1991) also suggest that rater bias effects alone are unlikely to account for the significant shared environmental estimates observed in our study or in other studies of childhood sex-typed behavior.

A third possible explanation involves differences in the types of measures used to assess gender role behavior. Gender researchers now consider sex typing as a multidimensional construct, encompassing diverse domains of behavior including personality, attitudes, activity preferences, and mannerisms, and acknowledge that these domains may have different etiological basis (e.g., Huston, 1983; Ruble & Martin, 1998). The majority of studies of adolescent and adult twins have limited their measurement of sex-typed behavior to personality characteristics, whereas the PSAI assesses a range of sex-typed behaviors and characteristics empirically demonstrated to differentiate between as well as within the sexes in the age group under investigation (Golombok & Rust, 1993a, 1993b). Other research has shown that although parents encourage sex-typed interests and activities in their children, they encourage personal attributes such as independence, aggression, and achievement equally in boys and girls (e.g., Lytton & Romney, 1991; Maccoby & Jacklin, 1974). Thus, one possible explanation for the divergent findings is that shared environmental influences may be more pronounced in nonpersonality aspects of sex-typed behavior. Consistent with this explanation, Lippa and Hershberger (1999) found significant shared environmental influences on teenagers' self-reported degree of participation in sex-typed activities (32%) but no shared environmental influence for sex-typed personality characteristics.

Twin-Specific Shared Environmental Effects

As well as estimating genetic and environmental influences on gender role behavior, the present study examined twin-specific shared environmental influences on gender role behavior and found that up to 22% of the shared environmental variance on the PSAI could be attributed to shared environmental factors specific to twins. Further research is needed to determine the exact nature of these effects. However, because twins share the same uterus at the same time, unlike nontwin siblings, these effects could reflect hormonal influences. Fetal hormonal levels are influenced by genetic factors (e.g., Harris, Vernon, & Boomsma, 1998; Sluyter et al., 2000) but are also affected by maternal hormones and placental enzymes (Tulchinsky & Ryan, 1980) and hormones and chemicals to which the fetus is exposed through the mother during a specific pregnancy (e.g., alcohol, drugs, and other environmental exposures; McGivern, Handa, & Redei, 1993). Maternal testosterone levels during pregnancy have been related to gender role behaviors as measured by the PSAI (Hines, Golombok et al., 2002). Thus, the shared hormonal environment experienced by twins prenatally could contribute to the twin-specific environmental effects for gender role behavior.

Another viable explanation for these twin-specific shared environmental effects is that parental ratings on the PSAI reflect something about the twin pair rather than the individual twins. That is, preschool play may be an area in which twins' experiences may differ from those of other children because at these ages twins may play predominantly with each other with shared or duplicate toys, especially if the twins are of the same sex. Preschool children with a same-sex sibling tend to be more sex-typed in their behavior than singleton children or children with opposite-sex siblings (Bigner, 1972; Brim, 1958; Koch, 1956; Rust et al., 2000; Stoneman, Brody, & MacKinnon, 1986). These sibling influences may be amplified in the case of twins who are exactly the same age and therefore age differences may contribute to the greater similarity observed for twins than for nontwin siblings. Alternatively, it may be that parents make fewer distinctions when reporting on toy play, games, and activities among twins than among nontwin siblings. Additional research is needed to evaluate these possibilities.

Although the significant twin-specific shared environmental effects found in this study for gender role behavior may be interpreted as demonstrating the limited generalizability of twin studies, it should be noted that twin-specific shared environmental effects have been found to be important for some behaviors but not for others (Koeppen-Schomerus et al., 2003). In addition, as noted earlier, the twin-specific shared environmental influence does not necessarily reflect only greater similarity in the postnatal social environment of twins versus nontwins but could also reflect intrauterine similarity, particularly in the prenatal hormone environment. Hormones could also provide a proximal mechanism through which genetic influences exert their effects because there are genetic influences on the amount of testosterone produced by an individual, particularly in females, and because prenatal levels of testosterone, both in the normal and abnormal range, are known to relate to postnatal gender role behavior as assessed using the PSAI (Hines et al., 2003; Hines, Golombok et al., 2002).

Sex Differences in the Contribution of Genetic, Shared, and Nonshared Environmental Influences

In addition to estimating genetic and environmental influences independently for boys and girls,

the size of our sample allowed us to examine sex differences in the contribution of genetic and environmental influences to gender role behavior. Although twin-specific shared environmental effects were similar for boys and girls, shared environmental factors were more important for boys than for girls, whereas genetic factors were more important for girls than for boys. Shared environmental effects may be greater for boys than for girls because boys are more influenced by the social environment. Parents and peers provide stronger feedback and are more likely to criticize boys for sex-inappropriate behaviors than they are girls (e.g., Fagot, 1977, 1978; Fagot & Hagan, 1991; Huston, 1983; Langlois & Downs, 1980; Maccoby, 1998; Ruble & Martin, 1998). Boys also are more likely than girls to believe that other people think of cross-gender typed play as ''bad'' and to be influenced by this belief (Banerjee & Lintern, 2000; Raag, 1999; Raag & Rackliff, 1998). Thus, in addition to experiencing stronger socialization influences, social norms of sex appropriateness may be stricter for boys than for girls, and boys may be more likely to go along with these norms.

Although this study's findings of greater shared environment for boys than girls are consistent with results from studies of older twin samples (Cleveland et al., 2001; Lippa & Hershberger, 1999; Loehlin & Martin, 2000; Rose, 1988), they are not entirely consistent with those reported by Elizabeth and Green (1984). The most likely explanation for the inconsistency involves the different types of measures used. Elizabeth and Green used two parent-rated instruments of sex-typed behavior: a single-item 10-point male–female scale, ranging from high femininity to high masculinity, and a 16-item male–female scale, focusing on gender nonconformity (e.g., expressed wish to be of the other sex). In line with the current study's findings, when sex typing was measured on the single-item 10-point male–female scale, Elizabeth and Green found that shared environmental influences were greater for boys than for girls and that genetic influences were greater for girls than for boys. However, in contrast to our findings, when sex typing was measured on the 16-item questionnaire, they found that shared environmental influences were greater for girls than for boys and that genetic influences were greater for boys than for girls. Thus, genetic influences may be more important in explaining gender identity or gender nonconformity in boys than in girls, whereas this is not true for sex-typical behaviors such as toy and activity preferences.

In summary, our results extend previous research, confirming that both genetic and shared environmental factors contribute to sex-typical behavior. Because we avoided some of the methodological problems of prior studies in this area, our results provide stronger evidence that both types of factors are important, at least for preschool-age children. In addition, our results make several novel contributions to understanding genetic and environmental influences on gender-typical behavior. First, they suggest that the extent to which these factors influence gender role behavior differs for boys and girls; in particular, the shared environment is more important for boys than for girls. Second, our results suggest that twin-specific shared environmental effects make a unique contribution to gender role behavior, perhaps reflecting intrauterine factors such as levels of testosterone, as well as postnatal socialization effects. Finally, consideration of our results in view of prior twin studies suggests that: (a) shared environmental influences on gender role behavior may decrease with age, and (b) genetic and environmental influences may differ for different sex-typed characteristics, with the shared environment having a larger influence on gender-related interests and activities than on gender-related personality traits.

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