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# From Specialist to Generalist: Developmental Transformations in the Genetic Structure of Early Child Abilities

***ABSTRACT:*** *The heritability of abilities increases substantially over development, and much of heritable variation in abilities is shared with other abilities. No study, however, has formally tested the extent to which developmental increases in heritability occur on shared versus unique variation in child abilities. A transactional perspective predicts that the relative proportion of shared to total genetic variance will increase with age, whereas an endogenous perspective predicts that such proportion will be invariant with age. We tested these competing predictions using data from a sample of 292 twins providing a total of 578 cross-sectional and longitudinal observations between ages 0 and 6 years on measures of Communication, Gross Motor, Fine Motor, Problem-Solving, and Personal-Social abilities. Consistent with predictions of the transactional perspective, developmental increases in heritability were localized to variance shared across abilities.* © *2015 Wiley Periodicals, Inc.* Dev Psychobiol.

***Keywords:*** *pleiotropy; generalist genes; genetic correlation; genetic commonality; genetic structure; increasing heritability; mutualism; dynamical systems; early child development*

## INTRODUCTION

In the statistical sense, a strong general factor, *g*, underlies many disparate domains of cognitive functioning at all stages of human development from infancy through old age (Carroll, 2003; Gignac, 2014; Gottfredson, 2002; Jensen, 1998; Spearman, 1914; Tucker-Drob, 2009). The generalist genes perspective holds that this general factor occurs primarily because most genes contributing to one domain of cognitive functioning also contribute to other domains of cognitive functioning (Kovas & Plomin, 2006; Plomin, Kovas, & Haworth, 2007). Consistent with this perspective, genetic correlations between many diverse abilities are moderate to strong in magnitude (Alarcón, Plomin, Fulker, Corley, & DeFries, 1999; Butcher, Kennedy, & Plomin, 2006; Chow, Ho, Wong, Waye, & Bishop, 2013; Luo, Petrill, & Thompson, 1994; Petrill et al., 1998; Petrill, 2002; Petrill, 2005; Plomin & Spinath, 2002; Rice, Carey, Fulker, & DeFries, 1989), a phenomenon referred to as statistical pleiotropy. Moreover, one recent study reported moderate genetic correlations among the brain structures underlying different abilities (Schmitt et al., 2007).

An outstanding question is whether statistical pleiotropy is a developmentally invariant property of the human biological system, or the product of dynamic processes that emerge and strengthen over development. In contrast to the well-documented age-related increase in heritability of cognitive abilities (Briley & Tucker-Drob, 2013; Haworth et al., 2010), age-related changes in statistical pleiotropy are not well-studied. Two different perspectives have been proposed in the literature to explain the existence of statistical pleiotropy, one of which would predict developmental

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increases in statistical pleiotropy (i.e., a *transactional* perspective; disproportionately more growth in generalist compared to specialist genetic influences), and the other of which would predict relatively stable associations among genetic influences on different abilities over development (i.e., an *endogenous* perspective; proportional increases in generalist and specialist genetic influences). Using data from a sample of young twins, the current study distinguishes between these competing predictions by investigating age moderation of the multivariate genetic structure of early child abilities.

## Mechanisms of Increasing Heritability and Genetic Commonality

The heritability of cognitive abilities increases across development (Bartels, Rietveld, van Baal, & Boomsma, 2002; Boomsma et al., 2002; Briley & Tucker-Drob, 2013; Davis, Haworth, & Plomin, 2009; Haworth et al., 2010; Tucker-Drob, Briley, & Harden, 2013). Analyzing cross-sectional data on 11,000 pairs of twins from four different countries, Haworth and colleagues observed that heritability of general cognitive ability increased from 41% in childhood to 66% in young adulthood. Briley and Tucker-Drob meta-analyzed 16 genetically informative longitudinal studies, totaling 11,500 sibling pairs of ages 6 months to 18 years, and confirmed that the heritability of cognitive abilities increases over development. Importantly, the heritability of a particular ability represents the combined effects of both general genetic factors, which also contribute to variation in other abilities, and specific genetic factors, which contribute uniquely to variation in that specific ability. Changes in the heritability of a given ability over development may result from changes in general genetic factors, specific genetic factors, or some combinations of the two. Here, we further describe two general classes of mechanisms that lead to different predictions about the pattern in which generalist and specialist genetic influences on abilities change with age.

***Transactional Perspective.*** One way that statistical pleiotropy may emerge is through the multiplier effects of different abilities on one another. In their mutualism model, van der Maas et al. (2006) proposed that causation between biologically independent abilities may contribute to their intercorrelations. Under the mutualism model, reciprocal causation between different abilities leads to the emergence and strengthening of shared genetic variance over time. van der Maas and colleagues suggest that genetic correlations across different abilities may be weak or negligibly small very early in development; as development progresses, reciprocal processes result in increasing statistical pleiotropy.

Similarly, Dickens (2007) proposed that the dynamic association between abilities and environments can result in the emergence and strengthening of statistical pleiotropy. If an individual has a particular advantage (or disadvantage) in a specific ability, this might prompt exposures to environments that broadly facilitate (or impede) the development of other abilities. For example, a child who has high verbal ability may be identified by parents and teachers as "smart" and consequently tracked into more challenging coursework both in reading and in math. The Dickens model predicts that the early genetically influenced individual differences in a specific ability result in evocation and active selection of environmental experiences relevant for the development of multiple abilities. As ability-environment dynamics accumulate, genetic correlations among different abilities are expected to strengthen.

***Endogenous Perspective.*** What might be termed an *endogenous* perspective holds that pleiotropic genetic variation results from individual genes that play multiple roles in biological and psychological functions. This can occur, for instance, when a gene codes for multiple proteins each of which serves as a physiological basis for a different ability, or when a gene codes for a single protein that is important for multiple physiological functions, each of which supports a different ability (see Kovas & Plomin, 2006 and Plomin & Spinath, 2002). Importantly, this endogenous perspective holds that statistical pleiotropy is an inherent property of the human biological systems that subserve cognition and behavior. The associations between different domains of functioning are therefore predicted to remain more or less the same across ages. In other words, all else being equal, no developmental changes in genetic correlations among different abilities are expected. Although the magnitude of overall genetic influence may grow with age, the extent to which genetic factors are generalist compared to specialist is not expected to change. This perspective resembles that of Juan-Espinosa et al. (2002), who wrote "basic structure does not change at all, although, like the human bones, the cognitive abilities grow up and decline at different periods of life" (p. 406). Gignac (2014) further speculated that perhaps "the reason the strength of the *g* factor is largely invariant across age is because it is mediated substantially by biological characteristics" (p. 96). Based on this endogenous perspective, genetic commonality is expected to remain relatively constant across development.

## Developmental Increase in Generalist Genes and Total Heritability

Importantly, total heritability of a phenotype is independent of its genetic commonality with other phenotypes. Two abilities that are highly heritable could share no genetic variance with one another (or they could share all genetic variance with one another). Moreover, two abilities that are only modestly heritable could share all of their genetic variance with one another (or they could share none of it). It is possible, however, that developmental changes in genetic commonality emerge simultaneously with developmental increase in heritability. In other words, increasing heritability could occur largely via increases in generalist genetic variance. The top panel of Figure 1 illustrates this scenario. Each circle represents the total heritability of a given phenotype at a given developmental period. Not only does the circle grows in size across development, indicating increasing total heritability, but the proportion representing common genetic influences also grows across development indicating increasing genetic commonality. Thus, in this scenario, both total heritability and genetic structure change across development.

Alternatively, increasing heritability could occur via proportional increases in both common and unique genetic variance. The bottom panel of Figure 1 illustrates this scenario. As in the top panel, the size of each circle represents the total heritability of a given phenotype at a given developmental period. While the size of circle increases across development, indicating increasing total heritability, the area representing common genetic influences takes up a similar proportion of the circle across development. Thus, in this scenario, total heritability increases but genetic overlap across phenotypes (or the genetic structure of each phenotype) remains comparable across development.

## Previous Evidence for Developmental Transformations in Genetic Commonality

Dynamic transactions between phenotypes, genes, and environments begin early in development (e.g., Tucker-Drob et al., 2013). During infancy and early childhood, average levels of abilities—and their longitudinal stability—dramatically increase (Tucker-Drob & Briley, 2014). Researchers, however, have rarely examined the structure of genetic and environmental influences on

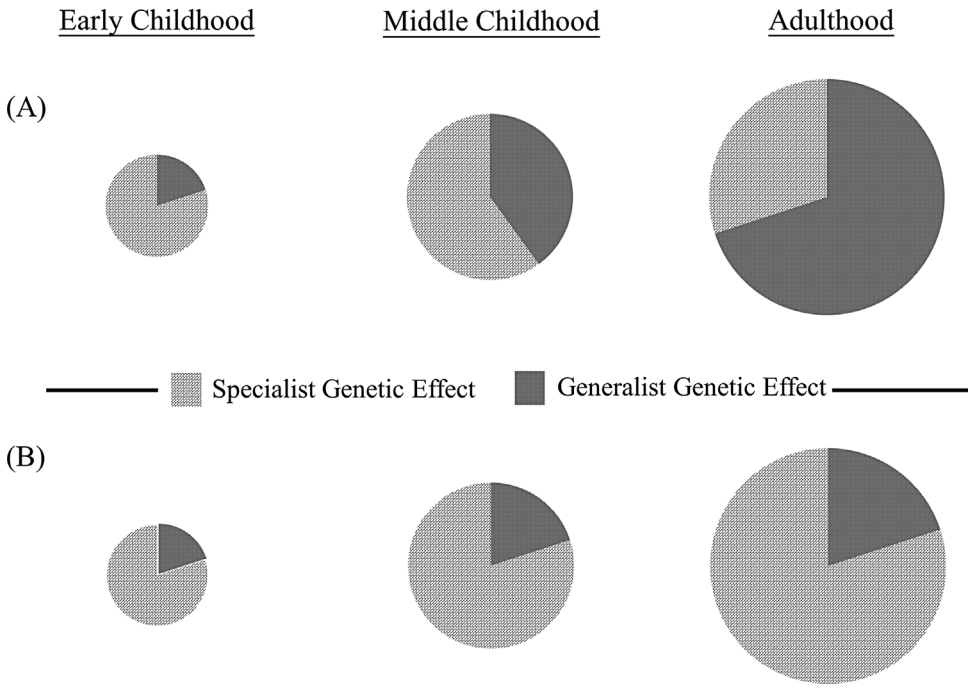


**FIGURE 1** Two hypothetical scenarios for developmental changes in domain-general (generalist) and domain-specific (specialist) genetic effects on domains of functioning. The size of each circle represents total heritability. Top panel: The structure of genetic effects changes with age, with an increasing proportion of genetic effects occurring at the domain-general level. Bottom panel: The structure of genetic effects is age-invariant, with constant proportions of domain-general and domain-specific genetic effects across development.

early abilities among children of very young ages. One possible reason is that conventional measures of early infant abilities tend to be unidimensional (e.g., Cherny et al., 1994). Exceptions include Petrill, Saudino, Wilkerson, and Plomin (2001) who, in a sample of 2-year-olds, observed a heritability of .26 and a nonshared environmental influence of .06 for the *g* factor (molarity) and heritabilities of .03–.32 and a nonshared environmental influence of .26–.56 unique to each of the subordinate tasks (modularity). The authors speculated that this finding of both common and specific genetic effects "suggest a developmental trend from modularity to molarity when considered in relation to multivariate genetic results later in life that show that genetic effects on cognitive abilities contribute primarily to molarity rather than modularity" (p. 31).

Based on an earlier analysis of the same data, which found that the correlation between genetic factors of verbal and nonverbal abilities was a modest .30 at age 2 years, Price, Eley, et al. (2000) speculated that "genetic effects on cognitive abilities are modular early in development and then become increasingly molar" (p. 948). Indeed, in a more recent paper (Trzaskowski, Shakeshaft, & Plomin, 2013) that made use of ages 7 years and 12 years data from later longitudinal assessments of what appears to have been the same sample, biometric twin models revealed genetic correlations between approximately .60 and .70. Similarly, in a sample of children aged 4 years from the Colorado Adoption Project, Rice et al. (1989) observed moderately high positive genetic correlations between verbal, spatial, perceptual speed, and visual memory abilities that range from .56 to .89. Piecing together, these snapshots of different age groups, statistical pleiotropy might emerge and strengthen during the first few years of life, as would be predicted by a transactional perspective.

## Current Study

The current study used multivariate data on early child abilities from an age-heterogeneous sample of young twins (ages 0–6 years) to test for transformations in the genetic and environmental structure of abilities with age. We applied models that capitalize on the known differences in genetic relationships between monozygotic twins (who share 100% of their genes) and dizygotic twins (who, on average, share 50% of their segregating genes), combined with the knowledge that both members of each twin pair (regardless of zygosity) have been reared together in the same home, to partition variation in both domain-general and domain-specific ability factors into additive genetic (*A*), shared environmental (*C*), and nonshared environmental (*E*) components. We then tested the extent to which each of these variance components differs with age.

# METHODS

### Participants

Data were collected as a downward extension of the Texas Twin Project (Harden, Tucker-Drob, & Tackett, 2013) to families with twins or multiples aged 0–6 years who live in the state of Texas. Qualifying families were identified both from birth records provided by the Texas Department of State Health Services and from community outreach. Community outreach efforts included attending annual conventions of Texas Mothers of Multiples, sending recruitment information to associated email list serves, and enrolling families who registered via the Texas Twin Project website. Most participating families completed surveys managed and stored on Research Electronic Data Capture (REDCap; Harris et al., 2009). Depending on a family's preference, an online or paper survey was sent immediately after the family enrolled in the study. Paper and online administration of the measurement we employed (the Ages and Stages Questionnaire, see below) have been found to have equivalent psychometric properties (Squires, Twombly, Bricker, & Potter, 2009). After a family completed the survey for the initial wave, follow-up surveys were sent every 2 months for children from birth until 2 years old, every 3 months for children between 2 and 3 years old, every 5 months for children between 3 and 5 years old, and one last survey for children between 5 and 6 years old. Data collection remains on-going.

For the current study, data were available from 296 individual twins and multiples. Among this sample, a pair of twins was diagnosed with Fragile X syndrome while another pair of twins showed substantially more advanced gross motor development but substantially delayed overall development when compared to the rest of the sample. Results were similar across analyses with and without data from these two twin pairs included. Here, we reported findings from analyses excluding these observations (i.e., based on a sample of 292 individual twins). The sample was 75.34% Caucasian, 4.11% Latino or Hispanic, 2.05% African-American, and 13.70% multi-racial. Less than 1% of these twin families reported having completed only high school, 7.53% reported having some college education, 36.99% reported having completed college, and 54.79% reported having completed education beyond college.

Zygosity for same-sex twins was determined from physical similarity ratings (e.g., hair structure, eye color, and shape of ear lobe, etc.). Primary caregiver of each twin pair rated four items on a 3-point Likert scale ranging from *Not Alike* to *Exactly Alike* and eight other items on a dichotomous scale. Zygosity assignment using physical similarity ratings is highly reliable and corresponds strongly with assignments based on DNA genotyping (Forget-Dubois et al., 2003; Heath et al., 2003; Price, Freeman et al., 2000; Rietveld et al., 2000). Following Harden, Kretsch, Tackett, and Tucker-Drob (2014), we conducted a two-class Latent Class Analysis

(LCA) on all 12 items to determine each same-sex twin pair's zygosity (opposite-sex twins are necessarily dizygotic). This resulted in the sample of 60 monozygotic twins (30 male and 30 female individual twins), 132 same-sex dizygotic twins (58 male and 74 female individual twins), and 100 opposite-sex dizygotic twins (50 male and 50 female individual twins). Sensitivity analyses indicated that models that excluded data from opposite-sex twins produced parameter estimates that were very similar to those in which data from opposite-sex twins were included. We, therefore, reported results from analyses of data from both same-sex and opposite-sex twins, in order to maximize our sample size, and, hence, the precision of our estimates.

In addition to data provided at the initial wave, most families in this sample provided data at one or more follow-up waves. Thus, data were available for up to nine different waves per family. To maximize the pool of observations available for our age-comparative analyses, we used all available data from both baseline and follow-up waves in conjunction with the Complex Survey option in *Mplus* statistical software (Muthén & Muthén, 2010) to account for the nonindependence of longitudinal repeated measurements from the same families across different survey waves. That is, we treated observations on the same twin from different waves as different lines of data and corrected the standard errors of model estimates for biases that could have otherwise potentially resulted from nonindependence of data obtained on the same individuals over time. This resulted in a total of 578 observations on 292 individual children—122 observations from monozygotic twins and 456 observations from dizygotic twins. The average age at measurement among these 578 observations was 2.45 years old ($SD_{age} = 1.24$ years).

### Measures

***Ages and Stages Questionnaire, Third Edition (ASQ).*** The ASQ (Squires & Bricker, 2009) is a multidimensional measure of the occurrence of developmental milestones related to various domains of cognitive and psychomotor functioning. It was standardized on a sample of 12,695 individuals representative of the U.S. young children population on various dimensions, including sex, ethnicity, and various socioeconomic indices (Squires et al., 2009). The ASQ has been shown to accurately reflect young children's progress in attaining developmental milestones in different domains (i.e., high levels of sensitivity and specificity—86% on average—across ages 2–60 months) and agree 86% on average with standardized developmental assessment based on observational tasks (Squires et al., 2009). A number of additional independent studies have also reported high levels of convergent validity of the ASQ with standardized researcher/clinician-administered measures, such as the Bayley Scales of Infant Development (Bayley; Gollenberg, Lynch, Jackson, McGuinness, & Msall, 2010; Schonhaut, Armijo, Schönstedt, Alvarez, & Cordero, 2013; Simard, Luu, & Gosselin, 2012; Yu et al., 2007). In comparison to many questionnaires that query the raters' intuitive judgments on the development of a child relative to other children of the same age, the ASQ minimizes rater bias by querying about children's performance on concrete tasks. Primary caregivers rated each of their twins' performance on these concrete tasks on a 3-point Likert scale for five domains: *Communication*, *Gross Motor*, *Fine Motor*, *Problem-Solving*, and *Personal-Social*. Table 1 defines these domains and gives sample items. These five domains encompass the neurocognitive, psychosocial, and motor milestones used routinely in clinical settings as indicators of young children's physical, psychological, and neurological development (Council on Children With Disabilities, Section on Developmental Behavioral Pediatrics, Bright Futures Steering Committee, & Medical Home Initiatives for Children with Special Needs Project Advisory Committee, 2006). Delays in reaching these developmental milestones may suggest early functional impairment, and may have cascading effects on later psychological development and real-world functioning across the lifespan (Murray, Jones, Kuh, & Richards, 2007; Sørensen et al., 2010; Taanila, Murray, Jokelainen, Isohanni, & Rantakallio, 2007; van Os, Jones, Lewis, Wadsworth, & Murray, 1997).

All items in ASQ are age-appropriate, meaning that twins at different ages are rated on different sets of items (Squires et al., 2009). Each domain contains 5–10 items depending on the age of the twins. Items from adjacent age-ranges (both above and below) at each age were administered in order to avoid floor and ceiling effects, and to allow the use of vertical scaling to capture children's age-related growth in each domain of development. For each domain of development, a minimum of three items were set to overlap in content for adjacent item sets. Domain scores were obtained from Rasch Item Response Theory (1PL IRT) analyses with higher scores indicating more advanced development. IRT-estimated item reliabilities (item communalities) for *Communication*, *Gross Motor*, *Fine Motor*, *Problem-Solving*, and *Personal-Social* were .92, .89, .87, .85, and .82, respectively. Scaling these item-reliabilities using the Spearman–Brown prophecy formula for a 5-item composite measures (the minimum number of items administered in a given domain for a given age) yields scale reliabilities of .98, .98, .97, .97, and .96 for the five ASQ domains, respectively.

## RESULTS

Descriptive statistics of domain scores obtained from the 1PL IRT analyses and the correlations between these domain scores and age are listed in Table 2. Regression analysis was conducted to account for the linear and quadratic influences of age on each domain of early child abilities (see Fig. 2 for age trends of all five ASQ domains). Resulting residuals were *z*-standardized for all analyses reported below. All results presented below are based on these age-corrected standardized residuals. Correlations between domain scores, corrected for both the linear and quadratic influences of age, are also listed in Table 2. Consistent

**Table 1. Definition and Sample Items for Each ASQ Domain**

| Domain | Definition | Sample Items |
|---|---|---|
| Communication | Effective expression of thoughts and processing of information or instructions | *Does your child correctly use at least two words like "me," "I," "mine," and "you?"*—for children aged 19–28.49 months<br>*Without giving your child help by pointing or using gestures, ask him/her to "put the book on the table" and "put the shoe under the chair." Does your child carry out both of these directions correctly?*—for children aged 25.5–44.99 months |
| Gross Motor | Motor development that involves large muscle groups and whole body movement | *Without holding onto anything for support, does your child kick a ball by swinging his/her leg forward?*—for children aged 21–38.99 months<br>*Does your child climb the rungs of a ladder of a playground slide or slide down without help?*—for children aged 39–50.99 months |
| Fine Motor | Coordination of small muscle movements that occur in body parts such as fingers | *When you put a toy in his/her hand, does your baby hold it in his/her hand briefly?*—for children aged 1–2.99 months<br>*Does your child unbutton one or more buttons?*—for children aged 39–56.99 months |
| Problem-Solving | Ability to use generic rules or logic and find solutions to problems | *Does your child finish the following sentences using a word that means the opposite of the word that is italicized? For example: "A rock is hard, and a pillow is soft.*"—for children aged 57–71.99 months<br>*When (shown three circles of different sizes and) asked, "which circle is the smallest?" does your child point to the smallest circle?*—for children aged 39–71.99 months |
| Personal-Social | Self-care ability and basic skills that prepare them for successful social interactions | *Does your child wash his/her hands using soap and water and dry off with a towel without help?*—for children aged 39–56.99 months<br>*Does your baby smile at you?*—for children aged 1–2.99 months |

with past literature, we observed a positive manifold of correlations among the five ability domains. As reported below, structural equation models were fit using *Mplus* statistical software (Muthén & Muthén, 2010) to investigate age differences in the multivariate structure of early child abilities at both phenotypic and behavioral genetic levels.

## Phenotypic Models

We began by examining whether the phenotypic structure of the five domains of early child abilities varies across ages. We specified a multivariate model in which the loadings of each ability on both the common factor and the ability-specific unique factor was mod-

**Table 2. Descriptive Statistics and Correlations Between Domains of Early Child Abilities**

| | | | Age- and Age$^2$-partialled Correlations | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | Cross-Domain Correlations | | | | Intra-Class Correlations | |
| | *M* (*SD*) | Correlation With Age | Communication | Gross Motor | Fine Motor | Problem-Solving | MZ | DZ |
| Communication | 17.31 (5.97) | **.91** | – | | | | **.91** | **.69** |
| Gross Motor | 15.07 (5.12) | **.89** | **.33** | – | | | **.83** | **.46** |
| Fine Motor | 13.81 (4.66) | **.90** | **.43** | **.43** | – | | **.77** | **.63** |
| Problem-Solving | 12.96 (4.29) | **.89** | **.43** | **.36** | **.41** | – | **.86** | **.60** |
| Personal-Social | 10.92 (3.88) | **.93** | **.42** | **.40** | **.46** | **.41** | **.90** | **.70** |

*Note*: Bolded = $p < .01$.

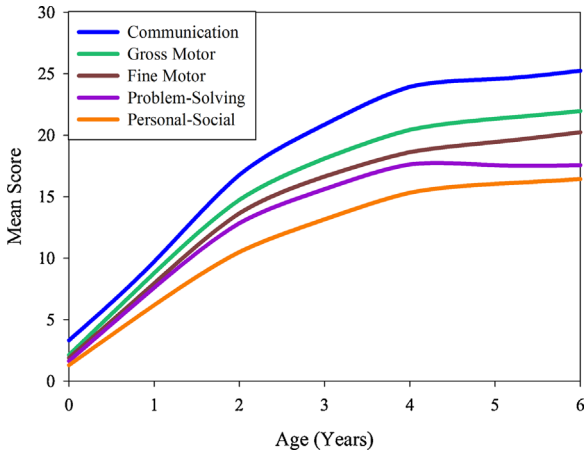


**FIGURE 2** Age trends of the five domains of early child abilities. Mean score for a given domain at a given age is the average of raw domain scores for that age group.

erated by age. Following Tucker-Drob (2009), this model is written as

$$G[x]_n = v[x] + \alpha[x] \times age_n + (\lambda_1[x] + \lambda_1'[x] \times age_n) \times g_n + (\lambda_2[x] + \lambda_2'[x] \times age_n) \times u[x]_n$$

In the above equation, $[x]$ indicates the domain of early child abilities to which a term corresponds. For example, $G[x]$ represents the score on a given domain of early child abilities (i.e., $G[Communication]$, $G[Gross\ Motor]$, $G[Fine\ Motor]$, $G[Problem\text{-}Solving]$, and $G[Personal\text{-}Social]$). Each score is determined by a combination of factors: $\upsilon[x]$ represents the regression intercept for a given domain of early child abilities, $\alpha[x]$ represents the main effect of age on a given domain of early child abilities (freely estimated, but expected to be 0, given that each ability was residualized for age prior to analyses), $g$ represents the common latent factor *Broad Ability*, $\lambda_1[x]$ represents the main effect of *Broad Ability* on a given domain of early child abilities, $\lambda_1'[x]$ represents the interaction between age and *Broad Ability* on a given domain of early child abilities, $\lambda_2[x]$ represents the main effect of the ability-specific factor on a given domain of early child abilities, $\lambda_2'[x]$ represents the interaction between age and the ability-specific factor on a given domain of early child abilities, and $u[x]$ represents the ability-specific (residual) factor in a given domain of early child abilities. The subscript $n$ signifies terms that are allowed to vary by individuals. The latent variables $g$ and $u[x]$ were scaled to a $z$-metric ($M = 0$, $SD = 1$).

As delineated in the above equation, in this phenotypic model, the effects of *Broad Ability* and ability-specific (residual) factors are each modeled as the sum of (1) its main effect on the corresponding domain (i.e., $\lambda_1[x]$ and $\lambda_2[x]$), and (2) its interaction with age (i.e., $\lambda_1'[x]$ and $\lambda_2'[x]$). If *Broad Ability* factor loading of any domain varies as a function of age (i.e., if $\lambda_1'[x]$ is statistically significant), this implies age differences in the general concept of early child abilities; any age differences in factor loadings observed from this phenotypic model would inform the subsequently conducted behavioral genetic analyses. Alternatively, if *Broad Ability* factor loadings do not vary as a function of age in the phenotypic model (i.e., $\lambda_1'[x]$ is statistically nonsignificant), this suggests measurement invariance and factor loading of each domain on the latent factor *Broad Ability* would then be specified to be invariant across ages in our behavioral genetic models. Increasing unique variance as a function of age (i.e., $\lambda_2'[x]$ is greater than zero at a statistically significant level) implies age-differentiation of early child abilities. That is, depending on the sign of the interaction coefficient $\lambda_2'[x]$, domains of early child abilities may become more (or less) distinct from each other as children age.

To elucidate age influences on the phenotypic structure of early child abilities, we fit four versions of the phenotypic multivariate model and compared their model fit statistics to identify the best-fitting model. We first fit a model in which all the interaction coefficients (i.e., $\lambda_1'[x]$ and $\lambda_2'[x]$) were freely estimated. Second, to increase the model's statistical power in detecting age differences, we constrained the interaction coefficient to be proportional to the corresponding main effect in each regression path (i.e., $\lambda_1'[x] = \lambda_1[x] \times \lambda_1'$ and $\lambda_2'[x] = \lambda_2[x] \times \lambda_2'$, where $\lambda_1'$ and $\lambda_2'$ are invariant across domains). Third, we constrained the interaction coefficients to be the same across domains of early child abilities at both the factor and residual levels (i.e., $\lambda_1'[x] = \lambda_1'$ and $\lambda_2'[x] = \lambda_2'$, where $\lambda_1'$ and $\lambda_2'$ are invariant across domains). Finally, we fixed all the interaction coefficients at zero (i.e., $\lambda_1'[x] = \lambda_2'[x] = 0$).

Table 3 lists the model fit statistics of all the phenotypic models. The model with no interactions fit the data no worse than the more complex models, and this model was therefore preferred. Table 4 lists the parameter estimates from all the phenotypic models. Results from the preferred model (Model 4) indicate that *Broad Ability* accounts for 34–48% (e.g., for *Gross Motor*, $.59^2$ / $[.59^2 + .83^2] \times 100\%$) of the variance in each ability at all ages, with the remaining variance being unique to that ability.

## Behavioral Genetic Models

Next, we fit a multivariate common pathways model to examine age differences in domain-general and domain-specific genetic and environmental influences on early child abilities. This model is written as

**Table 3. Phenotypic Model Fit Statistics**

| Model | | AIC | BIC | LL | *df* | MLR Scaling | $\chi^2$ for Model Comparison | $\Delta df$ |
|---|---|---|---|---|---|---|---|---|
| 1 | Freely estimated | 8773.99 | 8916.10 | −4354.00 | 33 | 2.01 | – | – |
| 2 | Proportional | 8782.49 | 8890.14 | −4266.24 | 25 | 2.23 | −132.72 | 8 |
| 3 | Same across domains | 8782.27 | 8889.93 | −4366.14 | 25 | 2.22 | 0.21[a] | 0 |
| **4** | **No interaction at all** | **8779.92** | **8878.97** | **−4366.96** | **23** | 2.23 | 0.78 | 2 |

*Note*. Bolded = preferred model. $\chi^2$ for model comparison was calculated by comparing the nested model with the previously listed comparison model. 1 = Phenotypic confirmatory factor analysis with each age-related interaction coefficient independently and freely estimated. 2 = Phenotypic confirmatory factor analysis with each age-related interaction coefficient constrained to be proportional to the main effect of the latent factor on the corresponding observed domain of development. 3 = Phenotypic confirmatory factor analysis with each age-related interaction coefficient constrained to be the same across domains of development. 4 = Phenotypic confirmatory factor analysis with no age-related interactions.

[a]difference in BIC is calculated to compare model fitness across Models 2 and 3 as the $\chi^2$ values are equivalent and the degrees of freedom are the same across the two models.

$$G[x]_n = \upsilon[x] + \alpha[x] \times age_n + (a_c + a_c' \times age_n) \times (\lambda[x] \times g_n) \times A_{cn} + (c_c + c_c' \times age_n) \times (\lambda[x] \times g_n) \times C_{cn} + (e_c + e_c' \times age_n) \times (\lambda[x] \times g_n) \times E_{cn} + (a_u[x] + a_u'[x] \times age_n) \times A_u[x]_n + (c_u[x] + c_u'[x] \times age_n) \times C_u[x]_n + (e_u[x] + e_u'[x] \times age_n) \times E_u[x]_n$$

In this behavioral genetic model, the factor loading of each domain on the latent factor *Broad Ability* was constrained to be age-invariant (as measurement invariance was observed from the preferred phenotypic model). Variance of *Broad Ability* and unique variance of each domain were each divided into three biometric components: genes, with $A_c$ representing common (domain-general) genetic factors and $A_u$ representing unique (domain-specific) genetic factors; shared environmental factors that made the twins more similar to each other, with $C_c$ representing domain-general shared environmental factors and $C_u$ representing domain-specific shared environmental factors; and nonshared environmental factors that are unique to each child and made the twins less similar to each other, with $E_c$ representing domain-general nonshared environmental factors and $E_u$ representing domain-specific nonshared environmental factors. $E_u$, at the measurement level, also includes measurement error.

In the equation for this behavioral genetic model, each score is determined by a combination of factors: $\upsilon[x]$ represents the regression intercept for a given domain of early child abilities, $\alpha[x]$ represents the main effect of age on a given domain of early child abilities (freely estimated, but expected to be 0, given that each ability was residualized for age prior to analyses), $\lambda[x]$ represents the main effect of *Broad Ability* on a given domain of early child abilities, $g$ represents the common latent factor *Broad Ability*, $a_c$ represents the main effect of domain-general genetic factors ($A_c$), $a_c'$ represents the interaction between age and $A_c$, $c_c$ represents the main effect of domain-general shared environmental factors ($C_c$), $c_c'$ represents the interaction between age and $C_c$, $e_c$ represents the main effect of domain-general nonshared environmental factors ($E_c$), $e_c'$ represents the interaction between age and $E_c$, $a_u[x]$ represents the main effect of genetic factors unique to a given domain of early child abilities ($A_u[x]$), $a_u'[x]$ represents the interaction between age and $A_u[x]$, $c_u[x]$ represents the main effect of shared environmental factors unique to a given domain of early child abilities ($C_u[x]$), $c_u'[x]$ represents the interaction between age and $C_u[x]$, $e_u[x]$ represents the main effect of nonshared environmental factors unique to a given domain of early child abilities ($E_u[x]$), and $e_u'[x]$ represents the interaction between age and $E_u[x]$. The subscript $n$ signifies terms that are allowed to vary by individuals. The latent variables $g$, $A_c$, $C_c$, $E_c$, $A_u[x]$, $C_u[x]$, and $E_u[x]$ were scaled to a $z$-metric ($M = 0$, $SD = 1$).

As seen in the equation for the multivariate behavioral genetic model, each path representing genetic or environmental influences is a sum of (1) the main effect of that genetic or environmental factor (i.e., $a_c$, $c_c$, $e_c$, $a_u[x]$, $c_u[x]$, and $e_u[x]$), and (2) its interaction with age (i.e., $a_c'$, $c_c'$, $e_c'$, $a_u'[x]$, $c_u'[x]$, and $e_u'[x]$). At the latent factor level, if genetic influences on *Broad Ability* increase as a function of age (i.e., $a_c'$ is greater than zero at a statistically significant level), this suggests that genetic commonality in early child development grows with age. If any of the domain-specific genetic influences increases as a function of age (i.e., $a_u'[x]$ is greater than zero at a statistically significant level), this suggests that the importance of specialist genes in early child development grows with age. Note that age differences can occur exclusively at the broad factor level, the measurement level, or co-occur at both levels.

**Table 4. Parameter Estimates (with C.I. in brackets) From Phenotypic Confirmatory Factor Analyses**

| | | 1 | | 2 | | 3 | | 4[a] |
|---|---|---|---|---|---|---|---|---|
| Measures | Latent Factor | Main Effect | Interaction | Main Effect | Interaction | Main Effect | Interaction | Main Effect |
| Communication | | **.65**[.36, .93] | −.02[−.11, .07] | **.65**[.42, .88] | | **.65**[.43, .87] | | **.60**[.46, .73] |
| Gross Motor | | **.49**[.19, .76] | .05[−.05, .14] | **.64**[.44, .84] | | **.65**[.43, .87] | | **.59**[.46, .72] |
| Fine Motor | Broad Ability | **.69**[.48, .91] | −.01[−.08, .07] | **.73**[.53, .94] | −.03[−.11, .05] × | **.73**[.53, .94] | −.02[−.08, .03] | **.68**[.56, .79] |
| Problem-Solving | | **.75**[.48, 1.02] | −.06[−.14, .03] | **.66**[.41, .91] | corresponding main effect | **.66**[.44, .88] | | **.61**[.47, .74] |
| Personal-Social | ON | **.78**[.51, 1.04] | −.06[−.15, .04] | **.70**[.47, .92] | estimate for each domain | **.70**[.48, .91] | | **.64**[.52, .77] |
| Communication | | **.82**[.66, .98] | −.03[.36, .93] | **.74**[.64, .84] | | **.74**[.64, .83] | | **.74**[.66, .81] |
| Gross Motor | | **.88**[.71, 1.05] | −.03[−.09, .02] | **.83**[.73, .93] | <.01[−.04, .04] × | **.83**[.73, .92] | <.01[−.03, .03] | **.83**[.76, .90] |
| Fine Motor | Unique variance | **.58**[.42, .73] | .05[−.01, .11] | **.71**[.61, .81] | corresponding main effect | **.71**[.60, .82] | | **.71**[.64, .78] |
| Problem-Solving | for each domain | **.86**[.68, 1.04] | −.04[−.10, .02] | **.76**[.65, .87] | estimate for each domain | **.76**[.66, .87] | | **.77**[.69, .84] |
| Personal-Social | | **.64**[.47, .81] | .04[−.03, .10] | **.72**[.62, .82] | | **.72**[.62, .82] | | **.72**[.65, .80] |

*Note*: Bolded = $p < .01$. 1 = Phenotypic confirmatory factor analysis with each age-related interaction coefficient independently and freely estimated. 2 = Phenotypic confirmatory factor analysis with each age-related interaction coefficient constrained to be proportional to the main effect of the latent factor on the corresponding observed domain of development. 3 = Phenotypic confirmatory factor analysis with each age-related interaction coefficient constrained to be the same across domains of development. 4 = Phenotypic confirmatory factor analysis with no age-related interactions.

[a]preferred model.

Similar to analyses conducted at the phenotypic level, we fit five versions of multivariate common pathways model and compared their model fit statistics to identify the best-fitting model. First, we fit a model in which all the interaction coefficients (i.e., $a_c'$, $c_c'$, $e_c'$, $a_u'[x]$, $c_u'[x]$, and $e_u'[x]$) were freely estimated. Second, we constrained the interaction coefficient to be proportional to the corresponding main effect in each regression path at the measurement level (i.e., $a_u'[x] = a_u[x] \times a_u'$, $c_u'[x] = c_u[x] \times c_u'$, and $e_u'[x] = e_u[x] \times e_u'$, where $a_u'$, $c_u'$, and $e_u'$ are invariant across domains). Third, we constrained the interaction coefficients to be same across domains of early child development for both genetic and environmental influences at the measurement level (i.e., $a_u'[x] = a_u'$, $c_u'[x] = c_u'$, and $e_u'[x] = e_u'$, where $a_u'$, $c_u'$, and $e_u'$ are invariant across domains). Fourth, we fixed all the interaction coefficients at the measurement level to zero (i.e., $a_u'[x] = c_u'[x] = e_u'[x] = 0$). Finally, we also fixed the interaction coefficients at the latent factor level to zero to test a model with no age interactions at all (i.e., $a_c' = c_c' = e_c' = a_u'[x] = c_u'[x] = e_u'[x] = 0$).

Table 5 lists the model fit statistics for all the behavioral genetic models. The behavioral genetic multivariate model with all interaction coefficients freely estimated fit the data best, and we, therefore, accept this model as the preferred behavioral genetic model. Combining information across the five abilities to conduct an omnibus test for age differences in genetic and environmental influences at the measurement level led to a significant loss of model fit. This suggests that age differences at the domain-specific level emerge independently for each domain.

Tables 6 and 7 list the parameter estimates from all the behavioral genetic models. At the domain-general level, we observed age differences in genetic and shared environmental influences but not nonshared environmental ones (see Tab. 6 and Figs. 3 and 4); genetic commonality increases while shared environmental commonality decreases with age. At the domain-specific level, there is little evidence for age differences in genetic and environmental influences, except for shared environmental influences on *Fine Motor* and nonshared environmental influences on *Fine Motor* and *Problem-Solving* (see Tab. 7 and Figs. 3 and 4). We focus on results from the preferred behavioral genetic model in the following section.

***Increasing Heritability.*** Unstandardized genetic variance increased from as low as .04 shortly after birth (e.g., for *Communication*, $[[.022 + 0 \times .105] \times 1]^2 + [.194 + 0 \times .070]^2$) to as high as .84 by age 6 (e.g., for *Personal-Social*, $[[.022 + 6 \times .105] \times 1.157]^2 + [.326 + 6 \times .032]^2$) for all domains of early child abilities

except *Gross Motor*, in which unstandardized genetic variance ranged from .59 (i.e., $[[.022 + 2.32 \times .105] \times .993]^2 + [.811 + 2.32 \times [-.038]]^2$) to .76 (i.e., $[[.022 + 6 \times .105] \times .993]^2 + [.811 + 6 \times [-.038]]^2$) across ages (see the fourth and fifth columns of Tabs. 6 and 7, and the first column of Fig. 3). Below, we describe the decomposition of genetic and environmental influences into generalist and specialist components.

***Generalist Genetic and Environmental Effects.*** Figure 3 illustrates age differences in generalist and specialist genetic and environmental influences on each domain of early child abilities. Areas highlighted in red (see the first column of Fig. 3) represent unstandardized variance in each domain explained by generalist genes. These panels demonstrate increasing unstandardized generalist genetic variance from almost 0 shortly after birth to approximately half a unit by age 6 for all domains of early child abilities. Areas highlighted in pink (see the second column of Fig. 3) represent unstandardized variance in each domain explained by generalist shared environmental factors. These panels demonstrate decreasing unstandardized generalist shared environmental variance from approximately half a unit shortly after birth to almost 0 by age 6 for all domains of early child abilities. Areas highlighted in dark red (see the third column of Fig. 3) represent unstandardized variance in each domain explained by generalist nonshared environmental factors. These panels demonstrate relatively trivial and constant influences of generalist nonshared environmental factors across ages on all domains of early child abilities.

***Specialist Genetic and Environmental Effects.*** Results indicate substantial genetic effects at the domain-specific level (see estimates for $A_u$ in the fourth and fifth columns of Tab. 7). However, none of the interactions between the domain-specific genetic factors and age reached statistical significance. In the first column of Figure 3, areas highlighted in blue represent unstandardized variance in each domain explained by specialist genes. These panels illustrate similar amounts of domain-specific genetic influences across ages for all domains of early child abilities.

We also observed substantial influences of specialist shared environmental factors (see estimates for $C_u$ in the fourth and fifth columns of Tab. 7). At the domain-specific level, environmental factors that made children more similar to each other explained a sizable amount of variations in each domain of early child abilities except *Gross Motor*, on which shared environmental factors had no effects at all. In the second column of Figure 3, areas highlighted in light blue represent unstandardized variance in each domain explained by specialist shared environmental factors. These panels illustrate relatively trivial age differences in domain-specific shared environmental influences except for *Problem-Solving*. At the domain-specific level, unstandardized variance in *Problem-Solving* explained by

**Table 5. Behavioral Genetic Model Fit Statistics**

| Model | | AIC | BIC | LL | *df* | MLR scaling | Model to be Compared With | $\chi^2$ for Model Comparison | $\Delta df$ |
|---|---|---|---|---|---|---|---|---|---|
| **1** | **Freely estimated** | **7153.69** | **7348.01** | **−3523.84** | **53** | **1.43** | 2 | 24.73* | 12 |
| | | | | | | | 3 | 29.94* | 12 |
| | | | | | | | 4 | 45.81* | 15 |
| | | | | | | | 5 | 70.40* | 18 |
| 2 | Proportional | 7156.46 | 7306.78 | −3537.23 | 41 | 1.54 | 3 | 4.57[a] | 0 |
| | | | | | | | 4 | 17.34* | 3 |
| | | | | | | | 5 | 40.55* | 6 |
| 3 | Same across domains | 7161.02 | 7311.35 | −3539.51 | 41 | 1.55 | 4 | 13.24* | 3 |
| | | | | | | | 5 | 35.49* | 6 |
| 4 | No interactions at domain-specific level | 7177.81 | 7317.13 | −3550.90 | 38 | 1.53 | 5 | 24.01* | 3 |
| 5 | No interactions at all | 7201.51 | 7329.84 | −3565.76 | 35 | 1.56 | – | – | – |

*Note*: Bolded = preferred model. 1 = Multivariate behavioral genetic model with each age-related interaction coefficient independently and freely estimated. 2 = Multivariate behavioral genetic model with each age-related interaction coefficient constrained to be proportional to the main effect of the corresponding latent factor on the corresponding observed domain of development. 3 = Multivariate behavioral genetic model with each age-related interaction coefficient constrained to be the same across domains of development for each type of domain-specific influences. 4 = Multivariate behavioral genetic model with no age-related interactions at the domain-specific level. 5 = Multivariate behavioral genetic model with no age-related interactions at all.

[a]difference in BIC is calculated to compare model fitness across Models 2 and 3 as the $\chi^2$ values are equivalent and the degrees of freedom are the same across the two models.

*$p < .05$.

**Table 6. Parameter Estimates (with C.I. in brackets) at the Domain-General Level**

| | | | 1[a] | | 2 | | 3 | | 4 | | 5 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Effects of | | | Main Effect | Interaction | Main Effect | Interaction | Main Effect | Interaction | Main Effect | Interaction | Main Effect |
| | | Communication | 1.00[b] | | 1.00[b] | | 1.00[b] | | 1.00[b] | | 1.00[b] |
| | | Gross Motor | **.99** [.69, 1.30] | | **.99** [.67, 1.29] | | **.99** [.69, 1.30] | | **1.03** [.70, 1.37] | | **1.00** [.69, 1.31] |
| Broad Ability | | Fine Motor | **1.21** [.91, 1.50] | – | **1.20** [.90, 1.50] | – | **1.21** [.90, 1.52] | – | **1.31** [.94, 1.68] | – | **1.25** [.93, 1.58] |
| | ON | Problem- Solving | **1.01** [.73, 1.28] | | **1.03** [.75, 1.31] | | **1.04** [.76, 1.32] | | **1.12** [.81, 1.43] | | **1.05** [.76, 1.34] |
| | | Personal- Social | **1.16** [.86, 1.45] | | **1.15** [.87, 1.43] | | **1.14** [.86, 1.42] | | **1.15** [.86, 1.44] | | **1.17** [.87, 1.46] |
| $A_c$ | | | .02[−.23, .28] | **.11** [−.02, .19] | −.07 [−.28, .14] | **.14** [.06, .21] | −.06 [−.28, .15] | **.14** [.06, .21] | .07 [−.15, .28] | **−.14** [−.21, −.06] | **.31** [.15, .46] |
| $C_c$ | | Broad Ability | **.71** [.44, .99] | **−.12** [−.22, −.02] | **.73** [.46, 1.01] | **−.13** [−.23, −.03] | **.73** [.46, 1.00] | **−.13** [−.23, −.03] | **.72** [.44, .99] | **−.13** [−.23, −.04] | **.44** [.28, .59] |
| $E_c$ | | | .13 [−.02, .28] | .02 [−.05, .08] | **.20** [.07, .33] | −.01 [−.07, .04] | **.20** [.05, .34] | −.01 [−.08, .05] | **−.14** [−.28, −.01] | −.01 [−.06, .05] | **.17** [.11, .24] |

*Note*: Bolded = $p < .05$. 1 = Multivariate behavioral genetic model with each age-related interaction coefficient independently and freely estimated. 2 = Multivariate behavioral genetic model with each age-related interaction coefficient constrained to be proportional to the main effect of the corresponding latent factor on the corresponding observed domain of development. 3 = Multivariate behavioral genetic model with each age-related interaction coefficient constrained to be the same across domains of development for each type of domain-specific influences. 4 = Multivariate behavioral genetic model with no age-related interactions at the domain-specific level. 5 = Multivariate behavioral genetic model with no age-related interactions at all.

[a]preferred model.

[b]first loading factor was fixed to one to facilitate model convergence.

**Table 7. Parameter Estimates (with C.I. in brackets) at the Domain-Specific Level**

| | | | 1[a] | | 2 | | 3 | | 4 | 5 |
|---|---|---|---|---|---|---|---|---|---|---|
| Effects of | | | Main Effect | Interaction | Main Effect | Interaction | Main Effect | Interaction | Main Effect | Main Effect |
| | | Communication | .19 [−.42, .81] | .07 [−.12, .26] | **.43** [.21, .65] | | **.44** [.21, .67] | | **.36** [.13, .59] | **.34** [.11, .57] |
| | | Gross Motor | **.81** [.59, 1.03] | −.04 [−.15, .07] | **.81** [.64, .99] | | **.82** [.66, .99] | | **.74** [.61, .87] | **.74** [.61, .87] |
| $A_u$ | | Fine Motor | **.39** [.01, .76] | −.09 [−.23, .04] | 0 ± .01 | −.05 [−.14, .04] | −.04 [−.24, .16] | −.04 [−.10, .02] | 0 ± .01 | 0 ± .01 |
| | | Problem-Solving | **.56** [.21, .90] | −.05 [−.15, .05] | **.51** [.13, .89] | | **.55** [.23, .87] | | **.44** [.16, .72] | **.48** [.20, .75] |
| | | Personal-Social | .33 [−.10, .75] | .03 [−.11, .17] | **.48** [.28, .67] | | **.48** [.28, .68] | | **.42** [.24, .59] | **.39** [.23, .56] |
| | | Communication | **.78** [.54, 1.02] | −.11 [−.21, <.01] | **.59** [.41, .76] | | **.59** [.44, .73] | | **.55** [.42, .68] | **.55** [.43, .68] |
| | | Gross Motor | 0 ± .01 | 0 ± .01 | 0 ± .01 | | .04 [−.07, .15] | | 0 ± .01 | 0 ± .01 |
| $C_u$ | ON | Fine Motor | **.48** [.27, .68] | .02 [−.06, .10] | **.59** [.47, .71] | −.04 [−.10, .03] | **.58** [.46, .70] | −.02 [−.05, .02] | **.54** [.46, .63] | **.54** [.45, .62] |
| | | Problem-Solving | **.74** [.55, .92] | **−.12** [−.17, −.07] | **.51** [.24, .77] | | **.50** [.28, .72] | | **.46** [.26, .65] | **.44** [.23, .65] |
| | | Personal-Social | **.45** [.15, .76] | .04 [−.07, .14] | **.58** [.42, .74] | | **.60** [.44, .76] | | **.54** [.40, .69] | **.55** [.42, .68] |
| | | Communication | **.36** [.15, .56] | <.01 [−.06, .07] | **.22** [.13, .31] | | **.23** [.13, .34] | | **.37** [.30, .44] | **.37** [.30, .44] |
| | | Gross Motor | .19 [−.14, .53] | .09 [−.05, .24] | **.27** [.16, .37] | | **.28** [.15, .42] | | **.41** [.28, .55] | **.41** [.28, .55] |
| $E_u$ | | Fine Motor | .12 [−.14, .38] | **.13** [.03, .22] | **.28** [.19, .37] | **.25** [.03, .46] | **.29** [.20, .39] | **.06** [.02, .09] | **.44** [.37, .52] | **.46** [.38, .53] |
| | | Problem-Solving | .16 [−.08, .40] | **.10** [<.01, .19] | **.25** [.14, .35] | | **.26** [.14, .37] | | **.41** [.29, .52] | **.41** [.29, .52] |
| | | Personal-Social | **.35** [.12, .58] | −.04 [−.14, .05] | **.15** [.07, .24] | | **.14** [.01, .27] | | **.26** [.16, .36] | **.25** [.15, .34] |

*Note*: Bolded = $p < .05$. 1 = Multivariate behavioral genetic model with each age-related interaction coefficient independently and freely estimated. 2 = Multivariate behavioral genetic model with each age-related interaction coefficient constrained to be proportional to the main effect of the corresponding latent factor on the corresponding observed domain of development. 3 = Multivariate behavioral genetic model with each age-related interaction coefficient constrained to be the same across domains of development for each type of domain-specific influences. 4 = Multivariate behavioral genetic model with no age-related interactions at the domain-specific level. 5 = Multivariate behavioral genetic model with no age-related interactions at all.

[a]preferred model.

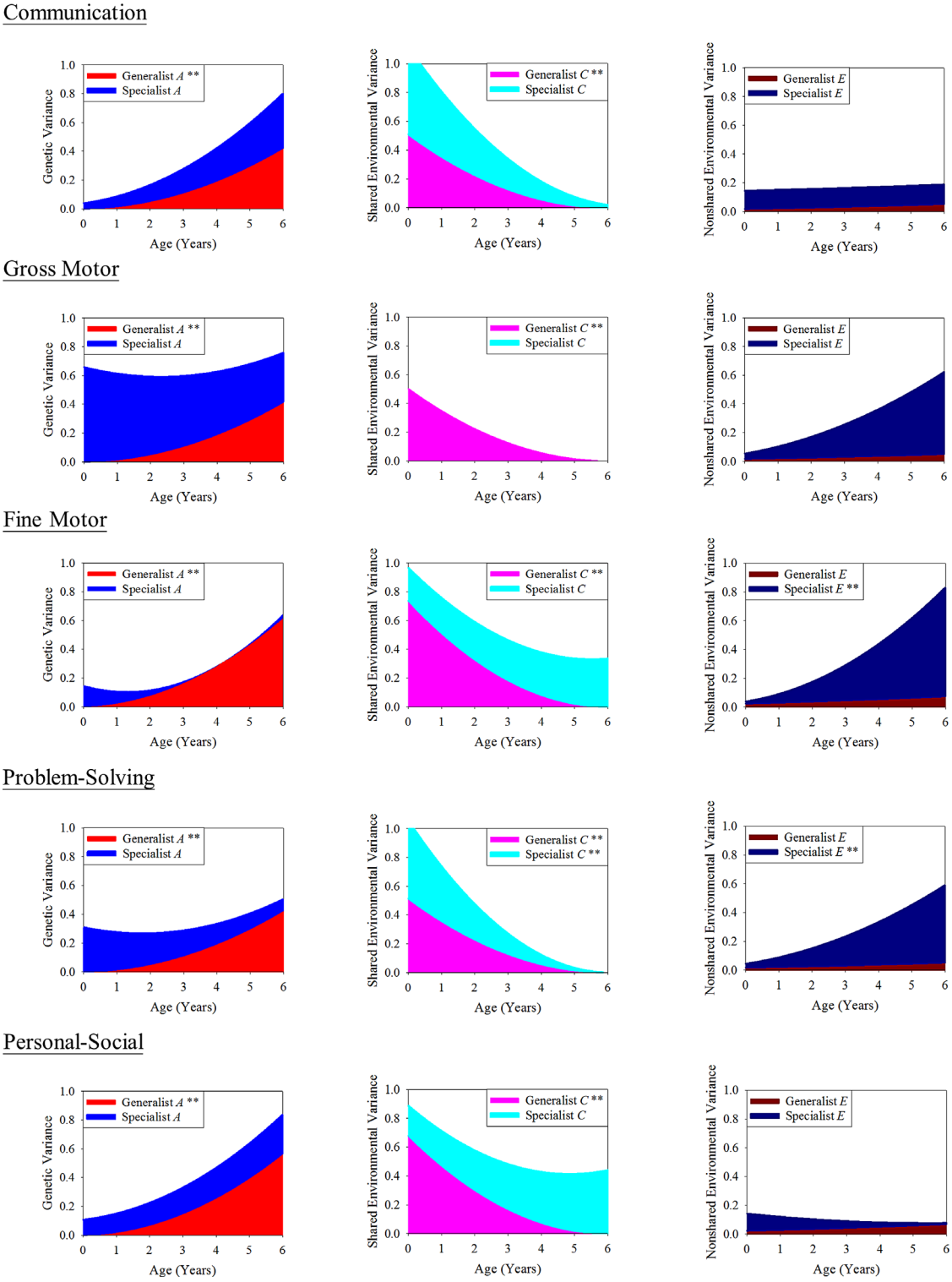


**FIGURE 3** Age trends in unstandardized genetic and environmental contributions to the five domains of early child abilities, decomposed into domain-general (generalist) and domain-specific (specialist) components. Estimates are based on expectations from the preferred behavioral genetic model (Model 1). Rows correspond to ability domains (*Communication*, *Gross Motor*, *Fine Motor*, *Problem-Solving*, and *Personal-Social*). Columns correspond to genetic, shared environmental, and nonshared environmental variance components.

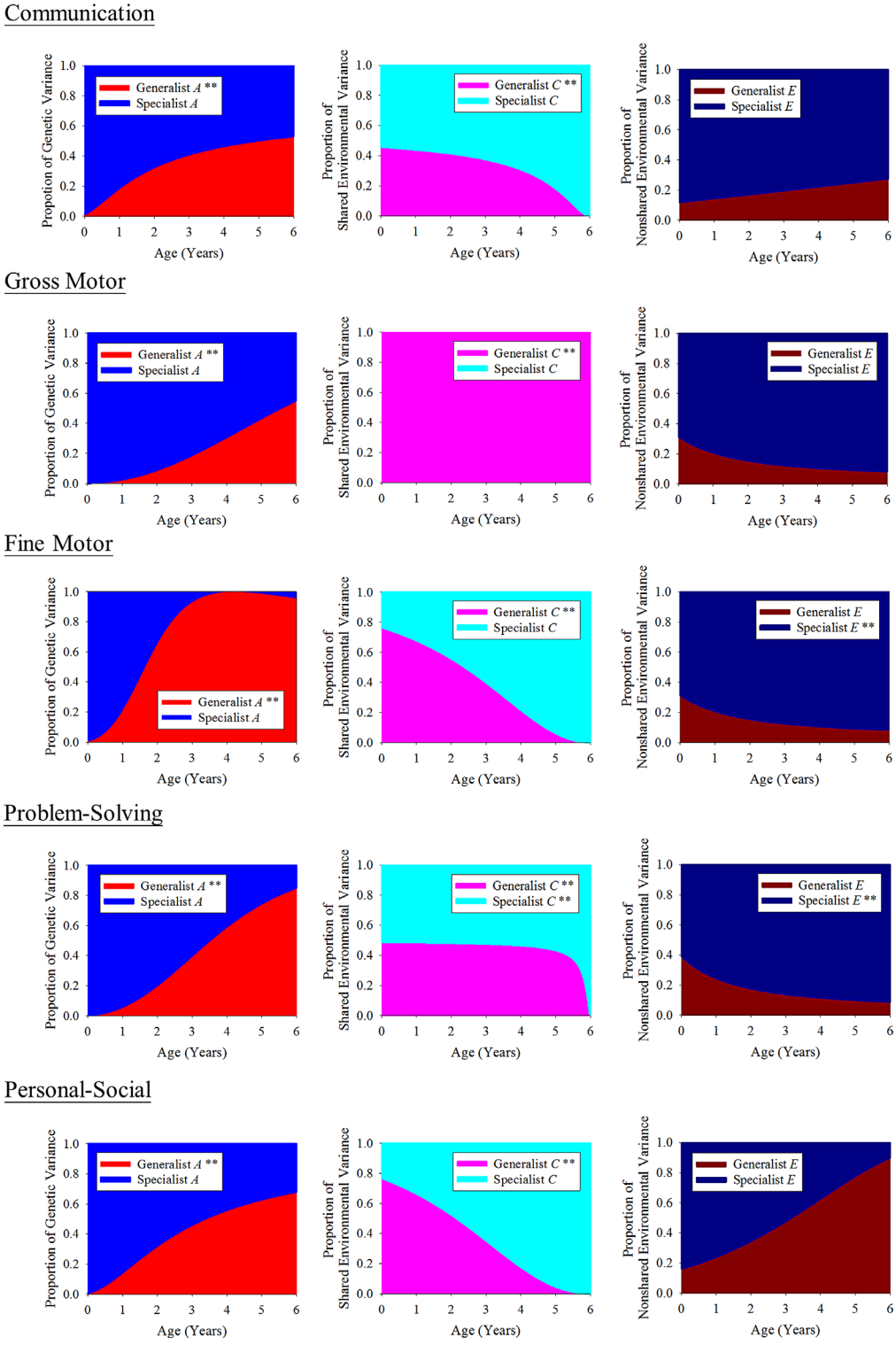


**FIGURE 4** Age trends in structure of total genetic and environmental contributions to the five domains of early child abilities, decomposed into domain-general (generalist) and domain-specific (specialist) proportions. Estimates are based on expectations from the preferred behavioral genetic model (Model 1). Rows correspond to ability domains (*Communication*, *Gross Motor*, *Fine Motor*, *Problem-Solving*, and *Personal-Social*). Columns correspond to genetic, shared environmental, and nonshared environmental variance components.

shared environmental factors decreased from .54 shortly after birth (i.e., $[.737 + 0 \times [-.120]]^2$) to almost 0 by age 6 (i.e., $[.737 + 6 \times [-.120]]^2$; see the fourth row of the second column of Fig. 3). For each of the other four domains of early child abilities, specialist shared environmental factors explained variation in young children's functioning to similar extents across ages.

Nonshared environmental influences at the domain-specific level are generally modest and only those on *Communication* and *Personal-Social* reached statistical significance (see estimates for $E_u$ in the fourth and fifth columns of Tab. 7). In the third column of Figure 3, areas highlighted in dark blue represent unstandardized variance in each domain explained by specialist nonshared environmental factors. These panels illustrate relatively trivial age differences in domain-specific nonshared environmental effects except for *Fine Motor* and *Problem-Solving*. Unstandardized nonshared environmental variance unique to *Fine Motor* increased from .01 shortly after birth (i.e., $[.120 + 0 \times .125]^2$) to .76 by age 6 (i.e., $[.120 + 6 \times .125]^2$; see the third row of the third column of Fig. 3). Similarly, unstandardized nonshared environmental variance unique to *Problem-Solving* increased from .03 shortly after birth (i.e., $[.164 + 0 \times .095]^2$) to .54 by age 6 (i.e., $[.164 + 6 \times .095]^2$; see the fourth row of the third column of Fig. 3). For the other three domains of early child abilities, influences of specialist environmental factors unique to a child remained modest across ages.

***Developmental Trends in Proportional Generalist and Specialist Genetic Effects.*** Our results, as detailed above, indicate that the importance of specialist genes remains similar while that of generalist genes grows with age. This suggests that age-related increase in heritability is localized to generalist genes in early child functioning. As demonstrated in the first column of Figure 4, this also means that the proportion of total heritability attributed to specialist genes decreases but the proportion attributed to generalist genes increases with age. These developmental changes in genetic structure demonstrate a growing statistical pleiotropy (i.e., overlap of genetic influences) across domains of early child abilities with age. Our results are, thus, consistent with the predictions of a transactional perspective, which holds that statistical pleiotropy strengthens over time.

## DISCUSSION

Domain-general genetic effects, i.e., statistical pleiotropy, on children's abilities have been well documented (e.g., Petrill, 2005; Plomin & Kovas, 2005; Plomin et al., 2007). Two major classes of underlying mechanisms have been postulated to account for domain-general genetic effects, and each provides different predictions for how statistical pleiotropy changes across development. The *endogenous* perspective predicts no age differences in the magnitude of statistical pleiotropy, whereas the *transactional* perspective predicts increasing statistical pleiotropy with age. With a twin sample ranging from ages 0 to 6 years, we tested for age differences in genetic and environmental influences on early child abilities at both the domain-general and domain-specific levels.

Consistent with the *transactional* perspective, our results indicate that age differences in genetic influences are localized to the domain-general level. Genetic influences on early child abilities gradually evolve from being predominantly modular shortly after birth to predominantly molar by school-entry age. Thus, as children develop, genes become more important in explaining variation in individual abilities as well as the association between different abilities. Transactional models (e.g., Dickens, 2007; van der Maas et al., 2006) predict that statistical pleiotropy emerges and strengthens over time via reciprocal effects of abilities on one another and between abilities and the environment. Such transactional processes may of course co-occur with other, possibly epigenetic, processes. For instance, as young children select and evoke experiences from their surroundings, those experiences may modulate gene expression (Tucker-Drob & Briley, 2014). At the same time, genes may become expressed or silenced over the course of development via biological maturation (see Briley & Tucker-Drob, 2013). Further research is needed to test and model such processes.

At the domain-specific level, we observed no age differences in genetic influences on early child abilities. At the same time, for four of the five domains of early child abilities, results indicate that total heritability increases with age. The combination of these findings suggests that, *relative to generalist genes*, specialist genes become less important in children's abilities with age. Our results also indicate that increasing heritability occurs mostly at the domain-general level. This is consistent with the hypothesis that, as children age and gain increasing autonomy, they mold and create experiences that both reinforce their initial genetic advantages (or disadvantages) and promote (or impede) their development in *multiple* domains of functioning. Because individual environmental experiences have the potential to stimulate multiple abilities simultaneously, an initial genetically influenced aptitude or proclivity in a particular domain might lead children to experiences

that promote their development across many different ability domains.

In contrast to our findings for the other four domains of early child abilities, we did not observe an increasing heritability for *Gross Motor*. While it is possible that there may truly be no substantial developmental changes in heritability of this particular domain of functioning until later in development, this possibility would appear at odds with the dramatic mean-level increases in gross-motor development during early life. Interestingly, at the descriptive level, Figure 3 indicates that generalist genetic influences on *Gross Motor* ability increase with age, while specialist genetic influences on *Gross Motor* ability decrease with age. Thus, the relatively stable total genetic effects on *Gross Motor* ability with age appear to have masked pronounced, yet opposing, developmental trends in its generalist and specialist genetic components.

Here, we reported one of the first studies of developmental changes in genetic structure of early child abilities. As sample sizes grow and longitudinal measures accumulate, we will be well-positioned to apply more specialized longitudinal models (e.g., growth curve models and cross-lagged models) to our data. Such models would allow us to more directly track developmental changes in total heritability and genetic commonality. Future work would also benefit from examining these topics at later developmental stages. As environmental exposures and life experiences in early and middle childhood tend to be very different, it is unclear how genetic commonality may unfold at later ages. For example, it is possible that genetic commonality may grow across lifespan as individuals gain more autonomy in creating experiences that reinforce their genetic predispositions and facilitate (or impede) their overall development. Alternatively, growth in genetic commonality may be slow later in development, as individuals become specialized in activities that they are particularly good at or enjoy doing.

## Strengths and Limitations

Our study is among the first to test for age differences in genetic and environmental influences on multiple domains of early child abilities at both the domain-general and domain-specific levels in the first years of life. This contrasts with behavioral genetic studies of cognitive development that conventionally begin following children only after school entry or, in instances in which early years of life are studied, typically focus on global, unidimensional measures of ability. Nevertheless, it is important that we also highlight our study's limitations.

First, the number of individual twins providing data for our analyses was relatively low in comparison to many behavioral genetic studies. However, higher ratios of indicator number to factor number and consistently high factor loadings have been shown to mitigate the impact of relatively small sample sizes on model results (see MacCallum, Widaman, Zhang, & Hong, 1999; also see Preacher & MacCallum, 2002). Moreover, we increased parameter precision by including longitudinal data from participants when available while employing estimation methods to prevent biases due to nesting of occasions within individuals. Additionally, rather than performing a large number of sequential hypothesis tests on a parameter-by-parameter basis, we used multivariate methods that compared different sets of parameter specification and constraint to one another. Our key findings derive from the joint pattern of results across all parameters in the model, and do not rely on a single key parameter or its $p$-value. Accordingly, we have taken an approach that emphasizes effect sizes, rather than significance levels.

Second, our findings are based on age-comparative analyses of data collected from individuals of different ages combined with those collected from the same individuals longitudinally. We used vertical scaling and created overlapping items across assessments for different age groups to ensure that scores produced are comparative across individuals of different ages for each domain. As mentioned earlier, we also used appropriate analytical procedures to account for the nonindependence of longitudinal data collected on the same individual. Nevertheless, future research would do well to capitalize on longitudinal data to fit explicit models of age-related change over time, rather than simply age-related difference.

Third, most parents of twins or multiples in this sample have completed college education or beyond. Developmental increases in heritability may not be as pronounced among more disadvantaged samples (Tucker-Drob et al., 2013; Tucker-Drob, Rhemtulla, Harden, Turkheimer, & Fask, 2011). While the initial sample included a larger portion of participants recruited through community outreach, the ongoing recruitment focuses more heavily on identifying eligible families from birth records provided by the Texas Department of State Health Services. Such effort should add further socioeconomic and ethnic diversity to the sample as it grows.

Fourth, data were collected using surveys completed by the twins' primary caregivers and are, thus, potentially subject to social desirability and the primary caregivers' biases about their children's abilities. However, instead of relying on parents' subjective impression of their children's development of various

skillsets, this assessment of early child abilities is based on parents' report on children's performance on concrete tasks (e.g., *Does your child count up to 15 without making mistakes? If so, mark "yes." If your child counts to 12 without making mistakes, mark "sometimes."*). These tasks are designed to objectively reflect children's attainment of various developmental milestones in different domains of early child functioning. This reduces primary caregivers' biases and misjudgments as compared to sole reliance on their subjective impression of their children's abilities relative to other same-age children. All primary caregivers also completed the surveys in the privacy of their homes, which can effectively reduce social desirability when self-reporting personal behaviors (Richman, Kiesler, Weisband, & Drasgow, 1999).

Most importantly, parent ratings on the ASQ have been shown to correlate with independent observer ratings at .86, indicating excellent inter-rater reliability across informants (Squires et al., 2009). The ASQ has also consistently demonstrated high convergent validity with researcher/clinician-administered scales of early mental development across a number of independent studies. Squires and colleagues compared the parent-administered ASQ's sensitivity to young children's progress in attaining developmental milestones to that of the examiner-administered Battelle Development Inventory and found that the ASQ demonstrated high sensitivity and specificity (i.e., .86 on both indices). Compared to classification based on scores on the examiner-administered Bayley, one gold standard in assessing early child development, the parent-administered ASQ demonstrated sensitivity as high as 1.00 and specificity as high as .97 (Gollenberg et al., 2010; Schonhaut et al., 2013; Simard et al., 2012). In particular, Schonhaut and colleagues found a moderately high correlation of .51–.75 between ASQ and Bayley scores among children of ages 8, 18, and 30 months. Using an international sample of 828 children of ages 12–60 months, Yu et al. (2007) found that ASQ demonstrated sensitivity of .63–.97 and specificity of .81–.84 when results were compared to those based on clinical examinations and neurodevelopmental assessments such as the Bayley, Griffiths Mental Development Scales, and Denver Developmental Screening Test.

Fifth, in our study, the same primary caregiver rated both twins in each pair. Using data from single informant may inflate the similarity in ratings across twins in a pair and, hence, the genetic and/or shared environmental variance estimates at a given time point. Yet, we do not expect such single-informant biases to systematically increase or decrease across age. Our key findings focus on the general pattern of developmental changes observed in heritability across major domains of early child abilities rather than the magnitude of a given estimate at a given time point.

Sixth, it is unclear whether the age-related increases we observed in nonshared environmental influences on *Fine Motor* and *Problem-Solving* at the domain-specific level indicate growing influences of environmental factors that are unique to a child or simply an increase in measurement error across item sets of increasing difficulty. Studies interested in testing for age differences in domain-specific nonshared environmental influences may, for example, include survey items as observed indicators in their multivariate models and set various domains of abilities as latent factors subordinate to the single latent factor representing overall ability. Because nonshared environments include measurement error only at the observed variable level, separating domain-specific variance into latent and observed components is one potential way to capture any true specialist nonshared environmental influences and their changes across development.

## CONCLUSION

Our study is among the first to test for age differences in the multivariate genetic structure of early child abilities. Results indicate that age-related increases in the heritabilities of early child abilities are mostly driven by the growing influence of generalist genes. These results are consistent with transactional models that predict strengthening of statistical pleiotropy over time via reinforcing transactions among different abilities and between these abilities and the environment.

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