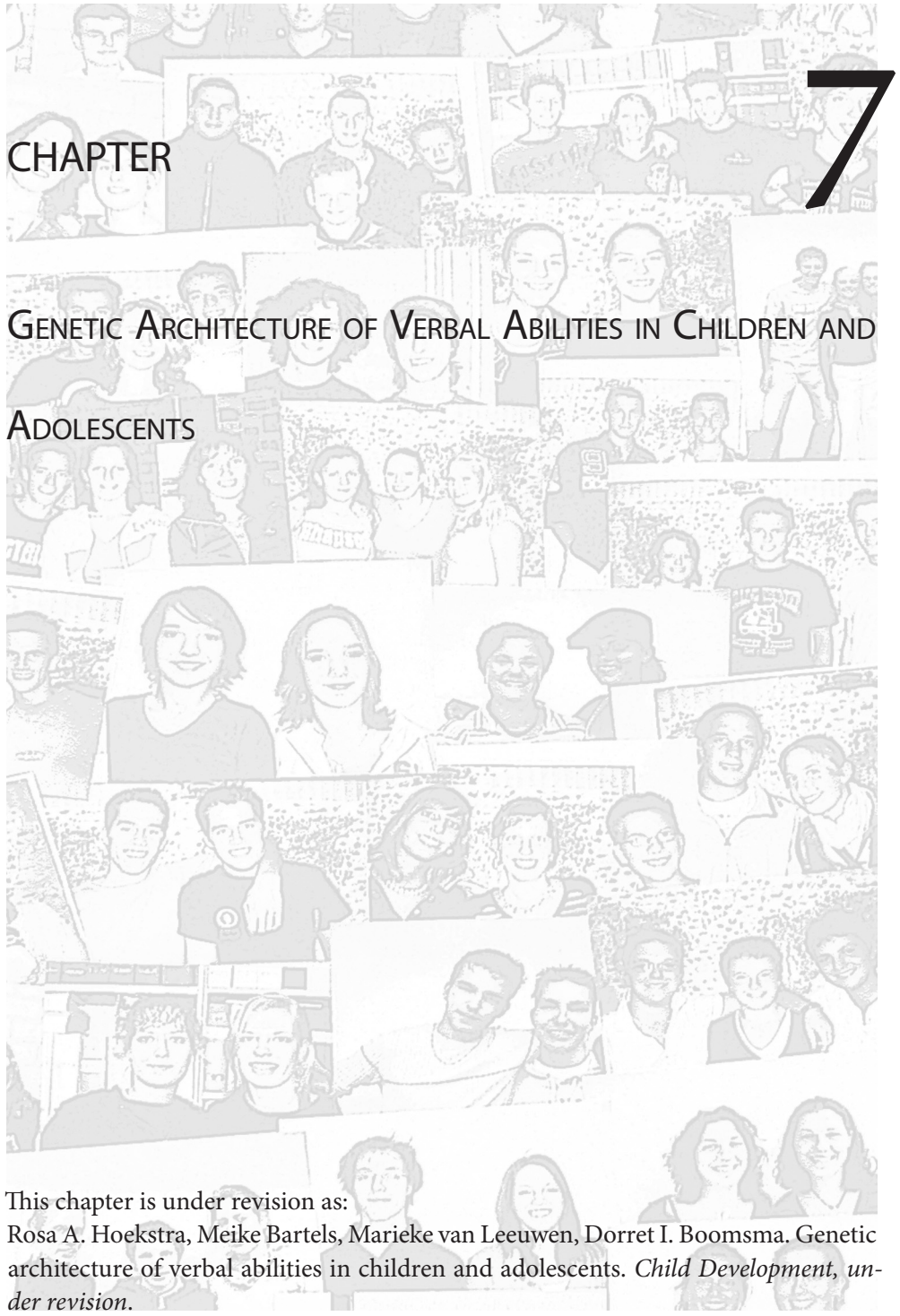

CHAPTER 7

Genetic Architecture of Verbal Abilities in Children and Adolescents

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Abstract

The genetic aetiologies of variation in verbal abilities, including Wechsler verbal IQ (VIQ), verbal learning and memory, and letter and category fluency were examined in two independent samples of 9-year-old and 18-year-old twin pairs and their siblings. In both age groups, genetic effects were strong for VIQ, and moderate for verbal learning, memory, and fluency. Remarkably, all familial resemblance was explained by shared genes without any influence of shared environment. There was significant covariance among the tests, which was mainly explained by genetic effects, both in middle childhood and in late adolescence. The genetic correlations between the verbal abilities were somewhat stronger in the adolescent cohort, suggesting increasing genetic unidimensionality with age.

Introduction

The development of verbal abilities form a crucial part of a child's maturation process. Verbal abilities are key components for acquiring language, and learning how to read and write. Moreover, verbal abilities are needed for healthy social communicative functioning. A thorough understanding of the aetiology of individual differences in verbal abilities, of how different verbal abilities are related to one another, and about the development of these abilities over time, is therefore highly relevant. Adolescence is a critical period in cognitive and brain development. In this phase of life prominent developmental changes are seen in the prefrontal cortex and the limbic brain regions, including the hippocampus (Spear, 2000). These brain changes coincide with the development of increasingly elaborate cognitive abilities (Casey et al., 2000; Durston & Casey, 2006). Important aspects of verbal development such as verbal learning, verbal memory and verbal fluency continue to improve during late childhood (Sincoff & Sternberg, 1988; Van den Burg & Kingma, 1999) and adolescence (Clark et al., 2006; Levin et al., 1991), and these advances may be related to the changes in the brain (Durston & Casey, 2006; Gaillard et al., 2000). For instance, Gaillard et al. (2000) examined verbal fluency performance in a functional MRI study and found worse performance in children compared to young adults, together with less focal activation of the cortex area in children. Before understanding how brain maturation and cognition are related in adolescence, a thorough understanding of the cognitive development by itself in this time period is needed.

Verbal learning and memory involve the process of registration, storage, retention, and retrieval of verbal information (Lezak, 1995). This process can be assessed with tests such as the Rey's Auditory Verbal Learning Test (AVLT) or the California Verbal Learning Test (CVLT), which yield reliable indices of word learning and declarative memory (Lezak, 1995; Mulder et al., 1996; Van den Burg & Kingma, 1999). Tasks of verbal fluency measure the spontaneous generation of words and thus provide a test for language production and retrieval and access to the verbal lexicon. Moreover, verbal fluency tasks require cognitive flexibility (for rapidly shifting from one word to the next) as well as response inhibition and therefore also provide a test of executive functioning (Mitrushina et al., 1999). Girls tend to outperform boys both on tests of verbal learning and memory (Van den Burg & Kingma, 1999), and verbal fluency (Sincoff & Sternberg, 1988). Moreover, these tests are found to be positively related to verbal intelligence (Bolla et al., 1990). However, the precise nature of the overlap between general verbal intelligence, such as Wechsler verbal IQ scores (Wechsler, 1997; Wechsler, 2002), and more specialised verbal abilities such as verbal learning,

memory and fluency, is not well known. Moreover, little is known about the relation between these abilities over the course of development.

A powerful method to examine the aetiology of individual differences in cognitive abilities is the study of genetically related individuals. In the last two decades, twin, family and adoption studies have generated a wealth of knowledge about the genetic and environmental influences on various cognitive abilities, including verbal abilities. These studies show that genetic factors on general verbal intelligence increase over the life time, while the influence of shared environmental factors (influences from the environment that are shared between family members, and that thus make relatives more alike) decrease. Within the Twins Early Development Study (TEDS), variance in general verbal intelligence in infants was found to be strongly influenced by shared environmental effects, and only moderately (about 25%) by genetic effects (Price et al., 2000). The heritability increases to about 50% in middle childhood (Hoekstra et al., 2007) and to about 85% in adulthood (Hoekstra et al., 2007; Posthuma et al., 2001; Rijsdijk et al., 2002). In parallel, shared environmental influences decrease at later ages in childhood (Hoekstra et al., 2007) and become non-significant by adolescence (Hoekstra et al., 2007; Posthuma et al., 2001; Rijsdijk et al., 2002). The Colorado Adoption Project (CAP) has collected data on the development of cognitive abilities in adopted children and their adoptive and biological parents, and in non-adoptive families. The heritability of verbal abilities increased from 11% when the children were 4 years of age (Rice et al., 1989) to 24% and 26% when the children were 7 (Alarcón et al., 2003) and 12 years old (Alarcón et al., 1998; Alarcón et al., 2003), to 64% when the offspring was 16 years old (Alarcón et al., 1999).

The genetic and environmental influences on early childhood verbal short term memory and verbal fluency have been studied both in twins from TEDS, and in an international twin sample. In 4.5-year-old twins from TEDS, the heritability of both abilities was moderate (respectively 36% and 40%), while shared environmental influences were not significant (Kovas et al., 2005). In a combined sample of Australian, Scandinavian, and American children (Samuelsson et al., 2005), more prominent genetic influences were detected, both for verbal short term memory (57%) and for "rapid naming" (a measure thought to reflect verbal fluency; 64%). In this sample, modest influences of the shared environment were found for verbal memory (29%). In a study in 6-year-old twin pairs overrepresented with children at risk for language impairment, phonological short term memory was found to be under substantial (61%) genetic influence, while shared environmental effects were non-significant (Bishop et al., 2006). Studies in later phases of childhood have mainly focused on reading abilities and found significant genetic influence on teacher-rated reading achievement (Harlaar et al., 2007) and word recognition (Gayan & Olson, 2003). Studies into specific verbal abilities in adult samples are scarce. Swan et al.

(1999) examined verbal learning and memory in aging male twins and reported a heritability of 56%. In the same study sample, individual differences in verbal fluency were explained by moderate genetic influences (34%), a (statistically non-significant) shared environmental component (18%) and nonshared environmental influences (48%; Swan & Carmelli, 2002). In 18- to 25-year-old female twins, free recall of unrelated words and categorised words were both moderately heritable (respectively 55% and 38%; Volk et al., 2006). Ando et al. (2001) studied verbal and spatial working memory in a sample of 16- to 29-year-old twins and also included verbal and spatial ability scores on a standardised intelligence test. Verbal working memory was moderately heritable (43-48%), while general verbal ability was under strong genetic influence (65%). A common genetic factor explained 20-26% of the variance in the verbal tasks, suggesting that some of the genetic influences were general, while the rest of the variance was modality or test-specific.

To summarise, twin and adoption studies into general verbal intelligence indicate increasing genetic effects and decreasing shared environmental influences over the course of development. Studies on verbal memory and verbal fluency in early childhood and in adulthood suggest moderate to strong genetic influences, while shared environmental effects appear not to play a major role. It is less clear whether these different verbal abilities are influenced by common genetic factors or by ability specific genetic factors. Rather than studying the aetiology of individual differences separately for each variable under investigation (using univariate genetic analyses), the use of multivariate genetic analyses enables disentangling genetic and environmental effects on the covariance *between* traits. This way, it can be examined whether specific cognitive abilities show genetic or environmental overlap with other cognitive abilities. Moreover, the genetic correlation (r_g, the extent to which the actual genes influencing one trait correlate with the genes involved in another trait) between specific cognitive abilities can be studied.

Using the TEDS sample, measures of vocabulary and grammar were found to be substantially correlated in 2-year-old twins, both at the phenotypic (r_{ph}=.66) and the genetic (r_g=.61) level (Dale et al., 2000). In 4.5-year-old twins from TEDS, verbal category fluency correlated moderately with other measures of language development and the genetic correlations were substantial, ranging from .48 to .96 (Hayiou-Thomas et al., 2006). These results suggest common genetic influences on diverse aspects of language. Hohnen and Stevenson (1999) found a common genetic factor influencing literacy, phonological awareness and language in 6- to 7-year-old children, but also found evidence for test-specific genetic influence. An international study including 7-year-old twins (Byrne et al., 2007) found high phenotypic and genetic correlations between measures of word identification, reading comprehension and spelling. On the other hand, the correlations between these measures and

verbal learning were only moderate. All in all, the results from multivariate genetic studies into specific verbal and language abilities yield mixed results. The phenotypic and genetic correlations vary, depending on the age of the studied population and on the measures under study.

The current study aims to examine the relationship between general verbal ability, as measured with the Wechsler verbal intelligence scale, with more specialised verbal abilities such as verbal learning, memory and fluency. We investigated the genetic and environmental influences on the overlap between these abilities in two distinct phases of development: 1) middle childhood, by studying verbal abilities in 9-year-old twins and their siblings (also referred to as the "child cohort"); and in 2) late adolescence, by studying performance on verbal tests in 18-year-old twins and their siblings (the "adolescent cohort"). In most twin studies, only data of the twins are collected. Our study includes siblings of twins and we are able to test whether the covariance structure in the data is the same for all first-degree relatives (twins and siblings) or whether there is evidence for twin-specific effects.

Methods

Participants

All twin families were recruited via the Netherlands Twin register (NTR), kept by the Department of Biological Psychology at the VU University in Amsterdam (Bartels et al., 2007; Boomsma et al., 2006). The current project includes data from two longitudinal studies. The child cohort took part in a study into brain development and cognition in early puberty and consisted of a group of 112 9-year-old twin pairs (mean age 9.10 years, sd=0.10) and their 9- to14-year-old siblings (n=100, mean age 11.84 years, sd=1.16). Since these twin families also took part in an MRI study, there were some exclusion criteria, such as having a pacemaker or braces (Van Leeuwen et al., 2007a). Of all participating twin pairs, 23 were monozygotic males (MZM), 23 dizygotic males (DZM), 25 monozygotic females (MZF), 21 dizygotic females (DZF), and 20 were dizygotic twin pairs of opposite sex (DOS). For the same sex twin pairs, zygosity determination was based on DNA polymorphisms (88 twin pairs), or on questionnaire items (4 pairs; (Rietveld et al., 2000). There were 43 male and 57 female siblings. The adolescent cohort took part in a longitudinal study into the development of cognition and behavioural problems (see Hoekstra et al. (2007) and Bartels et al. (2002) for details on the longitudinal data collection). This group consisted of 186 families of 18-year-old twin pairs (mean age 18.18 years, sd=0.21) and their siblings (n=93, mean age=18.51 years, sd=4.73), and comprised 33 MZM pairs, 34 DZM pairs, 44 MZF pairs, 38 DZF pairs, and 37 DOS pairs. The zygosity of the same sex twin pairs was determined by DNA analyses (128 pairs), blood group polymorphisms (19 pairs), or questionnaire items (2 pairs; Rietveld et al., 2000). There were 46 male and 47 female siblings in this cohort. Both studies were approved by the Central Committee on Research Involving Human Subjects and the institutional review board of the VU University Amsterdam. Written informed consent was obtained from all participants who were 18 years of age or older, and from the parents of all underage participants.

Test procedures

In both study cohorts, the cognitive testing took place at the laboratory of the VU University. The cognitive test protocol in the child cohort started in the morning and took approximately 5 hours to complete, including breaks. The families of the adolescent cohort were seen by a paediatrician in the morning, who studied their physical development. These twin families completed the cognitive test protocol in the afternoon, which took about 3.5 hours, including a break. In both studies, children from the same family were tested on the same day in different rooms by experienced test administrators.

Measures

All participants in the child cohort and the siblings of the older cohort who were younger than 16 years, completed the full Wechsler Intelligence Scale for Children-Third edition (WISC-III; Wechsler, 2002). Verbal IQ (VIQ) scores were determined as the standardised score on 5 verbal subtests. The standardised scores were based on results of same-aged children from the Netherlands. All participants of 16 years of age or above (all part of the adolescent cohort) completed 11 subtests from the Wechsler Adult Intelligence Scale-Third edition (WAIS-III; Wechsler, 1997). Verbal IQ was calculated as the mean subtest score on 6 verbal subtests. The subtests were standardised for the appropriate age group, based on a population sample of same-aged subjects in the Netherlands.

In the child cohort, verbal learning and memory was assessed using the Dutch version of Rey's Auditory Verbal Learning Test (AVLT; Van den Burg & Kingma, 1999). In this task, a list of 15 unrelated, concrete nouns (e.g. bird; pencil) is presented over 5 learning trials and immediate recall is tested following each presentation. Twenty to thirty minutes after the fifth presentation, delayed recall is assessed. During the time interval, the children completed an emotion recognition task and an inspection time task. These tasks are nonverbal and non-memory related, and would therefore not interfere with the AVLT performance. Verbal learning was measured as the total number of correct words over the 5 learning trials. Verbal memory was assessed as the total number of words recalled after the delay. The test retest reliabil-

ity of the AVLT has been examined using parallel tests in 225 Dutch school children (Van den Burg & Kingma, 1999). Verbal learning and memory were found to be the most reliable measures of the task, with test-retest correlations of .70 (learning) and .62 (memory).

The 18-year-old twins and their siblings completed the Dutch adaptation of the California Verbal Learning Test (CVLT; Mulder et al., 1996). In this task, a list of 16 items, with four words from each of four categories (fruits; herbs and spices; clothing; tools) is presented. Similar to the procedure in the AVLT, the list is presented 5 times, and the participant is instructed to recall as many words as possible from the list following each presentation. Subsequently, a second list consisting of 16 items is presented (the interference list). After hearing the interference list, the subject is asked to recall as many items from the original list (short delay free recall), and to recall as many items from each of the semantic categories (cued recall). After a time interval of 20 minutes (in which the participants completed an emotion recognition task and an inspection time task), long delay free recall of the items is assessed, followed by an assessment of cued recall, and recognition of the items. Verbal learning was measured as the total number of recalled items on the 5 trials; memory was assessed as the total number of items recalled in the long delay free recall phase. The test-retest reliability of verbal learning in the Dutch CVLT has been examined in 17-74 year-old healthy subjects (Mulder et al., 1996) and was found to be .62 using a parallel test (n=384), and .58 when compared to the AVLT (n=108). Mulder et al. (1996) did not examine the test-retest reliability of memory. In a pilot study of our twin family project we examined the test-retest reliability of the CVLT in 29 healthy subjects aged 14-20 years, with an inter test interval of 2-3 weeks (see Van Leeuwen et al. (2007b) for details on the procedure of this pilot), and found a test-retest correlation of .86 for verbal learning, and of .66 for memory.

In both the child and adolescent cohort, tests of verbal fluency were administered. This test evaluates the spontaneous production of words starting with a certain letter (verbal fluency letters) or belonging to a certain semantic category (verbal fluency categories) within a limited amount of time. The participants completed 2 trials for both conditions, and were instructed to name as many words as possible in one minute starting with an R or a T (letter trials), or belonging to the category "animals" or "professions" (category trials). Within the letter trials, subjects were prohibited from saying proper nouns (e.g. Robert or Rotterdam) or saying the same word twice using a different ending (e.g. roast and roasted). To control for quantitative differences between trials within one condition (e.g. on average, the subjects named more animals than professions), Z-scores were calculated for each trial. Letter fluency was measured as the mean Z-score over the two letter trials; category fluency was calculated as the mean Z-score of the semantic trials. We also examined the test-retest reliability of verbal fluency in our pilot and found a test-retest correlation of .70 for letter fluency and of .93 for category fluency. To our knowledge, no other studies have assessed the test-retest reliability of verbal fluency in the Dutch language.

Statistical analyses

All analyses were carried out using structural equation modelling in the software package Mx (Neale et al., 2006). The significance of the effects of sex and age on the means of all verbal abilities was tested in a saturated model, which only specified that the multivariate data from family members could be correlated, but which did not impose any theoretical model on the covariance structure. The saturated model was used to estimate the correlations between all phenotypes in both age cohorts. It was tested whether the phenotypic covariance structure was the same in the two cohorts. The saturated model was also used to estimate twin and twin-sibling correlations, and twin and twin-sibling cross correlations (e.g. the correlation between VIQ in the oldest of the twin and verbal learning in the youngest of the twin). All data were analysed, including data of incomplete twin pairs and data of twins without an additional sibling using the raw data option in Mx.

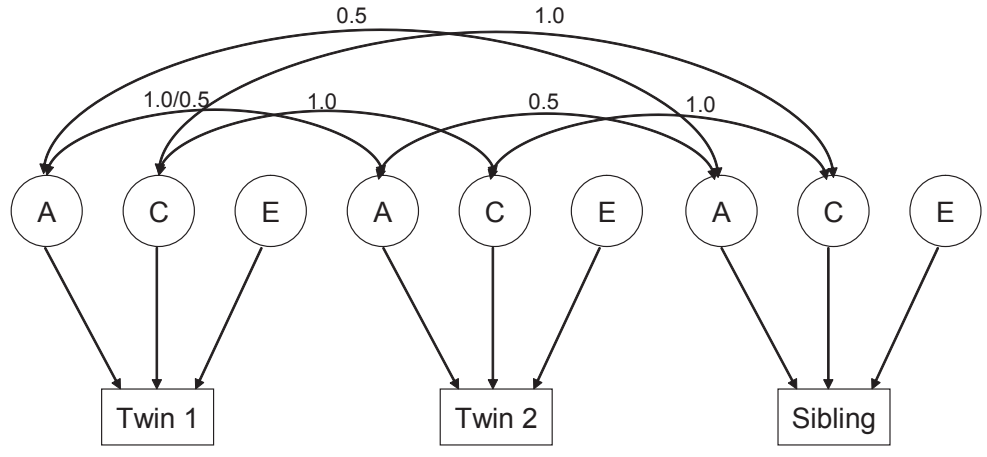

FIGURE 1. Univariate path diagram representing the contribution of additive genetic (A), shared environmental (C) and nonshared environmental (E) influences on the trait under investigation. The correlation of additive genetic factors is 1.0 in monozygotic twins, and, on average, 0.5 in dizygotic twins and between twins and siblings. The correlation of shared environmental effects is 1.0 between twins and between twins and siblings. Nonshared environmental effects represent influences unique to a family member and are thus uncorrelated.

Genetic modelling

Monozygotic (MZ) twins are (nearly) genetically identical, while dizygotic (DZ) twins and non-twin siblings share on average 50% of their segregating genes. Genetic model fitting of twin-sibling data allows for separation of the phenotypic variance into its genetic and environmental components (Figure 1). Additive genetic influences (A) result from the additive effects of alleles at each contributing genetic locus. Shared environmental influences (C) represent the environmental effects common to all offspring of the family. Nonshared environmental influences (E) are the effects of the environment that are not shared by the family members (including measurement error). Comparing the resemblance of MZ twins with the resemblance of DZ twins and twin-sibling pairs can give a first indication of what influences are important in explaining the variance in test performance. If MZ and first-degree relative correlations are similar, shared environmental influences are likely to be important. Higher MZ correlations compared to DZ and twin-sibling correlations indicate that genetic effects play a role (Boomsma et al., 2002). By comparing the resemblance of DZ twins with the resemblance between twins and their non-twin siblings, it is possible to test whether there is evidence for a twin specific environment. Higher DZ twin correlations compared to twin-sibling correlations would suggest such an effect. The use of multivariate genetic analyses enables the distinction of genetic and environmental effects on the covariance between traits. If the cross correlations in MZ twins and first-degree relatives are similar, shared environmental effects are important for explaining the covariance between the different verbal abilities. Higher MZ twin cross correlations compared to the cross correlation in first-degree relatives would indicate that genetic effects play a role in the overlap between verbal abilities.

The relative importance of the components A, C, and E was estimated using structural equation modelling in Mx (Neale et al., 2006). Genetic modelling was performed following several steps. The influences of A, C, and E on all verbal measures and on the overlap between the measures were first examined in a multivariate triangular or Cholesky decomposition (Neale & Cardon, 1992). This model decomposes the phenotypic relations into genetic, shared environmental and nonshared environmental contributions to the variance of a trait and to the covariance between traits. All possible contributions are parameterised in the Cholesky decomposition; therefore it yields the best possible fit to the data. It is useful to gain a first insight in what factors are important for the variance and covariance of verbal abilities. An ACE Cholesky decomposition was applied both to the data of the child cohort and to the data of the adolescent cohort. We then tested whether the genetic and shared environmental effects were of significant importance, by assessing the deterioration of the model fit after each component was dropped from the model. Next, it was tested whether the genetic influences on all tests could be described by a genetic common factor model (Figure 2). This model assumes that there is one underlying factor (e.g. a general verbal intelligence factor) that influences the individual differences in performance in each verbal test. To take the variance specific to each test into account, test specific genetic influences were also allowed in this model. Similarly, a common factor model including test specific influences was applied for the environmental influences. A good fit of this model would imply that there is one environmental factor that influences variance in performance on all verbal tests. Lastly, a model was tested in which the nonshared environmental influences were constrained to be test specific. Nonshared environmental influences were still allowed to covary between verbal learning and memory and between letter fluency and category fluency, as these variables were derived from the same test. After comparing the fit of these models, a best fitting most parsimonious model was established for the child cohort. Next, it was tested whether this model also fitted well to the data of the adolescent cohort.

The fit of the different submodels was evaluated against the Cholesky decomposition using likelihood ratio tests and Akaike's information criterion. The likelihood ratio, which is the difference between minus twice the log likelihoods (-2 LL) of two

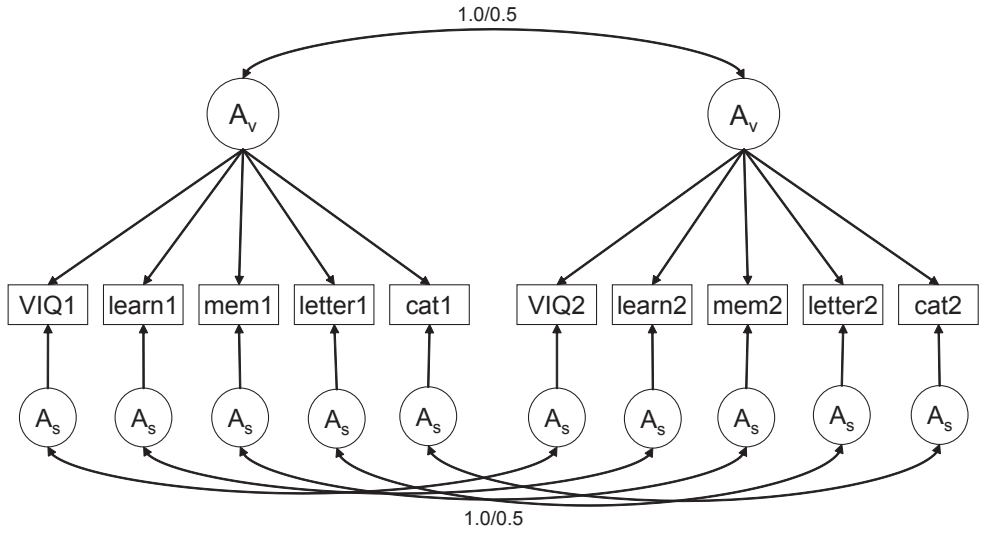

FIGURE 2. Path diagram depicting a common factor model including test specific influences. Path diagram shown for genetic effects, and for 2 family members only. This model can be expanded to include 3 family members, and can also be applied to shared environmental effects. Av = Common genetic factor exerting its influence on all verbal abilities. As = test specific genetic influences. Genetic effects correlate 1.0 between monozygotic twins, and on average 0.5 between dizygotic twins and twins and siblings. VIQ1/2 = verbal IQ family member 1/2; learn = verbal learning; mem = verbal memory; letter = letter fluency; cat = category fluency.

nested models, follows a χ^2 distribution. The degrees of freedom (df) are given by the difference in the number of parameters estimated in the two models. A high increase in χ^2 against a low gain of degrees of freedom denotes a worse fit of the submodel compared to the full model. The most parsimonious model, with still a limited χ^2, is chosen as the best fitting model. Akaike's information criterion (AIC=χ^2 – 2df) reflects the best balance between goodness of fit and parsimony with a model with the lowest AIC being the preferred model.

RESULTS

The descriptives of the five different verbal abilities in both the child and the adolescent cohort are given in Table 1. A positive age effect (i.e. better performance with increasing age) was found for all specific verbal measures. The age effect was not

TABLE 1. Sample sizes, means, standard deviations, and effects of age and sex for the verbal ability measures in 9-year-old twins and their siblings (child cohort), and in 18-year-old twins and their siblings (adolescent cohort).

	N	Mean	SD	Sex effect	Age effect
Child cohort					
VIQ	324	102.35	15.47	-0.06	N/A
verbal learning	323	38.40	7.44	2.95*	2.61*
verbal memory	323	7.96	2.45	0.86*	0.61*
letter fluency	324	-0.39	0.77	0.28*	0.26*
category fluency	324	-0.28	0.72	0.11	0.25*
Adolescent cohort					
VIQ	457	105.61	18.77	-6.94*	N/A
verbal learning	456	56.35	7.99	2.72*	0.27*
verbal memory	455	12.26	2.37	0.63*	0.13*
letter fluency	456	-0.096	0.87	0.12	0.09*
category fluency	457	-0.02	0.84	-0.03	0.09*

Note: * p<.01. A negative sex effect denotes superior male performance; a positive effect represents better performance in females.

TABLE 2. Phenotypic correlations between verbal ability measures in the child cohort (above diagonal) and the adolescent cohort (below diagonal).

Task	VIQ	verbal learning	verbal memory	letter fluency	category fluency
VIQ	-	.28	.22	.33	.44
verbal learning	.48	-	.68	.26	.24
verbal memory	.45	.74	-	.21	.18
letter fluency	.40	.28	.20	-	.26
category fluency	.39	.37	.31	.47	-

tested for VIQ, as this measure is standardised for age. Females outperformed males on verbal learning and memory in both cohorts. Girls performed better than boys on the letter fluency task in the child cohort, but this sex difference was not significant in the adolescent cohort. Males showed superior performance on the VIQ scale in the adolescent cohort. In the genetic model fitting the means were corrected for these effects.

Differences in variance and covariance due to zygosity or twin-sibling status were found to be absent in both cohorts. However, constraining the variance (χ^2=18.548, df=5, p=.002) and within person covariance (χ^2=33.257, df=10, p<.001) to be equal across the cohorts resulted in a significant deterioration of the fit. This indicates that the variance in the tests and the phenotypic covariance between the tests are significantly different in these two distinct phases of development. The phenotypic correlations between all measures are presented in Table 2, separately for the child (above diagonal) and the adolescent cohort (below diagonal). The phenotypic correlations are strongest between verbal learning and memory. Lower correlations are found between learning and memory and tests of verbal fluency. Overall, the phenotypic correlations are higher in the adolescent cohort (average r_{ph}=.41) than in the child cohort (average r_{ph}=.31).

Constraining the covariance matrices to be equal in DZ twins and in twin-siblings did not result in a deterioration of the fit of the saturated model, neither in the child cohort (χ^2=24.762, df=15, p=.053), nor in the adolescent cohort (χ^2=17.927, df=15, p=.267). This implies that the resemblance is similar in all first-degree relatives, and yields no evidence for a twin-specific environment. Table 3 displays the correlations in MZ twins (first figure on diagonal), and in DZ twins and twin-siblings (second

TABLE 3. Twin correlations and cross-correlations in MZ twins (below diagonal), and in DZ twins and twin-siblings (above diagonal) for five verbal ability tasks, in two cohorts.

Task	VIQ	verbal learning	verbal memory	letter fluency	category fluency
			Child cohort		
VIQ	.82/.47*	.18	.11	.22	.15
verbal learning	.24	.52/.18*	.19	.12	.07
verbal memory	.12	.41	.40/.22*	.14	.00
letter fluency	.34	.30	.15	.36/.23*	.05
category fluency	.36	.31	.11	-.03	.47/.01*
			Adolescent cohort		
VIQ	.84/.38*	.21	.20	.22	.18
verbal learning	.42	.34/.11*	.08	.11	.06
verbal memory	.39	.37	.47/.06*	.13	.05
letter fluency	.40	.28	.26	.51/.33*	.16
category fluency	.33	.21	.11	.37	.55/.19*

Note: *first figure correlation MZ twins, second figure correlation DZ twins and twin-siblings.

figure on diagonal). For all measures, MZ correlations are higher than DZ and twin-sibling correlations, indicating genetic influences. Moreover, apart from the correlations for verbal learning, the difference between the MZ twin and first-degree relative correlations is stronger in the older cohort than in the younger cohort. This suggests that the genetic influences on the variance in verbal abilities are stronger in late adolescence compared to middle childhood. In the child cohort, the MZ correlations for VIQ, verbal memory and letter fluency are not twice as high as the DZ and twin-sibling correlations, suggesting that shared environmental influences may also play a role. In both the child and the adolescent cohort, the MZ cross correlations (off-diagonal of Table 3) are higher than the DZ and twin-sibling cross correlations. This pattern suggests that the overlap between various measures of verbal abilities is

TABLE 4. Model fitting results for multivariate analyses of verbal abilities in the child and the adolescent cohort.

model		df	-2LL	cpm	χ^2	df	p	AIC
			Child cohort					
1.	ACE Cholesky	1559	7334.189					
2.	**AE Cholesky**	1574	7337.279	1	3.090	15	.999	-26.910
3.	CE Cholesky	1574	7371.181	1	36.992	15	.001	6.992
4.	AE A common factor + test specific	1579	7365.431	2	28.152	5	<.001	18.152
5.	AE E common factor + test specific	1579	7355.408	2	18.129	5	.003	10.129
6.	AE E test specific + correlated E between verbal learning and memory, and between verbal fluency tests	1582	7353.357	2	16.078	8	.041	.078
			Adolescent cohort					
1.	ACE Cholesky	2222	10600.738					
2.	**AE Cholesky**	2237	10603.722	1	2.984	15	.999	-27.016
3.	AE A common factor + test specific	2242	10620.318	2	16.596	5	.005	6.596
4.	AE E common factor + test specific	2242	10617.822	2	14.100	5	.015	4.100
5.	AE E test specific + correlated E between verbal learning and memory, and between verbal fluency tests	2245	10634.837	2	31.115	8	<.001	15.115

Note: df = degrees of freedom; -2LL = -2 log likelihood; cpm = compared to model; AIC = Akaike's Information Criterion; A = additive genetic influences; C = shared environmental influences; E = nonshared environmental influences.

influenced by genetic effects. In the child cohort, most MZ cross correlations are not twice as high as the DZ and twin-sibling cross correlations, suggesting that shared environmental influences may also explain part of the overlap between tests.

Table 4 gives the results of the model fitting for the Cholesky decomposition and the more parsimonious submodels. We started the model fitting procedure in the child cohort by testing the significance of the shared environmental influences (model 2 in Table 4) and the additive genetic influences (model 3) on the variance

TABLE 5. Contributions of additive genetic (A) and nonshared environmental (E) effects to the variance and covariance in verbal abilities, and the genetic correlations (r_g) between these abilities, in the child cohort. Estimates are based on the best fitting model (95% confidence interval in parentheses).

A

	VIQ	verbal learning	verbal memory	letter fluency	category fluency
VIQ	.82 (.72–.88)				
verbal learning	.94 (.61–1.00)	.46 (.30–.61)			
verbal memory	.59 (.00–.97)	.58 (.38–.76)	.42 (.25–.58)		
letter fluency	1.00 (.79–1.00)	.99 (.46–1.00)	.99 (.63–1.00)	.42 (.24–.58)	
category fluency	.71 (.46–.91)	.96 (.64–1.00)	.45 (.00–.97)	.19 (.00–.66)	.32 (.15–.51)

E

VIQ	.18 (.12–.28)				
verbal learning	.06 (.00–.39)	.54 (.39–.70)			
verbal memory	.41 (.03–1.00)	.42 (.24–.62)	.58 (.42–.75)		
letter fluency	.00 (.00–.21)	.01 (.00–.54)	.01 (.00–.37)	.58 (.42–.76)	
category fluency	.29 (.09–.54)	.04 (.00–.36)	.55 (.03–1.00)	.81 (.34–1.00)	.68 (.39–.85)

r_g

VIQ	-				
verbal learning	.40 (.19–.60)	-			
verbal memory	.20 (.00–.42)	.90 (.75–.99)	-		
letter fluency	.58 (.40–.79)	.51 (.20–.79)	.47 (.19–.76)	-	
category fluency	.64 (.41–.85)	.61 (.33–.85)	.24 (.00–.61)	.14 (.00–.54)	-

TABLE 6. Contributions of additive genetic (A) and nonshared environmental (E) effects to the variance and covariance in verbal abilities, and the genetic correlations (r_g) between these abilities, in the adolescent cohort. Estimates are based on the best fitting model (95% confidence interval in parentheses).

A

	VIQ	verbal learning	verbal memory	letter fluency	category fluency
VIQ	.84 (.77–.89)				
verbal learning	.92 (.76–1.00)	.28 (.15–.43)			
verbal memory	.90 (.72–1.00)	.37 (.21–.54)	.28 (.16–.44)		
letter fluency	.99 (.84–1.00)	.82 (.46–1.00)	.85 (.49–1.00)	.55 (.42–.66)	
category fluency	.88 (.69–1.00)	.41 (.09–.70)	.15 (.00–.50)	.70 (.47–.88)	.48 (.32–.62)

E

VIQ	.16 (.11–.23)				
verbal learning	.08 (.00–.24)	.72 (.57–.85)			
verbal memory	.10 (.00–.28)	.63 (.46–.79)	.72 (.56–.84)		
letter fluency	.01 (.00–.16)	.18 (.00–.54)	.15 (.00–.51)	.45 (.34–.58)	
category fluency	.12 (.00–.31)	.59 (.31–.91)	.85 (.50–1.00)	.30 (.11–.53)	.52 (.39–.68)

r_g

VIQ	-				
verbal learning	.90 (.70–1.00)	-			
verbal memory	.83 (.63–.98)	.95 (.78–.99)	-		
letter fluency	.60 (.46–.73)	.59 (.32–.85)	.47 (.22–.72)	-	
category fluency	.54 (.37–.70)	.42 (.10–.70)	.13 (.00–.44)	.65 (.47–.81)	-

and covariance in verbal abilities. Shared environmental effects were non-significant, dropping these effects from the model did not result in a deterioration of the model fit (χ^2=3.090, df=15, p=.999). The additive genetic influences were of significant importance (χ^2=36.992, df=15, p=.001). Subsequently it was tested whether the genetic effects on verbal abilities could be captured by a common factor model including test specific effects (model 4). Application of this model lead to a significant drop in model fit (χ^2=28.152, df=5, p<.001). Likewise, a model in which the nonshared environmental influences were constrained to a common factor including test specific effects (model 5) did not fit the data well (χ^2=18.129, df=5, p=.003). Lastly, a model with solely test specific influences of the nonshared environment, but permitting covariance between verbal learning and memory, and between letter fluency and category fluency (model 6) was fitted to the data of the child cohort. Application of this model resulted in a significant deterioration of the fit (χ^2=16.078, df=8, p=.041). All in all, the data of the child cohort were best described by a Cholesky decomposition including additive genetic and nonshared environmental effects (model 2). This model also had the lowest AIC-value, indicating that it showed the best balance between parsimony and model fit. Subsequently, the model fitting procedure was repeated for the adolescent cohort data. Similar to the results in the child cohort, the AE Cholesky model fitted the data best (χ^2=2.984, df=15, p=.999, AIC=-27.016).

The relative importance of additive genetic and nonshared environmental effects on the variance in each ability is given on the diagonal in Table 5 (child cohort) and Table 6 (adolescent cohort). In both age groups, the heritability was strongest for VIQ. Against expectation, the heritability of verbal learning and memory was stronger in the child compared to the adolescent cohort, although the confidence intervals overlap. The heritability estimates for verbal fluency were slightly higher in the adolescent cohort. The contributions of genes and environment on the covariance between the different verbal abilities are given on the subdiagonals of Table 5 and 6. In both cohorts, genetic effects account for most of the overlap between the tests, especially for the covariance between VIQ and the more specialised tests, and for the covariance between letter fluency and the other tests. In the child cohort, nonshared environmental effects are substantial for explaining the covariance between VIQ and memory, learning and memory, memory and category fluency, and between letter and category fluency. However the confidence intervals around these estimates are large. In the adolescent cohort, the nonshared environmental influences are substantial for the covariance between learning and memory, learning and category fluency, and between memory and category fluency. The genetic correlations between the different tests are given in the bottom of Table 5 and 6. On the whole, the genetic correlations are stronger in the adolescent cohort (average r_g over al tests=.61) compared to the child cohort (average r_g=.47). The genetic correlations in the adolescent cohort are

close to unity between VIQ, verbal learning, and verbal memory. The other genetic correlations are moderate, and range from .42 to .65, with one exception. The genetic correlation between memory and category fluency is estimated at .13, which is not significantly different from zero. In the child cohort, the genetic correlation is close to unity between verbal learning and memory. The other correlations are modest to moderate, ranging from .14 to .64.

Discussion

In this paper we report on the genetic and environmental influences on verbal abilities in two age cohorts. We studied the aetiology of the overlap in VIQ, verbal learning, verbal memory, and verbal fluency in two distinct phases of maturation: middle childhood and late adolescence. We found stronger correlations between the verbal tests in late adolescence, both at the phenotypic and the genetic level, suggesting progressing unidimensionality with age. We used an extended twin design and found that DZ twins did not resemble each other more closely than twin-sib pairs, yielding no indication for a twin-specific environment. In both cohorts, the individual differences in VIQ were strongly influenced by genetic effects, while the performance in more specific verbal abilities was under moderate genetic influence. The remaining variance was explained by nonshared environmental effects. Moreover, genetic effects were of major importance in explaining the overlap between the different verbal abilities, both in the child and the adolescent cohort. These results and their implications are discussed in more detail below.

Phenotypic correlations between verbal tasks in middle childhood and late adolescence

The within person variance and covariance on all tests was found to be significantly different in middle childhood than in late adolescence. Overall, the phenotypic correlations between the different verbal tests were stronger in adolescents, especially the correlations between VIQ and the more specific verbal measures (r ranging from .22 to .44 in the child cohort, and from .39 to .48 for the adolescent cohort). In a longitudinal cognition study, we found increasing phenotypic correlations between verbal and nonverbal abilities with age (Hoekstra et al., 2007). Together with the current data, these results suggest that cognitive abilities may be more generalised in a later stage of development. However, the adolescent cohort and child cohort participated in a somewhat different study protocol, and verbal learning and memory were assessed using slightly different tests (the AVLT in the child cohort vs. the CVLT in the adolescent cohort). Although previous studies indicated strong overlap between

AVLT and CVLT performance (Mulder et al., 1996; Stallings et al., 1995), with correlations close to the test-retest correlations of the CVLT itself (Mulder et al., 1996), we cannot exclude the possibility that these differences have affected the pattern of phenotypic correlations.

In both cohorts, the phenotypic correlations were strongest between verbal learning and memory. This is not surprising, as successful memory depends on successful learning, and both measures involve registration, storage, and retrieval of words. The phenotypic correlations between letter fluency and category fluency were relatively low (r=.26 in the child cohort and .47 in the adolescent cohort), indicating that these tasks tap different aspects of word fluency. Letter fluency requires phoneme analysis, while category fluency relies more heavily on semantic memory. Category fluency performance is shown to be superior in children who frequently use schemata to guide their recall (Sincoff & Sternberg, 1988). For instance, the subjects in our study could improve their performance on the "animal" trial of category fluency by thinking of all the animals that live in a zoo or on a farm. It is also possible to follow strategies in a letter fluency task (e.g. name words starting with the same consonants, such as *rep*tile and *rep*lication), but these strategies are not as obvious, and less often used.

Heritability of general vs. specific verbal abilities

Individual differences in general verbal abilities, as measured with the Wechsler VIQ, were found to be highly heritable, both in middle childhood and in late adolescence. The heritability estimate of 84% found in our sample of 18-year-old twins and their siblings is similar to heritability estimates of VIQ in other adult samples, that reported a heritability of 84 % (Rijsdijk et al., 2002) and 85% (Posthuma et al., 2001). The heritability estimate of 82% for VIQ in 9-year-old twins and their siblings is somewhat higher than the estimates reported in other studies in middle childhood (e.g. Hoekstra et al., 2007). However, the MZ and first degree relative correlations found in the child cohort (respectively r=.82 and r=.47) are very similar to the MZ and DZ twin correlations (rMZ=.82; rDZ=.42) found in our previous study in 10-year-old twins (Hoekstra et al., 2007). The latter study incorporated a longitudinal design, and found evidence for shared environmental influences in early and middle childhood. Therefore, the different heritability estimates in these two studies are most likely due to the different designs used.

The genetic influences on the variance in verbal learning and memory were moderate in both cohorts. Against expectation, the point estimate of the genetic effects was higher in middle childhood (46% and 42% respectively) than in late adolescence (both 28%), although the confidence intervals overlap. The attenuated genetic effects in the adolescent cohort compared to the child cohort are most likely explained

by differences in the tests used to measure these abilities. In the child cohort, verbal learning and memory was assessed with the AVLT, in which a list of unrelated words is used. In the adolescent cohort, learning and memory performance was determined with the CVLT, including a list of words belonging to different categories. One previous twin study examined the heritability of uncategorised word learning versus categorised word learning (Volk et al., 2006), and found stronger genetic effects on uncategorised (55%) than on categorised learning (38%). This difference in heritability could explain why the heritability estimates for learning and memory were higher in the child cohort compared to the adolescent cohort. Genetic effects on letter and category fluency were moderate in the child cohort (42% and 32% respectively). Although confidence intervals overlap, the point estimates for the genetic effects on both measures were slightly higher in the adolescent cohort (55% and 48%), consistent with the increasing genetic influences on verbal abilities found in previous studies (Alarcón et al., 1998; Alarcón et al., 1999; Alarcón et al., 2003; Hoekstra et al., 2007).

The remaining variance in all tasks was explained by nonshared environmental effects. Shared environmental influences failed to be significant, both in the child and the adolescent cohort. The lack of shared environmental influences on individual differences in adult verbal abilities is in accordance with findings from previous studies (Ando et al., 2001; Posthuma et al., 2001; Rijsdijk et al., 2002; Swan et al., 1999; Swan & Carmelli, 2002; Volk et al., 2006). An earlier study on the heritability of Wechsler VIQ in middle childhood reported modest shared environmental influences (Hoekstra et al., 2007). Studies in early to middle childhood on verbal fluency and verbal memory reported modest (Samuelsson et al., 2005) or non-significant effects of the shared environment (Bishop et al., 2006; Kovas et al., 2005; Thompson et al., 1991). The results of the current study do not provide evidence for a strong influence of the shared environment on verbal abilities in middle childhood.

Genetic and environmental covariation between different verbal tests

Genetic influences appeared to be the driving force behind the covariation between verbal abilities. The genetic effects on the overlap between VIQ and the more specialised verbal tasks were not significantly different from unity in the adolescent cohort, and approached unity in the child cohort (although some of the confidence intervals varied widely). The genetic effects on the overlap between verbal learning, memory, and fluency was modest to strong, and especially strong between letter fluency and verbal learning and memory in both cohorts. The finding of strong genetic influences on the covariance between verbal tests is in line with earlier studies, suggesting that overlap between tests is mainly accounted for by genetic effects, while nonshared environmental effects induce differences in test performance (Deary et

al., 2006; Petrill, 1997; Plomin & Spinath, 2002). However, fitting a model in which the nonshared environmental effects were constrained to be test-specific did not fit the data, neither in the child, nor in the adolescent cohort. This finding implies that these environmental effects, albeit of moderate impact, were also of importance in explaining the overlap between verbal abilities. Possible nonshared environmental effects on verbal abilities could include traumatic experiences unshared with the other family members, or consequences of an accident or illness. Also, if the children are in separate classes, the influences of the teacher or other school-related influences will be nonshared. Furthermore, subject specific influences, such as weariness on the day of testing, may account for the covariance between tests.

Verbal IQ, verbal learning and verbal memory appear to be largely influenced by the same set of genes in late adolescence. The genetic correlations between these measures were close to unity. The genetic correlation between verbal learning and verbal memory in the child cohort was also very high, but its associations with VIQ were somewhat lower. On the whole, the genetic correlations between the different verbal tasks were higher in the adolescent cohort (average r_g=.61) compared to the child cohort (average r_g=.47). This finding is in line with previous studies into specific cognitive abilities, that found a genetic correlation between verbal and nonverbal abilities of about .30 in infancy (Price et al., 2000), increasing genetic correlations between these abilities from early childhood to young adulthood (Hoekstra et al., 2007), up to a genetic correlation of around .70 in adulthood (Hoekstra et al., 2007; Posthuma et al., 2001). The steady increase in genetic correlations between middle childhood and late adolescence in our study suggests a progressive unidimensionality underlying verbal abilities at the genetic level. The high genetic correlations between cognitive domains suggest the existence of "generalist genes": genes that exert a general effect within and between cognitive abilities (Kovas & Plomin, 2006; Plomin & Kovas, 2005). However, a genetic common factor model could not be fit to our data without a significant reduction of the model fit, neither in the child nor in the adolescent cohort, indicating that verbal abilities are not entirely unidimensional. This is also supported by the notion the genetic correlations between verbal learning, memory, and letter and category fluency are still significantly different from one, even in the sample of 18-year-old twins and their siblings.

Implications for future studies

The results from this study are also relevant to research in psychopathology. Several clinical studies have reported impaired performance on verbal learning and memory as measured with the CVLT or the AVLT in patients suffering from schizophrenia (Appels et al., 2003; Egan et al., 2001; Simon et al., 2007; Weickert et al., 2000), or their relatives (Appels et al., 2003; Egan et al., 2001; Snitz et al., 2006; Szoke et al., 2005). Moreover, some studies reported impaired performance on letter or category verbal fluency in schizophrenia patients and their relatives (Appels et al., 2003; Chen et al., 2000; Snitz et al., 2006; Szoke et al., 2005) and in (relatives of) children diagnosed with an autism spectrum disorder (Geurts et al., 2004; Hughes et al., 1999). Following this, measures of verbal learning, memory, and fluency have been proposed as promising endophenotypes for psychiatric illness. One of the criteria for a good endophenotype is that the endophenotype itself should be under substantial genetic control (De Geus & Boomsma, 2001; Viding & Blakemore, 2007). The current study provides a direct test of this criterion. The results of our study indicate that individual differences in verbal learning and memory, as measured with the AVLT, are more strongly genetically determined than verbal learning and memory as measured with the CVLT. Therefore, we suggest the use of the AVLT if researchers plan to use verbal learning and memory as an endophenotype. Both letter fluency and category fluency are under moderate genetic influence, and could therefore both serve as useful endophenotypes. However, it is important to note that the genetic correlation between these measures is relatively low (and not significantly different from zero in the child cohort). Researchers should probably avoid including a composite score of "overall verbal fluency" as an endophenotype, as these measures reflect genetically different cognitive constructs. Lastly, VIQ was under stronger genetic influence than the more specialised verbal abilities in both age groups. The Wechsler VIQ scale comprises an extensive and well validated test battery. Researchers interested in the genetic effects on general verbal abilities should be aware that these abilities are not simply captured by a quick and easy to administer test such as verbal fluency.

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