

# The phenotypic and genotypic relation between working memory speed and capacity

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## Abstract

This study examined the phenotypic and genotypic relationship between working memory speed (WMS) and working memory capacity (WMC) in 12-year-old twins and their siblings ( $N=409$ ). To assess WMS all children performed a reaction time task with three memory loads from which a basic mental speed measure and the derived slope were used. WMC was measured with two subtests of the WISC-R, namely Arithmetic and Digit Span. The phenotypic correlations among the WMS and WMC indices were around  $-0.30$ . Heritabilities for all variables ranged from 43% to 56%. Structural equating modelling revealed that a model with two genetic factors, representing WMS and WMC, which were correlated ( $-0.54$ ) fitted the data best, indicating that WMS and WMC are partly mediated by the same set of genes and partly by separate sets of genes. When general IQ was simultaneously analysed with the data the correlation between the genetic factors for WMS and WMC decreased ( $-0.25$ ), but was still significant. This means that  $\sim 50\%$  of the genetic correlation between WMS and WMC is explained by IQ.

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## 1. Introduction

Working memory is conceptualised as a limited capacity system for information processing. It plays an important role in all forms of cognition and is essential in normal daily functioning, for example when reading

the paper or watching a football game. It is now widely accepted that WM is not a unitary system, but that it can be divided into subsystems. An influential model was proposed by Baddeley who presented a theoretical WM framework with three distinguishable subcomponents (Baddeley & Hitch, 1974; Baddeley, 1992). First, the visuospatial sketch pad that manipulates visual images. Second, the phonological loop that stores and rehearses acoustic information. And third, the central executive, which is an attentional controlling system that coordinates and processes the information of the two other components. Baddeley (2000) extended this model with

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the episodic buffer. The episodic buffer represents a limited capacity system, controlled by the central executive that is capable of integrating information from various sources into an episodic representation.

Several other authors proposed to partition WM in different components. Miyake and Shah (1999) described working memory as a non-unitary system of processes and mechanisms that allows task-relevant information to be stored temporary in an active state, for further processing or recall. In a similar vein Cowan et al. (2005) stated that WM is a set of mental processes holding limited information in a temporary accessible state in service of cognition. Oberauer, Süß, Wilhelm, and Wittman (2003) defined WM as a set of limited factors for performance in complex cognitive tasks, organized as a hierarchy of related constructs. Partition of WM in a neuro-anatomical way was for example suggested by Owen (2000) who proposed a process-specific distinction between maintenance and active manipulation of information in WM, which is supported by ventral and dorsal prefrontal cortical regions, respectively. Smith and Jonides (1999) reviewed neuro-imaging studies of the storage and executive components of WM. They concluded that the storage component of WM is activated by different frontal regions like Broca's area and premotor areas while the executive component involves the anterior cingulate and dorsolateral prefrontal cortex.

It is hypothesized that  $g$  (with  $g$  being the operational definition of 'general intelligence') is largely responsible for better performance on various tasks in which speed and accuracy are involved (Gray & Thompson, 2004). A large number of studies explored the relationship between WM and  $g$  (for an overview see Buehner, Krumm, & Pick, 2005). High correlations between WM and reasoning were found in early studies by Kyllonen and Christal (1990), and recently Colom, Rebollo, Palacios, Juan-Espinosa, and Kyllonen (2004) found that WM was almost perfectly predicted by  $g$ . Conway, Cowan, Bunting, Theriault, and Minkoff (2002) found that among processing speed, short-term memory capacity and WMC the latter was the best predictor for general fluid intelligence. Other studies could not replicate these very strong relationships (for example Ackerman, Beier, & Boyle, 2005; Conway, Kane, & Engle, 2003) but a general finding is that WM and  $g$  are positively and significantly related. Which specific components of WM play a role in this relation, and how strong these relations are, remains unclear. A small number of adult twin studies addressed the question whether a genetic approach could be used to clarify genetic components underlying WM per se, and

of the relationship between WM and intelligence. Ando, Ono, and Wright (2001) studied a twin sample of young adults to investigate the genetic structure of storage and executive functions in the spatial and verbal working memory domain. They also examined the relation between the WM tasks and cognitive ability which was measured with a Japanese intelligence test (Kyodai NX 15, Osaka & Umemoto, 1973). It was found that the phenotypic variances on the spatial and verbal task were significantly due to genetic influences, with heritability estimates between 43% and 48%. The genetic variance was due to modality specific factors (spatial and verbal) and a storage specific factor (7–30%). However, another part of the genetic variance was due to a common genetic factor explaining storage and executive functions in both spatial and verbal functions (11–43%). These findings suggested that multiple, partly overlapping genetic factors influence spatial and verbal working memory. The authors hypothesized that besides the important function of the prefrontal lobes in working memory, modality specific regions of the brain, such as Wernicke's regions (verbal) and the right parietal lobe (spatial) are involved, and that these regions are mediated by separate genetic influences. When they included cognitive ability in the analyses, it was shown that the common genetic factor found for the WM tasks, also explained a substantial part of the phenotypic correlation between the WM tasks and cognition.

Similar findings were presented by Luciano et al. (2001) who measured processing speed, working memory and IQ in 166 monozygotic and 190 dizygotic twin pairs. Subjects were young adults with a mean age of 16.17 (S.D.=0.34). Processing speed was measured by a choice reaction task, and working memory was measured by a visual spatial delayed response task. IQ was derived from the Multidimensional Aptitude Battery (MAB, Jackson, 1998). Analysis showed the presence of a common genetic factor influencing all variables. In addition there were specific genetic factors influencing processing speed, working memory and IQ. Based on their findings the authors speculated that the genes common to all variables might actually affect the central executive component of WM, whereas those genes specific to the WM task, relate to the storage component.

Neubauer, Spinath, Riemann, Angleitner, and Borkenau (2000) pointed out that a distinction should be made between WM capacity and WM speed. In a large sample of adult twins they focused on speed of information processing. The relationship between psychometric IQ and two measures of speed of information processing was investigated. Psychometric

intelligence was measured with shortened forms of the Raven's Advanced Progressive Matrices (APM, [Raven, 1958](#)) and the Leistung-Prüf-System (LPS, [Horn, 1962](#)), which is a well known German intelligence test. Processing speed was measured with two Elementary Cognitive Tasks. The first task was a memory scanning test based on [Sternberg's \(1969\)](#) Short Term Memory paradigm. In this test subjects have to randomly store one, three or five digits. After a warning signal, a target digit is shown and subjects have to indicate as quickly as possible if the target digit was part of the previously shown memory set. The second task was a Posner's letter-matching test ([Posner & Mitchell, 1967](#)). In this test subjects have to judge physical identity (i.e., visual discrimination) or name identity (i.e., LTM retrieval) of two characters. The phenotypic correlations between the RTs on the Elementary Cognitive Tasks and IQ were about  $-0.40$  and were largely due to genetic factors. However, there were also specific genes affecting both phenotypes. The phenotypic correlations between the derived slope (i.e., linear increasing RT with increasing memory load) of the memory scanning task and IQ were relatively low ( $0.00$ – $0.12$ ). In discussing their results [Neubauer et al. \(2000\)](#) suggested that future studies should include both mental speed and WM capacity to see if this joint contribution yields higher (genetic) correlations with human intelligence.

The present study investigates the genetic covariance between WM speed (WMS) and WM capacity (WMC) in children. In addition the influence of general IQ ( $g$ ) on this genetic covariance is examined. Twelve-year-old twin pairs and their siblings ( $N=409$ ) performed a choice reaction task with three memory loads from which a basic mental speed measure and the derived slope, as a reflection of delay caused by higher memory load, were used. Two subtests of the Wechsler Intelligence Scale for Children Revised (WISC-R, [Van Haasen et al., 1986](#)) that index capacity components of WM, namely Arithmetic and Digit Span ([Engle, 2002](#); [Kaufman, 1975](#)) were analysed. General IQ was estimated by two verbal (Vocabulary and Similarities) and two performance (Block Design and Object Assembly) subtests of the WISC-R ([Sattler, 1982, 1992](#)).

The first aim is to assess the heritability of WMS and WMC and to examine to what extent individual differences in WMS and WMC performance are due to genetic variation. The second aim is to investigate whether covariance between WMS and WMC is explained by pleiotropic genetic effects. We explore through genetic factor analyses if a common set of genes influences both the WMS and WMC component.

Structural Equating Modelling was used to test whether the genetic influences which are important for WMS are correlated with the genetic influences underlying WMC. This is established by modelling two genetic factors, one for WMS and one for WMC, which are allowed to correlate. If this correlation is one, this means that WMS and WMC are influenced by a common genetic factor (i.e., completely overlapping sets of genes). If this correlation is zero, the two components are influenced by independent sets of genes. If the correlation has a value between zero and (minus) one, WMS and WMC are partly mediated by the same set of genes and partly by separate sets of genes.

The third aim is to investigate whether  $g$  plays a role in the genetic covariance between WMS and WMC. Therefore the original model with two correlated genetic factors for WMS and WMC is extended to a hierarchical factor model in which genetic influences on  $g$  are modelled as a latent genetic variable influencing the genetic covariance between WMS and WMC. If, after incorporating  $g$  in the model, the genetic correlation between WMS and WMC disappears,  $g$  explains the genetic covariance. If the correlation does not change significantly from the correlation in the original model, WM itself explains the genetic covariance between WMS and WMC. If the correlation reduces but is significantly different from zero, both  $g$  and WM explain the genetic correlation between WMS and WMC.

## 2. Methods

### 2.1. Subjects

The sample consisted of 177 Dutch twin pairs, born between 1990 and 1992, and 55 of their siblings. The twins were 12 years old (mean age = 12.42, S.D. = 0.16) and the siblings were between 8 and 15 years old. Twenty-seven siblings were younger than their twin brothers or sisters (mean age = 9.60, S.D. = 0.71) and 28 siblings were older (mean age = 14.69, S.D. = 0.60). There were 41 monozygotic male twin pairs (MZM), 28 dizygotic male twin pairs (DZM), 56 monozygotic female twin pairs (MZF), 25 dizygotic female twin pairs (DZF) and 27 dizygotic opposite-sex twin pairs (DOS). Zygosity was determined on the basis of DNA polymorphisms. The twins were registered at birth with the Netherlands Twin Registry ([Boomsma, 1998](#); [Boomsma et al., 2002](#)). None of the children suffered from severe physical or mental handicaps. There were 172 twin pairs who had participated in a similar study at the age of 5 ([Groot, De Sonneville, Stins, & Boomsma,](#)

2004; Stins et al., 2005). The selection at that time was based on age and a sample evenly distributed across zygosity groups. To gain power for the current analyses five extra, dizygotic female twin pairs and 55 siblings of the twins were recruited (Posthuma & Boomsma, 2000).

The parents were invited for participation of their children in the continuing study entitled 'Genetics of Attention'. In the mailing information about the goals and procedures of the study were included. After 2 weeks the parents were contacted by phone and asked if they were willing to participate. Prior to the assessment parents and children signed an informed consent form.

## 2.2. Procedure

Assessments always started before 11 a.m. Children were tested at the same time but in separate rooms by separate experimenters. All subjects performed the same neuropsychological test battery consisting of 6 subtests of the Wechsler Intelligent Scale for Children Revised (WISC-R, Van Haasen et al., 1986) and computerized reaction time tasks, measuring a diverse range of executive functions such as working memory, divided, sustained, selective and focused attention. The entire test battery took ~4 h, including breaks. After finishing the assessment, each child received a small present.

WMS was assessed with 'Memory Search' which is one of the tasks of the Amsterdam Neuropsychological Tasks (ANT, De Sonneville, 1999). In this task memory load, operationalized as target set size, increases from one to three target letters. The computer screen shows a fixed display of four consonants arranged in a square from which subjects must detect one or more target letters. For Load I the target signal requiring a yes-response is 'k' (40 trials; 50% target signal). For Load II, target signals are 'k'+ 'r' (72 trials; 36 complete target sets, 18 trials one target signal, 18 trials no target signals) and for Load III target signals are 'k'+ 'r'+ 's' (96 trials; 48 complete target sets, 16 trials one target signal, 16 trials two target signals, 16 trials no target signals). Children were instructed to press the yes button only when a complete set of target letters was present. In all other instances a no-response was required. An example of the stimuli is shown in the bottom part of Fig. 1. Responses were made by pressing the left or right mouse button. A yes-response was made with the preferred hand, a no-response with the unpreferred hand. In the instruction, both speed and accuracy were emphasized. Twelve practice trials were provided to ensure instructions were well understood.

WMC was assessed with two subtests of the WISC-R. Factor analyses exploring the structure of the WISC-

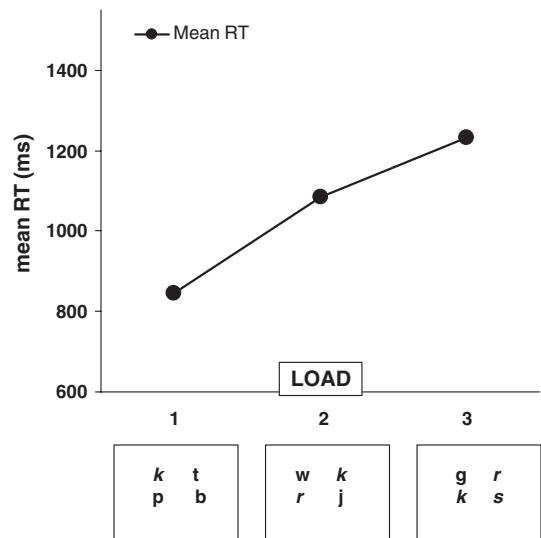


Fig. 1. Pattern of mean RTs over correct responses, including examples of stimuli (requiring a yes-response) of Load I, Load II and Load III of the WMS task.

R showed that a three factor solution fitted the data best (Kaufman, 1975, 1979; Kroonenberg and Berge, 1987; Reynolds and Kaufman, 1985). One of these factors is Working Memory and the accompanying tasks are Arithmetic, Digit Span and Substitution from which the first two tests were assessed. For general IQ (g) 4 subtests of the WISC-R were used, namely Similarities, Vocabulary, Block Design and Object Assembly. Standardized scores of this short form of the WISC correlates 0.94 with standardized IQ scores based on all subtests of the WISC-R (Sattler, 1982, 1992).

## 3. Analyses

### 3.1. Descriptives

Only correct WMS responses were used for the analyses. None of the subjects had more than 30% misses or false alarms. The results of children who had a mean reaction time (RT) that was higher than three times the standard deviation above mean RT of the sample ( $N=8$ ) and children with a negative slope (i.e., children who had a lower mean RT for Load 3 than for Load 1,  $N=3$ ) were excluded. Data of seven children were not recorded. The increase in RT across the loads (i.e., the Slope) was computed as  $(RT \text{ Load III} - RT \text{ Load I})/2$ . ANOVA (SPSS, 11.5) was used to test whether there was a significant increase in RT with increasing memory load. To summarize the WMS data the variables Load I, as a basic mental speed measure, and Slope, as a measure of delay caused by higher load, were used for further



analyses. WMC was measured as the number of correct responses on Arithmetic, and on Digit Span. Data of 3 children were missing. Standardized IQ scores of Similarities, Vocabulary, Block Design and Object Assembly were used to estimate general IQ ( $g$ ). Table 1 gives an overview of total numbers of subjects and total number of complete twin pairs, and twin-sib pairs for each variable.

### 3.2. Univariate genetic analyses

The different degree of genetic relatedness between monozygotic (MZ) twins, dizygotic (DZ) twins, and non-twin siblings (i.e., MZ twins share all their genes while DZ twins and siblings share on average half of their genes) was used to estimate the genetic and environmental contributions to the (co)variance of the variables. The total variation of each variable can be decomposed into sources of additive genetic variance (A), common environmental variance (C) and unique environmental variance (E). A is due to additive effects of different alleles, C is due to environmental influences shared by members of a family, and E is due to environmental influences not shared by members of a family. E also includes measurement error and is therefore always included in the models. A first impression of the relative importance of each component is obtained by inspecting the standardized covariances, which are the twin correlations and twin-sib correlations. MZ correlations twice as high as DZ (and twin-sib) correlations indicate additive genetic influences. DZ correlations higher than half the MZ correlations designate common environmental influences. MZ correlations as high as DZ correlations indicate only common and unique environmental influences and no genetic sources of variance (Boomsma, Busjahn, & Peltonen, 2002).

The proportion of phenotypic variance due to genetic influences is known as the heritability ( $h^2$ ). As power analyses revealed that the power to detect sex differences in heritability was low, male and female data were combined for both zygositys (see Appendix A).

Table 1  
Total numbers of first-born twins, second-born twins, and siblings, and total number of complete twin pairs for each variable

<i>N</i>	Load I	Load II	Load III	Slope	Arithmetic	Digit span	IQ
First-born twins	172	170	170	168	175	175	176
Second-born twins	175	175	173	171	175	174	177
Siblings	53	52	52	52	53	53	52
Total <i>N</i>	400	397	395	391	403	402	405
Complete twin pairs	171	170	167	166	175	174	176

Structural equating modelling, as implemented in the statistical software package Mx (Neale, Boker, Xie, & Maes, 2003), was used to analyse the data. Mx provides parameter estimates by maximizing the raw data likelihood. The goodness of fit of different models is evaluated by hierarchic likelihood ratio ( $\chi^2$ ) tests. Specifically, the  $\chi^2$  statistic is computed by taking twice the difference between the log-likelihood of the full model and the log-likelihood of a reduced model ( $\chi^2 = -2LL0 - (-2LL1)$ ). The associated degrees of freedom are computed as the difference in degrees of freedom between the two hierarchic models (Neale & Cardon, 1992). In addition to the  $\chi^2$ -statistic, Aikake's Information Criterion (AIC) can be computed ( $AIC = \chi^2 - (2 \cdot df)$ ). A low AIC indicates a relative good fit of the model. In a so-called saturated model means and standard deviations and phenotypic twin and twin-sib correlations were estimated. A saturated model is fully parameterized and yields the best possible fit to the data. It is a useful model for evaluating the fit of more restricted models. It was tested whether means and variances of each variable were equal for first-born and second-born members of a twin pair, for MZ and DZ twins, and for siblings. In addition, it was tested whether DZ correlations and twin-sib correlations were equal for all variables. Full ACE models were fitted to the data of each variable to see if the phenotypic twin and twin-sib correlations derived from the saturated models were attributable to A, C or E. In addition, more parsimonious models (i.e., AE, CE and E models) were compared to the ACE model.

### 3.3. Multivariate analyses

First, an unconstrained decomposition of the covariance structure of WMS and WMC into genetic and environmental covariance matrices was considered by means of triangular (or Cholesky) decomposition, including three variance components A, C and E. Based on the estimates of the A, C and E covariance matrices the genetic and environmental correlations between the variables were computed. The genetic correlations provide a measure of the extent to which variables are influenced by the same genes. The environmental correlations reflect the extent to which variables are influenced by the same environmental processes. The most parsimonious Cholesky model (i.e., an ACE, AE, CE or E model) was used as a baseline model against which to compare the hypothesized factor model for WMS and WMC.

In the factor model the genetic (A) and environmental components (C and E) were modelled with two latent factors, one for WMS and one for WMC. This was done

by deleting four pathways in the original Cholesky model in such a way that the latent WMS factor loaded on the WMS variables and the latent WMC factor loaded on the WMC variables. To examine whether all the variance could be explained by the latent factors, it was tested whether genetic effects specific for each variable could be deleted from the model without worsening the fit (i.e., all variance is explained by the latent factors). To examine whether the two factor structure fitted to the data it was tested if it was allowed to delete the latent factors for A, C or E (i.e., all variance is explained by specific factors). If the two latent factors for A, C, or E could not be deleted from the model they were allowed to correlate in three different sub models. In the first sub model the correlation between the two latent factors for WMS and WMC was estimated freely. This represented a model with partly independent and partly overlapping factors. In the second sub model the correlation between the two factors was constrained to be 1, reflecting a model with one factor for all variables. In the third sub model the correlation between the two factors was constrained to be zero, indicating two uncorrelated separate factors.

To investigate whether the genetic covariance between WMS and WMC could (partly) be explained by *g*, the factor model was extended with a third latent

genetic factor *Ag* which loaded on general IQ. This factor *Ag* was modelled as a higher order factor controlling the genetic correlation between WMS and WMC (see Fig. 2). It was tested to what extent the correlation between the latent WMS and WMC factors changed in this hierarchical model, compared to the original factor model. This was done in three ways. First, the correlation between WMS and WMC was estimated freely in the hierarchical model. Second, the estimated correlation from the original model was fixed in the hierarchical model to test whether the original correlation changed significantly. If not, the genetic correlation between WMS and WMC would be explained solely by *WM*. Third, the correlation was fixed to zero to test whether *Ag* could explain all the covariance between WMS and WMC.

## 4. Results

### 4.1. Descriptives

For WMS, RTs were highest in Load III, lower in Load II and lowest in Load I. These load effects were significant for MZ and DZ twins, and siblings ( $p < 0.001$ ). Fig. 1 shows the pattern of mean RTs of the three memory loads in the entire sample.

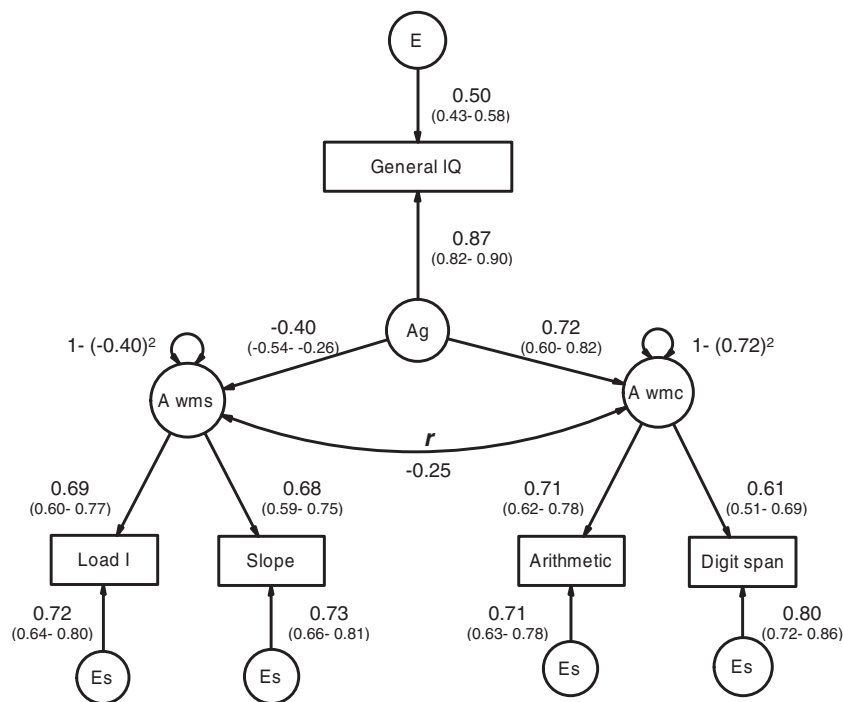


Fig. 2. Factor loadings of the best fitting hierarchical model with three latent factors for additive genetic influences (A-WMS, A-WMC and *Ag*), for the WM variables specific factors for *E*, and for the IQ variable a factor for *E*. Standardized path loadings are shown with confidence intervals in brackets. The correlation between the two latent factors A-WMS and A-WMC is represented by *r*.

Table 2

Upper part: means and standard deviations for each variable, with the deviation from the mean for boys, older siblings and younger siblings

	RT Load I	RT slope	Arithmetic	Digit span
Mean (deviation: boy/older sib/younger sib)	811.49 (24.49/–72.95/253.3)	351.08 (23.89/–81.83/288.69)	16.88 (0.92/0.89/–3.75)	11.38 (0.74/1.64/–2.14)
S.D.	144.72	203.20	2.94	2.84
Phenotypic correlations				
MZ	0.53 (0.37–0.66)	0.40 (0.22–0.55)	0.59 (0.45–0.69)	0.58 (0.43–0.68)
DZ/twin-sib	0.23 (0.08–0.37)	0.28 (0.11–0.42)	0.11 (–0.04–0.27)	0.24 (0.08–0.39)

Lower part: phenotypic correlations for MZ and DZ/twin-sibling pairs.

#### 4.2. Univariate genetic modelling

Table 2 shows means and standard deviations including the effects of sex and age on the observed data, and phenotypic twin and twin-sib correlations for Load I, Slope, Arithmetic and Digit Span. Means and variances were equal for twins and siblings, and DZ correlations and twin-sib correlations were equal for all variables.

Compared to the saturated models, univariate full ACE models did not worsen the fit significantly. Evaluating more restricted models against the full ACE models showed that for Load I, Arithmetic and Digit Span C could be dropped from the full model. For Slope it was allowed to drop either A or C from the full model but not both. This indicated that the variance was explained by familial influences; however, it was not possible to distinguish between genetic or common environmental influences. A and E contributed equally to the total variance with heritabilities ranging between 43% and 56%. Table 3 shows univariate model fitting results for full ACE

models and more restricted models per variable, including parameter estimates.

#### 4.3. Multivariate genetic modelling

Multivariate analyses revealed that the most parsimonious Cholesky model, which was used as a baseline model, included an additive genetic component (A) and a unique environmental component (E). Common environmental influences (C) could be dropped from the full Cholesky model without significantly worsening the fit, indicating that common environmental influences played no important role in the covariance between WMS and WMC. Hence, C was not included in the factor analyses.

Table 4 shows phenotypic, genetic and environmental correlations between all variables. Phenotypic correlations between WMS variables (Load I and Slope) and WMC variables (Arithmetic and Digit Span) were 0.50 and 0.45 respectively, and between WMS and WMC variables –0.30 (Load I and Digit Span), –0.32 (Slope and Digit Span), –0.33 (Load I and

Table 3

Univariate model fitting results

	Model	$\Delta\chi^2$	$\Delta df$	$p$	AIC	$h^2$	$c^2$	$e^2$
<b>Load I</b>	ACE	22.86	14	0.06		0.51 (.20–.63)	0.00 (.00–.23)	0.49 (.37–.65)
	<b>AE</b>	<b>0.00</b>	<b>1</b>	–	<b>–2.00</b>	<b>0.51 (.36–.63)</b>		<b>0.49 (.37–.64)</b>
	CE	8.65	1	0.00	6.65		0.32 (.20–.43)	0.68 (.56–.80)
	E	34.51	2	0.00	30.51			
<b>Slope</b>	ACE	23.81	17	0.12		0.26 (.00–.55)	0.14 (.00–.42)	0.60 (.45–.78)
	<b>AE</b>	<b>0.72</b>	<b>1</b>	<b>0.40</b>	<b>–1.28</b>	<b>0.43 (.27–.56)</b>		<b>0.57 (.44–.73)</b>
	CE	1.54	1	0.21	–0.46		0.33 (.19–.45)	0.67 (.55–.81)
	E	25.24	2	0.00	21.24			
<b>Arithmetic</b>	ACE	15.19	17	0.58		0.54 (.36–.67)	0.00 (.00–.12)	0.46 (.33–.61)
	<b>AE</b>	<b>0.00</b>	<b>1</b>	–	<b>–2.00</b>	<b>0.54 (.39–.67)</b>		<b>0.46 (.33–.61)</b>
	CE	15.97	1	0.00	13.97		0.29 (.17–.41)	0.71 (.59–.83)
	E	39.14	2	0.00	35.14			
<b>Digit Span</b>	ACE	27	17	0.06		0.56 (.28–.68)	0.00 (.00–.22)	0.44 (.32–.58)
	<b>AE</b>	<b>0.00</b>	<b>1</b>	–	<b>–2.00</b>	<b>0.56 (.42–.68)</b>		<b>0.44 (.32–.58)</b>
	CE	11.21	1	0.00	9.21		0.38 (.25–.48)	0.63 (.52–.75)
	E	46.62	2	0.00	42.62			

Submodels AE, CE and E are compared with the full ACE model, which in turn is compared with the saturated model. Confidence intervals of the parameter estimates are put in brackets. Bold indicates the best fitting model.

Table 4

Phenotypic, genetic and unique environmental correlations among measures of WMS, WMC, and IQ

	Slope	Arithmetic	Digit span	IQ
Load 1	0.50/0.99/ –0.11	–0.33/–0.49/ –0.06	–0.30/–0.46/ 0.00	–0.21/–0.36/ –0.06
Slope		–0.26/–0.57/ 0.15	–0.32/–0.51/ –0.05	–0.25/–0.42/ –0.10
Arithmetic			0.45/0.73/ 0.04	0.47/0.75/ 0.03
Digit span				0.33/0.48/ 0.03

Arithmetic) and –0.26 (Slope and Arithmetic). Phenotypic correlations with IQ were –0.21 (Load I), –0.25 (Slope), 0.47 (Arithmetic) and 0.33 (Digit Span). Lower mean RTs of WMS were, as expected, negatively correlated with higher WMC and IQ scores. Genetic correlations (i.e., the extent to which variables are influenced by the same genes) were 0.99 between the WMS variables and 0.73 between the WMC variables. Genetic correlations between WMS and WMC variables were lower (~0.50). This suggested for the genetic influences (A) on WM two factors, one for the WMS variables and one for the WMC variables. All unique environmental correlations were low varying between –0.10 and 0.15 and suggested no factor structure but only specific factor loadings for E.

We first tested a model that reflected the genetic (A) and environmental (E) correlation patterns. In the Cholesky decomposition, the pathways between the genetic latent WMS factor (A) and WMC variables were omitted, and in a similar way, pathways from the genetic latent WMC factor (A) to the WMS variables were omitted. For unique environment (E) a specific factor for each variable was specified. The factor model thus contained two latent factors for A (one for the WMS variables and one for the WMC variables), and four specific factors for E. It was tested which of the path loadings were significant. Neither the two factors for A, nor the specific factor loadings for E could be dropped from the model. For A it was allowed to drop specific factor loadings for the variables Load I, Slope and Digit Span. It was then tested whether the two genetic factors were correlated. First, by freely estimating the correlation, second, by constraining the correlation to be one (i.e., a one factor model), and third, by constraining the correlation to be zero. Table 5 shows that a model with two genetic factors, including a freely estimated correlation (–0.54), one specific factor loading for A (Arithmetics), and four specific factor loadings for E, fitted best to the WMS and WMC data. Constraining the correlation to one or zero showed a significantly worse fit of the model.

Table 5

Multivariate model fitting results for WMS and WMC

	–2 LL	$\chi^2$	df	p	AIC	rA
i. Cholesky ACE	12,960.63					
ii. Cholesky AE	12,960.97	0.34 <sup>1</sup>	10	0.99	–19.66	
<i>Two factor model</i>						
<b>Correlate A</b>	<b>12,970.04</b>	<b>9.07<sup>2</sup></b>	<b>10</b>	<b>0.53</b>	<b>–10.93</b>	<b>–0.54</b>
<b>factors free</b>						
Correlate A	13,029.42	68.45 <sup>2</sup>	11	0.00	46.45	1.00
factors 1						
Correlate A	13,009.24	48.27 <sup>2</sup>	11	0.00	26.27	0.00
factors 0						

<sup>1</sup>Compared to model i.<sup>2</sup>Compared to model ii.

Bold indicates the best fitting model.

Secondly, a hierarchical model for WM and IQ with Ag as a third latent genetic factor was tested. The latent factor Ag loaded on general IQ and on the latent genetic factors for WMS and WMC. The correlation between WMS and WMC dropped from –0.54 (as in the first model) to –0.25 in the hierarchical model. Fixing the correlation to –0.54 showed a significantly worse fit of the model indicating that *g* explained a significant part of the genetic WM correlation. However, fixing the correlation between WMS and WMC to zero also showed a significant worsening of the fit, which means that it is not only Ag that explains the genetic covariance between WMS and WMC. Table 6 shows the results of the hierarchical sub models. Comparing the correlation of –0.25 in the hierarchical model to the correlation (–0.54) in the original factor model, we can conclude that WM and *g* contribute *both* (about 50%) to the genetic correlation between WMS and WMC. In Fig. 2 the hierarchical model with three latent factors for A (WMS,

Table 6

Multivariate model fitting results for WMS, WMC, and IQ

	–2 LL	$\chi^2$	df	p	AIC	rA
<i>Hierarchical factor model</i>						
i. Genetic covariance	<b>16,155.32</b>					<b>–0.25</b>
WMS–WMC						
explained by WM						
and <i>g</i>						
Genetic covariance	16,170.73	15.41 <sup>1</sup>	1	0.00	13.41	–0.54
WMS–WMC						
explained by						
WM only						
Genetic covariance	161,66.99	11.68 <sup>1</sup>	1	0.00	9.68	0.00
WMS–WMC						
explained by <i>g</i> only						

Bold indicates the best fitting model.

<sup>1</sup> Compared to model i.



WMC and Ag), the correlation between WMS and WMC, factor loadings and confidence intervals are shown.

## 5. Discussion

The present study investigated the phenotypic and genotypic relationship between WM speed and WM capacity in a sample of 12-year-old twins and their siblings. It is the first study that investigated the heritabilities of WM in children of this age, and that examined the genetic structures underlying WM Speed (WMS) and WM Capacity (WMC). WMS was assessed with a choice reaction task with three memory loads from which a basic mental speed measure and the derived slope were analysed. For WMC we used two subtests of the WISC-R namely Arithmetic and Digit Span. To examine whether the genetic covariance between WMS and WMC could be explained by general IQ (*g*) we performed a hierarchical model that tested this hypothesis. General IQ (*g*) was based on 4 subtests of the WISC-R, Similarities, Vocabulary, Block Design and Object Assembly.

The heritabilities for the WM variables were moderately high, ranging from 43% to 56%, indicating that about half of the phenotypic variance could be explained by genetic variation. These results are comparable to the genetic WM studies in adults (Ando et al., 2001; Luciano et al., 2001). Different heritabilities were reported by Neubauer et al. (2000). For slope they reported a heritability of 11% and for memory scanning set size 1, which is comparable with the basic speed variable of the current study, they found no heritability at all. Other studies did find genetic influences on basal speed measures in adult studies (Boomsma & Somsen, 1991; McGue, Bouchard, Lykken, & Feuer, 1984). However, it is suggested that when the complexity of a task increases, the heritability estimate increases as well (Neubauer et al., 2000; Vernon, 1989). Children might experience a simple WMS task or an increasing load (i.e., the Slope) as more complex than adults do, and therefore use cognitive resources, which adults do not need. The prefrontal lobes play an important role in WM performance and the fact that these brain areas are not completely matured before late adolescence (Anderson, 2002; Kanemura, Aihara, Aoki, Araki, & Nakazawa, 2003) may explain the extra efforts, and hence higher heritabilities in the current age group. The few genetic studies that investigated WM speed in children showed conflicting results. A partly overlapping sample of 5-year-old children performed a similar WM speed task but in a more child friendly version (i.e., this task consisted of two loads and used picture stimuli instead of consonants). Their results were comparable with the present study showing a heritability

of 54% for overall RT, and 29% for the derived slope (Stins et al., 2005). Petrill, Thompson, and Detterman (1995) tested 287 twins between 6 and 13 years old with a set of basic cognitive tasks (Cognitive Abilities Test, CAT; Detterman, 1990). Simple and Choice RT tasks were primarily determined by common environmental factors while a Stimulus Discrimination task appeared to be more influenced by genetic factors. WMC in this study, as measured with a self-paced probe recall task, showed a heritability of 22%.

We tested the hypothesis that WMS and WMC are genetically two different constructs. It was found that our data were best described by two latent factors, one for WMS (Load I and Slope) and one for WMC (Arithmetic and Digit Span). These latent factors were correlated ( $-0.54$ ) but did not completely overlap. In other words, variation in WMS and WMC is influenced by separate genetic factors but also by a common set of genes. How should the correlated and separate genetic factors be interpreted? Referring to the existing theories about WM one might speculate that these findings hold up the theoretical framework as proposed by Baddeley (1992). The correlated factors (i.e., the same set of genes) influencing both WM constructs possibly represent the general controlling system while the separate factors (i.e., separate sets of genes) involve the two slave systems, responsible for the rehearsal of acoustic information, in this case WMC, and for the manipulation of visual input (WMS). Ando et al. (2001) and Luciano et al. (2001) found a common genetic factor influencing different WM domains (i.e., verbal and spatial) and they also hypothesized that the common set of genes found in their studies affected the central executive. Another suggestion for the common genetic factor is general intelligence (*g*). It is found that on a phenotypic level intelligence and WM performance are strongly related. Kyllonen and Christal (1990) claimed that 'reasoning ability is (little more than) working memory capacity' and Colom et al. (2004) revealed that working memory was 'almost perfectly predicted by *g*'. A recent genetic study of Finkel, Reynolds, McArdle, and Pedersen (2005) showed that the heritability of cognitive abilities in adulthood results, for the most part, from genetic influences associated with perceptual speed, instead of genes for cognitive functioning specifically.

In the light of these findings the substantial genetic correlation that we found between WMS and WMC might be 'perfectly' explained by *g*, instead of a genetic relation between WMS and WMC per se. This hypothesis was tested with a hierarchical factor model in which a third latent genetic factor (Ag) was allowed to replace the genetic correlation between WMS and WMC and hence

could explain the genetic covariance between WMS and WMC. The results of these analyses showed that  $Ag$  could not explain the genetic correlation completely, but took out about half of the genetic covariance. This means that both  $g$  and WM are responsible for the shared genes between WMS and WMC. Looking at the path loadings from the latent WM factors to  $Ag$  (0.72 and  $-0.40$  respectively) it is clear that  $g$  is (genetically) closer related to WMC than WMS. This is in line with previous (phenotypic) research, but it might also have to do with the choice of WMC tasks. As both  $g$  and WMC were based on subtests of the WISC-R, and WMS tasks were reaction time measures, it is may be not surprising that the former relationship turned out to be stronger. However, Conway et al. (2002) measured WMC with primary verbal tasks and fluid intelligence with nonverbal tasks; still they found a very strong link between both constructs. This suggests that the relation between WMC and fluid  $g$  is domain-free. The question might be whether WMC in this study was measured in an optimal way. Kyllonen and Christal (1990) already had serious reservations about their battery of WMC tasks, and still there is discussion about the estimation of WM in general and pure estimation of WMC specifically (Conway et al., 2003; Cowan et al., 2005; Oberauer et al., 2003).

Beside a significant genetic correlation between WMS and WMC, our results showed that WMS and WMC are also mediated by different sets of genes. These may be interpreted from a neuro-anatomical point of view. It is reasonable to hypothesize that different WM processes are driven by different parts of the brain which are mediated by separate genetic influences. The existence of distinct neuro-anatomical substrates for different domains, such as spatial, verbal and object WM has been suggested by studies on brain

lesions in humans (Müller, Machado, & Knight, 2002) and by several studies using brain imaging techniques (Courtney, Ungerleider, Keil, & Haxbey, 1996; Goldman-Rakic, 1996; Smith, Jonides, & Koeppe, 1996; Postle, Berger, Taich, & D'Esposito, 2000). Cornette, Dupont, Salmon, and Orban (2001) proposed that for visual stimuli, maintenance of orientations involved a distributed fronto-parietal network, while a more medial superior frontal sulcus region was identified for the manipulative operation of updating orientations retained in the WM. Cowan et al. (2005) emphasized that, especially for WM capacity, parietal lobe mechanisms probably play an important role. They addressed the question whether cortical areas related to WM reflect indeed distinct processes or whether they function as an integrated system. To get a better understanding of cognitive processes resulting from complex mechanisms in the brain, extensive research of different disciplines is required. For future research a joint contribution of genetic and cognitive investigations might be a useful and promising approach to further clarify the mechanisms underlying WM, and in addition illuminate the relation between WM,  $g$  and other cognitive processes.

Summarizing the current results it is firstly shown that WMS and WMC are heritable traits. Secondly, that the variance in WMS and WMC is explained by both an overlapping set of genes, and a separate set of genes. Thirdly, that the overlap of genes involves not only WM processes but is also explained by general IQ ( $g$ ).

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### Appendix A

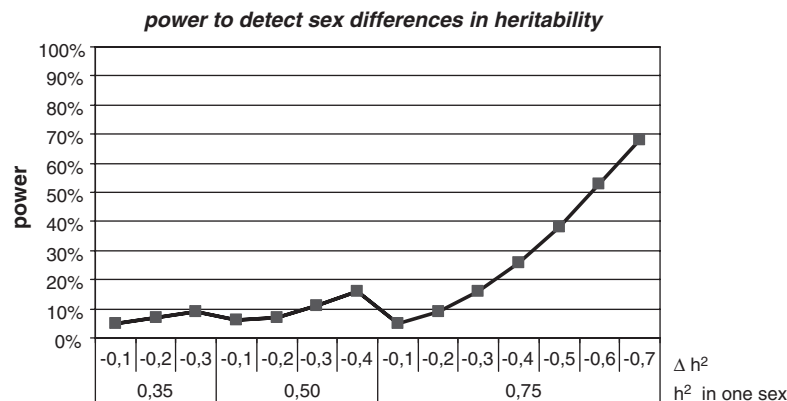


Figure shows the power ( $y$ -axis) to detect differences in heritability ( $x$ -axis) between boys and girls given a sample size of 177 twin pairs and 55 siblings. For a fixed heritability ( $h^2$ ) of 0.35, 0.50 and 0.75 in one sex, the power to detect a difference of heritability ( $\Delta h^2$ ) between boys and girls of respectively  $-0.1$ ,  $-0.2$  and  $-0.3$ ,  $-0.1$ ,  $-0.2$ ,  $-0.3$  and  $-0.4$ , and  $-0.1$ ,  $-0.2$ ,  $-0.3$ ,  $-0.4$ ,  $-0.5$ ,  $-0.6$  and  $-0.7$  is shown.

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