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Heritability of children's Secure Base Script Knowledge in middle childhood: a twin study with the Attachment Script Assessment

Jana Runze,[1†] Annemieke M. Witte,[2†] Marinus H. van IJzendoorn,[3,4] and Marian J. Bakermans-Kranenburg[5,6]

[1]Research Institute for Child Development and Education, University of Amsterdam, Amsterdam, The Netherlands; [2]Clinical Child & Family Studies, VU Amsterdam, Amsterdam, The Netherlands; [3]Department of Psychiatry, Monash University, Melbourne, Victoria, Australia; [4]Research Department of Clinical, Education and Health Psychology, Faculty of Brain Sciences, UCL, London, UK; [5]University Institute of Psychological, Social and Life Sciences, Lisbon, Portugal; [6]Facultad de Psicología y Humanidades, Universidad San Sebastián, Valdivia, Chile

Background: Are individual differences in attachment security inborn or shaped by the social environment? In infancy and early childhood, the evidence points to a substantial role of the environment, but a large twin study in early adolescence showed considerable heritability. Here we examined the twin heritability of attachment in middle childhood. We hypothesized that in middle childhood some heritability would emerge. Furthermore, we expected a role for cognitive and language abilities in explaining variance in attachment in middle childhood, partly related to the measurement of attachment, and we therefore examined associations with IQ. **Methods:** This pre-registered study included 415 same-sex twin pairs (52% girls, 58% monozygotic) between 8 and 11 years old ($M = 9.59$, $SD = 0.79$). Participants were recruited from an experimental cohort-sequential study including two age-overlapping longitudinal cohorts. Secure Base Script Knowledge was assessed with the Middle Childhood Attachment Script Assessment . Zygosity of the twins was determined using DNA samples. In the younger cohort, cognitive development was assessed with the Dutch version of the Wechsler Preschool and Primary Scale of Intelligence . In the older cohort, the Dutch version of the Wechsler Intelligence Scale for Children was used . **Results:** Significant additive heritability (38%) was found in the absence of a common environment component. This result diverges from findings in infancy and early childhood and aligns with the results in early adolescence. **Conclusions:** The gene–environment correlation hypothesis suggesting that older children more actively shape their experiences in social contexts may offer a plausible explanation for the heritability of attachment in middle childhood. In middle childhood this mechanism might tip the balance toward a larger role for additive genetics. Larger longitudinal twin studies are needed to replicate the heritability of attachment after preschool age. **Keywords:** Attachment; heritability; ACE models; twins; Secure Base Script Knowledge.

Introduction

According to attachment theory, every human being is born with the inherited ability to become attached to a protective conspecific, usually the parents or other family members. This species-wide inborn competence might enable the infant to trigger caregiving and protection from parents or other caregivers when fearful or distressed. However, individual differences in attachment security emerge in the first 1,000 days of life and have been shown to be largely determined by environmental influences, in particular the quality of parenting (Bokhorst et al., 2003; Madigan et al., 2024; O'Connor & Croft, 2001; Roisman & Fraley, 2008). Attachment thus is genetically based as well as environmentally determined in a way comparable to acquiring a language that is inborn as well as determined by the language environment one is born into (Van IJzendoorn & Bakermans-Kranenburg, 2024). The central question of the current study is whether attachment security is not only in infancy but also in middle childhood, mostly shaped by the (caregiving) environment.

Like learning a language, attachment behaviors change with development from mostly nonverbal proximity seeking, contact maintaining, and avoidant and ambivalent behaviors in the first few years to verbal representations of attachment indicating a more or less coherent narrative about stressful events such as feeling frightened or suffering an injury and how the child or adolescent is coping with this stress. A core feature of being securely attached is reflected in a coherent narrative describing that the attachment figure will offer comfort and support in case of distress, which consequently alleviates the distress and helps redirecting the individual's attention to ongoing activities and the environment. This balance between exploration from the secure base of an attachment relationship and getting back to the safe haven of the attachment figure in times of (di-)stress characterizes a secure child or adolescent. Some children, however, do not develop the sense of basic trust, presumably because they were rebuffed in their

†Shared first authors.

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seeking comfort with their parent or caregiver. This balance (or the absence thereof) is measured in various ways. For example, in infancy it can be measured with two brief separations and reunions (the Strange Situation Procedure, Ainsworth et al., 1978) or the Attachment Q-Sort (Vaughn et al., 2021); in middle childhood with the Secure Base Script Knowledge (Waters & Waters, 2006) story completion task with attachment-relevant prompt words; whereas in adolescence and adulthood the Adult Attachment Interview (Main & Hesse, 1990) may be used to assess mental representations of past and current-attachment relevant events and relationships.

Evidence for the absence of heritability of attachment differences has been shown in behavioral genetic studies in infancy and early childhood (Bakermans-Kranenburg, Van IJzendoorn, Bokhorst, & Schuengel, 2004; Bokhorst et al., 2003; O'Connor & Croft, 2001; Roisman & Fraley, 2008). To our knowledge, only one study found evidence for the heritability of interview-based attachment quality in adolescent twins (Fearon, Shmueli-Goetz, Viding, Fonagy, & Plomin, 2014) using the Child Attachment Interview (Shmueli-Goetz, 2014), an adaptation of the Adult Attachment Interview to measure attachment representations (Hesse, 2016; Main & Hesse, 1990). In their study, Fearon et al. (2014) found that 40% of the variance in attachment quality could be explained by genetic factors and that the role of the shared environment was negligible. The diverging heritability findings of this study compared to earlier twin studies on attachment might be interpreted in at least three ways. First, observational assessments of attachment differences in young children might be less confounded with (heritable) differences in children's cognitive and language abilities than hour-long semi-structured interviews such as the Child Attachment Interview or the Adult Attachment Interview. Second, beyond considering cognitive or language abilities as mere confounders, the attachment constructs measured in infancy (interactive behavior patterns) versus adolescence (cognitive-affective representations of their attachment relationships) differ also in their level of complexity. Third, the heritability component explaining the variance in attachment differences might increase with development from infancy to adolescence when children become less dependent on their parents. Their genetic make-up becomes more prominent in eliciting (evocative gene–environment correlation or rGE), selecting (active rGE), or experiencing (passive rGE) features of their family environment, including parental behavior as well as the widening social environment (peers, school, neighborhood) that shape their attachment representation.

The field of heritability research using behavioral genetic methods such as ACE modeling based on twin pairs has moved on to more complex models taking into account gene-by-environment interaction and correlation (Knafo & Jaffee, 2013) and has been supplemented with molecular genetics approaches (Plomin, 2019; Warrier et al., 2021). One of the reasons has been that individual differences in almost any psychological trait showed around 40–60% additive genetic influences, with mostly unique environment and measurement error explaining the other part of the variance (Polderman et al., 2015). Infant attachment security is one of the very few exceptions to this rule, with mostly shared and unique environments accounting for the variance. A central question, therefore, is whether and when a genetic component emerges, in view of the fact that in a large twin study on adolescents, a substantial amount of the variance in attachment was explained by additive genetic influences (Fearon et al., 2014) and in the absence of twin studies on attachment in middle childhood. This gap in our knowledge of the heritability of attachment in middle childhood should be filled before examining more complex models and large-scale molecular approaches.

Thus, middle childhood is a missing link in our knowledge of the development of the heritability of attachment. With the current pre-registered study on two cohorts of twins in middle childhood, we hope to fill this gap. Using the age-appropriate Middle Childhood Attachment Script Assessment (ASA; Waters, Bosmans, Vandevivere, Dujardin, & Waters, 2015; Waters et al., 2019) to assess Secure Base Script Knowledge (SBSK) differences in mono- and dizygotic twins, we test the following hypotheses. Our first hypothesis is that children in middle childhood already show partly heritable attachment representations, in line with the findings of Fearon et al. (2014) in adolescence. Our second hypothesis is that the additive genetic component in our group will be smaller than that found in the adolescent sample by Fearon et al. (2014), as we expect that the additive genetic component increases with age. It may even be found that the additive genetic component will be larger in our older childhood cohort compared to the younger cohort, although the difference in age between the two cohorts is only about 1 year (see Methods, below). Third, we expect a role for cognitive and language abilities in SBSK in middle childhood (in contrast to infancy), and we therefore examine the associations between SBSK and verbal (and total) IQ. If these associations are substantial, we explore whether they affect the outcome of our models of heritable and environmental components in explaining variance in SBSK, and we test the associations between genetic and environmental factors influencing IQ and SBSK in a bivariate genetic model.

Methods

Participants and procedure

The sample included 415 same-sex twin pairs (52% girls, 58% monozygotic) between 8 and 11 years old (M = 9.59,

SD = 0.79). Participants were recruited from the Leiden Consortium on Individual Development (L-CID; see website: http://www.developmentmatters.nl/) cohort-sequential study, including two age-overlapping longitudinal cohorts: an Early Childhood Cohort (ECC, *N* = 184 twin pairs) and a Middle Childhood Cohort (MCC, *N* = 231 twin pairs) (Crone et al., 2020; Euser et al., 2016). Families with twin children living in the western part of the Netherlands were selected from municipality records and received an invitation for participation. Families interested in participation received a phone call from a research assistant to assess eligibility. For a more detailed description of the recruitment procedures of the ECC and MCC, see Euser et al. (2016) and Van der Meulen, Steinbeis, Achterberg, Van IJzendoorn, and Crone (2018), respectively. Families were included when their 3–4-year-old (or 7–8-year-old) twin children had the same sex, parents and children were fluent in speaking Dutch, and when both parents and grandparents were of European descent, for reasons of avoiding population stratification in the molecular genetics part of the study, not included here. Families were excluded in case of congenital disabilities, psychological disorders, chronic illnesses, hereditary diseases, visual/hearing impairments, or having an IQ below 70. Families participated in six yearly waves of data collection (T1–T6; alternating home and laboratory visits) with additional ambulatory assessments. The present study used data from the ECC and MCC from measurement time points T6 and T3, respectively. Children in the ECC were between 8.04 and 10.86 years old (*M* = 9.09, *SD* = 0.61), and children in the MCC were between 8.97 and 11.67 years old (*M* = 9.98, *SD* = 0.69). More than 90% of the primary parents were female, and families were mostly from middle-class backgrounds (Euser et al., 2016). Families received a financial compensation for their participation, and children received a small gift. Written informed consent was obtained from parents prior to the start of the study. Ethical approval was received from the central committee on research involving human subjects in the Netherlands (CCMO; ECC: NL49069.000.14; MCC: NL50277.058.14). The pre-registration of the study has been published on the Open Science Framework (https://osf.io/eusfw/?view_only=0455a84f375345bcb7aef8b78aa60247

Secure Base Script Knowledge. Secure Base Script Knowledge (SBSK) was assessed with the Dutch version of the Middle Childhood Attachment Script Assessment (Waters et al., 2015, 2019). This cognitive script is based on eight key components that outline a temporal-causal sequence aimed at eliciting effective secure base support: (1) An individual engages constructively with the environment. (2) The individual is confronted with a threat that prevents constructive engagement and/or elicits distress. (3) The individual communicates a need for support. (4) The attachment figure appropriately recognizes the signal and offers support, which is (5) accepted and (6) effectively addresses the threat. (7) Distress is alleviated through the support, and (8) the individual can resume constructive engagement with the environment. The Attachment Script Assessment has been validated in adults and children from age six onwards (Waters et al., 2015; Waters, Ruiz, & Roisman, 2017; Waters & Waters, 2006). Children were asked to tell four stories using prompt word outlines. Each prompt word outline contained a title and 12 words or statements organized in four columns that imply a prototypical storyline with a beginning, middle, and end. The first prompt word outline was used as a practice story and contained no distressing element. The other three prompt word outlines portrayed familiar scenarios with a distressing element that would prompt the child to seek out their secure base (i.e., scary dog in the yard, injury at the beach, losing a field hockey game). The title of one prompt word set was changed from 'Soccer' to 'Field hockey' because field hockey was a sport commonly played by both boys and girls, whereas soccer still was predominantly played by boys in The Netherlands. Children were instructed to speak as if they were narrating about themselves and their parent, thus speaking in first-person tense. Each story was double coded and assigned a score ranging from 1 to 7. Scores between 4 and 7 indicated that SBSK elements were integrated into the story, with higher scores in this range assigned to stories with more elaborated descriptions of SBSK elements. Scores of 3 were assigned to event-focused stories with minimal to no SBSK elements. Scores of 1 and 2 were assigned to stories with atypical or disjointed content. In case scores of the two coders differed more than 1.5 points, a consensus score was created through discussion. Scores of the two coders were averaged, and we then calculated the mean score of the three stories. The mean score was used as an indicator of children's SBSK, with higher scores indicating more SBSK. The mean Intraclass Correlation Coefficient (ICC) with the expert coder was .81 and .80 among the six coders on average, indicating good reliability.

Zygosity. To determine the zygosity of the twins, an identity by descent analysis was performed using DNA samples collected via buccal swabs. DNA samples were not available for four twin pairs (ECC; *n* = 3, MCC; *n* = 1). For these twin pairs, zygosity was determined through a questionnaire completed by the primary parent with eight items about the twins' physical resemblance and perceived difficulties with distinguishing the twins (Rietveld et al., 2000). The questionnaire was reliable in predicting zygosity classification in 93% of the cases as compared to our own DNA analyses. Two samples were reanalyzed due to parental doubts about the DNA-based zygosity classification. We suspected that there was an accidental switch, and reanalysis of the DNA samples confirmed that one twin pair was monozygotic (not dizygotic), and the other twin pair was dizygotic (not monozygotic).

Covariates. In the ECC, we assessed children's cognitive development at T3 and T4, with the Dutch version of the Wechsler Preschool and Primary Scale of Intelligence, Third Edition (WPPSI-III; Wechsler, 1967). The test provides a full-scale IQ, performance IQ, and a verbal IQ with reference means (*SD*) of 100 (15). Verbal IQ was assessed at T3, and performance IQ was assessed at T4. We combined both subscales into a total IQ score. In the MCC, we assessed children's cognitive development at T1 with the Dutch version of the Wechsler Intelligence Scale for Children (WISC-III, Wechsler, 1991). Similar to the WPPSI-III, the test yields a full-scale IQ, performance IQ, and verbal IQ with reference means of (*SD*) of 100 (15). In the present study, we controlled for children's verbal IQ scores.

Data analytic strategy

Analyses were conducted in R (version 4.2.2, R Core Team, 2017). First, we computed descriptive statistics, correlations, and within-twin correlations of the variables of interest. We conducted univariate genetic covariance structure models using the umx package (Bates, Maes, & Neale, 2019) to estimate the contributions of genetic and environmental factors to the phenotypic variance of the SBSK. The genetic covariance structure model contains the contributions of additive genetic factors (A), shared environmental factors (C), and unique environmental factors, including measurement error (E), which can be estimated because monozygotic (MZ) twins share 100% of their alleles (i.e., are genetically identical), while dizygotic (DZ) twins on average, share 50% of their alleles. We first fitted the saturated model (baseline model), in which we estimated the (unconstrained) MZ and DZ covariance matrices. Then, we fitted the ACE model. We use the log-Likelihood Ratio Test (LRT) statistic with an alpha of .05. In additional analyses, we added cohort (younger vs. older), age

of the child, and verbal IQ scores as covariates into the univariate ACE model. As sensitivity and exploratory analyses, we conducted bivariate genetic covariance structure models on children's verbal IQ and SBSK. Moreover, we performed analyses for each cohort as well as for boys and girls separately. We computed power for the genetic covariance models using the shiny app "twin power calculator" using different combinations of heritability coefficients (https://shiny.cnsgenomics.com/TwinPower/, Visscher, 2004). Power to detect A was acceptable when A was at least .30 and excellent when A was at least .40 (see Appendix S1).

Results

Descriptive statistics, percentage of missingness per variable, and correlations between all variables can be found in Table 1. There were no outliers in verbal IQ and 10 outliers in SBSK. These outliers were included in our analysis, as they represent real data points, but we repeated the analyses with winsorized outliers (see Appendices S2 and S3). Using Little's MCAR test from the naniar package (Tierney & Cook, 2020), we found that missingness did not occur completely at random (χ^2 (2) = 25.57, $p < .001$); therefore, we used full information maximum likelihood estimation (Newman, 2014). Cross-twin correlations, standardized estimates, and unstandardized estimates, including confidence intervals of the ACE models, can be found in Table 2.

We conducted a univariate ACE twin model investigating the heritability of SBSK. The model fitted the data in comparison to the saturated model ($\Delta\chi^2$[6] = 10.14, $p = .119$, AIC = 791.13 vs. AIC = 792.98). Cross-twin correlations, as well as standardized and unstandardized estimates, including confidence intervals, can be found in Table 2. Goodness of fit indices of all analyses can be found in Table 3. An AE model provided the best fit according to Akaike's information criterion (AIC), but the CE model had no worse fit than the full ACE model either. Heritability of SBSK was estimated to be 38%.

In a second step, we controlled for the cohort (older vs. younger); in a third step, we controlled for children's verbal IQ scores; and in a fourth step, we controlled for the age of the child. Results were comparable to the main model, with 36% and 35% of variance in SBSK being explained by genetic factors, respectively (see Tables 2 and 3). Sensitivity and exploratory analyses are reported in the Supporting Information. In both cohorts analyzed separately, ACE modeling suggested a preference for CE instead of AE models (Appendices S2 and S3).

To followup on this result, we conducted a multi-group approach to examine whether the variance components differed between the two cohorts (see Appendix S9). Constraining all variance components to be equal for both cohorts resulted in significant model worsening (Model 1). Constraining each component separately (Models 2–4) showed that the decrease in model fit was caused by differences in the amount of variance explained by E (not A or C) in the two cohorts. Splitting the sample into two cohorts results in less statistical power, leading to less precise estimates and more variability in model fit statistics, which might explain finding different models than in the total group. Modeling

Table 1 Descriptive statistics and correlations of all study variables for both cohorts combined

Variable	*N*	*M*	*SD*	Range	% Missing	Correlations 1	2	3
Full sample								
1 Parent Age	806	42.13	4.71	27.00–58.00	2.89			
2 Child Age	810	9.59	0.79	8.04–11.67	2.41	**.20**		
3 Verbal IQ	816	104.77	14.70	65.00–145.00	1.68	−.02	.06	
4 SBSK	733	3.27	0.39	1.00–4.67	11.69	.05	**.23**	**.18**
ECC								
1 Parent Age	354	41.66	4.65	27.00–58.00	3.80			
2 Child Age	354	9.09	0.61	8.04–10.86	3.80	.08		
3 Verbal IQ	356	102.25	15.04	65.00–135.00	3.36	.05	−.08	
4 SBSK	311	3.18	0.47	1.00–4.67	15.49	−.00	.00	.11
MCC								
1 Parent Age	452	42.50	4.73	31.00–56.00	2.26			
2 Child Age	456	9.98	0.69	8.97–11.67	2.30	**.26**		
3 Verbal IQ	460	106.72	14.13	705.00–145.00	0.43	−.09	.02	
4 SBSK	422	3.33	0.31	2.25–4.17	8.66	.08	**.33**	**.21**

Bold estimates are significant at $p < .05$. SBSK, Secure Base Script Knowledge.

Table 2 Cross-twin correlations and standardized estimates of the univariate ACE models

	r_{MZ}	r_{DZ}	A	C	E
Main model	.38 [0.28, 0.48]	.24 [0.10, 0.36]			
ACE					
Unstandardized [CI]			.55 [0.00, 0.69]	.27 [0.00, 0.59]	.79 [0.72, 0.86]
Standardized			.31	.07	.62
AE					
Unstandardized [CI]			.62 [0.60, 0.69]		.78 [0.72, 0.85]
Standardized			.38		.62
Controlling for Cohort	.39 [0.29, 0.49]	.23 [0.10, 0.36]			
ACE					
Unstandardized [CI]			.54 [0.00, 0.68]	.25 [0.00, 0.58]	.80 [0.74, 0.87]
Standardized			.30	.06	.64
AE					
Unstandardized [CI]			.60 [0.54, 0.68]		.80 [0.73, 0.86]
Standardized			.36		.64
Controlling for Age	.39 [0.29, 0.49]	.23 [0.10, 0.36]			
ACE					
Unstandardized [CI]			.53 [0.00, 0.68]	.26 [0.00, 0.58]	.81 [0.74, 0.88]
Standardized			.28	.07	.65
AE					
Unstandardized [CI]			.60 [0.50, 0.68]		.80 [0.74, 0.87]
Standardized			.36		.64
Controlling for Verbal IQ	.38 [0.28, 0.48]	.23 [0.10, 0.35]			
ACE					
Unstandardized [CI]			.52 [0.00, 0.67]	.27 [0.00, 0.58]	.81 [0.74, 0.88]
Standardized			.27	.07	.66
AE					
Unstandardized [CI]			.59 [0.49, 0.67]		.81 [0.74, 0.87]
Standardized			.35		.65

Table 3 Full and best-fitting Cholesky decomposition fit statistics for univariate models of the main analysis and all sensitivity analyses

	−2LL	*df*	AIC	$\Delta\chi^2$	Δdf	*p*
Full sample						
ACE	783.13	856	791.13	NA	NA	NA
AE	**783.36**	**857**	**789.35**	**0.23**	**1**	**.634**
CE	786.18	857	792.18	3.05	1	.081
E	826.69	858	830.69	43.456	2	<.001
Controlling for cohort						
ACE	762.07	855	772.07	NA	NA	NA
AE	**762.22**	**856**	**770.22**	**0.15**	**1**	**.695**
CE	764.85	856	772.85	2.78	1	.095
E	800.13	857	806.13	38.06	2	<.001
Controlling for age						
ACE	741.75	853	751.75	NA	NA	NA
AE	741.95	854	749.95	0.20	**1**	.656
CE	744.30	854	752.30	2.55	1	.110
E	779.07	855	785.07	37.32	2	<.001
Controlling for verbal IQ						
ACE	738.33	840	748.33	NA	NA	NA
AE	**738.54**	**841**	**746.54**	**0.21**	**1**	**.645**
CE	740.63	841	748.63	2.30	1	.129
E	773.51	842	779.51	35.19	2	<.001

The −2LL = −2 log-likelihood ratio test statistic. AIC = Akaike's information criterion; Δdf = change in degrees of freedom when model parameters were dropped; $\Delta\chi^2$ = change in −2LL when model parameters were dropped; *p* = *p*-Value of significance of the chi-square test. Best fitting models are presented in bold. Note that in all four models, the CE model did not fit worse compared to the full ACE model.

ACE in the sub-groups of boys and girls separately, the CE model was preferable in girls, whereas the AE model was replicated in boys based on model fit assessed using Akaike's information criterion (AIC). However, power was lower (i.e., power to detect $A = .38$ was .51 in the girls sample) in these smaller samples, hampering strong conclusions. Because of a significant correlation between SBSK and verbal IQ, we conducted a bivariate ACE model (see Appendices S4, S5, and S6 for cross-twin correlations between both phenotypes and for the proportion of phenotypic variance and covariance accounted for by A, C, and E, respectively). The model fitted the data in comparison to the saturated model ($\Delta\chi^2[17] = 20.38$, $p = .255$, AIC = 5029.29 vs. AIC = 5042.91). The heritabilities of SBSK and verbal IQ were 32% and 18%, respectively. Unique environmental factors explained 62% and 66% of the variance in SBSK and verbal IQ, respectively. Based on marginal differences in fit, common environmental factors played no significant role. However, low power to detect C might have prevented finding a significant estimate of C. A substantial genetic correlation was found between SBSK and verbal IQ ($r = .51$), implying that genetic factors influencing SBSK are partly overlapping with genetic factors influencing IQ.

In response to one reviewer's suggestion, we also conducted a bivariate ACE model using SBSK and

observed parental sensitivity (see Appendices S7 and S8). The heritabilities of SBSK and parental observed sensitivity were 30% and 5%, respectively. Common environmental factors explained 7% and 47% of the variance in SBSK and parental observed sensitivity, and unique environmental factors explained 62% and 48% of the variance in SBSK and parental observed sensitivity, respectively. The correlation between shared environmental factors explaining the variance and covariance in SBSK and parental sensitivity was .56, implying that shared environmental factors influencing SBSK are partly overlapping with shared environmental factors influencing parental sensitivity.

Discussion

Human newborns are equipped with a genomic constitution that enables them to become attached to potentially protective conspecifics. However, the quality of attachment relationships is assumed to be dependent on the social environment in which they are born. Twin studies on infant and preschool samples have documented the role of common and unique environments and the absence of heritability of attachment differences between the children (for a review see Bakermans-Kranenburg & Van IJzendoorn, 2016). In the largest adolescent twin study to date, however, genetic effects were estimated to account for 35%–37% of the variance in security (Fearon et al., 2014), and a crucial question is when this change from environmentally to genetically explained attachment variations emerges. Middle childhood might serve as a bridge between the two developmental stages. In this first, pre-registered twin study on attachment in middle childhood, we used the Secure Base Script Knowledge (SBSK) approach to assess attachment in mono- and dizygotic same-sex twins. Overall, we found a significant additive heritability component and a rather substantial role for the unique environment and error component in combination with a low shared component in the ACE model. This diverges from findings for earlier periods in the children's lives and aligns with the results in early adolescence.

The estimate of the heritability component did not change when we controlled for verbal or total IQ and for the somewhat younger versus older cohort of twins. However, the ACE results seemed not robust in sensitivity analyses examining the A, C, and E components in the younger and older cohorts separately. In those separate analyses in smaller subgroups we found some evidence for a larger role of the shared environment compared to the additive genetic component, with a CE model preferable based on marginal differences in fit. For the subgroup of female twins, we also found this preferred CE model, whereas in male twins, the AE was supported based on marginal differences in fit. Based on these results, we cannot conclude that there is no role for the shared environment in attachment representations in middle childhood. It should be noted that the power of these exploratory analyses was considerably lower than the power in the analyses on the overall sample. These findings should be replicated in order to increase robustness and to allow firmer conclusions.

In the introduction we proposed two hypotheses to explain the role of heritability in the variance of attachment representations in the adolescent twins study (Fearon et al., 2014). First, compared to non-verbal behavioral observation procedures for measuring infant attachment (such as the Strange Situation Procedure and the Attachment Q-Sort), the *language hypothesis* points to the larger role of language and cognitive competence in actively producing verbal answers to rather complicated questions about (early) childhood and the current perception thereof in the Child Attachment Interview (Fearon et al., 2014). Second, the influence of genetics on adolescent attachment representations might increase with the widening of the family environment to experiences in peer groups, schools, and other socializing contexts that are 'sought after' or evoked by the adolescents, the *gene–environment correlation hypothesis* (see Knafo & Jaffee, 2013). Attachment theory predicts that early attachment (in-)security affects children's relationships with peers later in childhood (Sroufe, Egeland, Carlson, & Collins, 2005), and meta-analytically, rather substantial predictive associations have been found between attachment and peer competence (Groh et al., 2014). Peer relationships in turn may reinforce children's attachment representations developed earlier (Bowlby, 1973; Waddington, 1942). A third explanation might be related to the possibly lower reliability or stability of early attachment assessments that are still 'works in progress' ('working models' of attachment, Bowlby, 1969) and more open to environmental influences than the more internalized and solidified attachment scripts and representations in adolescence. This *stability hypothesis* might imply a larger E component consisting of unique events and error in early attachment assessments compared to later measurements.

Relevant for the language hypothesis, we found a significant additive genetic component in explaining individual differences of SBSK in middle childhood even when controlling for verbal and total IQ. This finding suggests that differences in language competence and general cognitive ability cannot fully explain the presence of the heritability component, despite rather strong genetic correlations between SBSK and IQ in our exploratory bivariate genetic covariance structure models (see Appendix S4). The solidifying of internalized attachment and possibly lower error rates in attachment assessments at a later age than infancy and early childhood seems also not very plausible in view of our results. The majority of variance in individual SBSK assessments

in middle childhood twin pairs was unique (E), which combines transient or stable but unique environmental experiences as well as measurement error. Consequently, the gene–environment correlation hypothesis, which suggests that developing children are more actively shaping their experiences in widening social contexts, seems the most plausible hypothesis. In middle childhood this might tip the balance toward a larger role for additive genetics, although the estimated genetic influence shows still considerable variation and might be more pronounced in some sub-groups, such as in middle-childhood boys.

Obviously, our findings should be replicated in independent and larger twin studies before being taken for granted. With larger twin studies, we also mean including participants from a larger variety of environments outside the over-represented, homogeneously white, middle-class families from WEIRD (Henrich, Heine, & Norenzayan, 2010) countries (Polderman et al., 2015). For twin studies, this is especially important because a homogeneous environment logically implies a larger role for the genetic component in the ACE models. Smaller C means larger A, all things else being equal. The implication is that heritability estimates are tied to specific populations and cannot be generalized easily (Van IJzendoorn & Bakermans-Kranenburg, 2024).

In conclusion, the current study on twins in middle childhood supports the call of Fearon et al. (2014) for a closer look at the genetic and environmental factors that shape attachment differences after early childhood. Additional twin studies in middle childhood and adolescence would be helpful to decide how replicable these first heritability estimates are. Longitudinal twin studies with a variety of attachment assessments might provide even more convincing evidence for the role of genetic and environmental factors in the development of attachment over time. In addition, going beyond behavioral genetics studies, a move toward measuring genome-wide genetic differences aggregated in polygenic scores might address the heritability of attachment differences in a more definite way-though if and only if the complex trait of attachment is measured in a valid way.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article:

Appendix S1. Power calculation for univariate twin models with differing levels of A.

Appendix S2. Cross-twin correlations and standardized estimates of the univariate ACE models.

Appendix S3. Full and best-fitting Cholesky decomposition fit statistics for univariate models of the main analysis and all sensitivity analyses.

Appendix S4. Cross-twin pair correlations across phenotypes.

Appendix S5. Bivariate ACE model with secure base script knowledge and verbal/total IQ.

Appendix S6. Independent standardized estimates of the bivariate ACE models examining SBSK and verbal/total IQ.

Appendix S7. Cross-twin pair correlations between secure base script knowledge and parental sensitivity.

Appendix S8. Bivariate ACE model with secure base script knowledge and observed parental sensitivity.

Appendix S9. Model fitting results for the genetic ACE model across cohorts.

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The authors have declared that they have no competing or potential conflicts of interest.

Ethical considerations

Written informed consent was obtained from parents prior to the start of the study. Ethical approval was received from the central committee on research involving human subjects in the Netherlands (CCMO; ECC: NL49069.000.14; MCC: NL50277.058.14).

Data availability statement

The data that support the findings of this study are available upon reasonable request (see http://www.developmentmatters.nl/data-access/).

Correspondence

Marian J. Bakermans-Kranenburg, William James Center for Research, Ispa – Instituto Universitário, Rua Jardim do Tabaco 34, 1149-041 Lisboa, Portugal; Facultad de Psicologia y Humanidades, Universidad San Sebastián, Gral Lagos 1163, 5110693 Valdivia, Chile; Email: contact@marianbakermanskranenburg.nl

Key points

- Behavioral genetic studies in infancy and early childhood showed a substantial role for the (common) environment in explaining differences in attachment, but a twin study in early adolescence showed considerable heritability.
- In our study on children in middle childhood, we found a significant additive heritability component in combination with a low common environment component.
- A plausible interpretation seems to be the gene–environment correlation hypothesis, which suggests that older children more actively shape their experiences in widening social contexts.
- In middle childhood this might tip the balance toward a larger role for additive genetics in the development of attachment security and insecurity.

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