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The Heritability of Parents' Emotion Socialization Behaviors: Evidence from a Population-based Extended Twin Study

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Abstract

Objectives. This study investigated genetic and environmental contributions to parental emotion socialization, addressing the limited attention given to heritability in previous research and frameworks. Additionally, we examined the intergenerational transmission of emotion socialization by decomposing the covariance between parents' current practices and the emotion socialization they experienced in their family of origin.

Methods. We employed a population-based extended twin design that included monozygotic and dizygotic twins, full siblings, and cousins, with both maternal and paternal relatives. Questionnaire data were collected from Norwegian parents (N = 1803; 1106 mothers) of children aged 3-12 years (M = 7.4 years) and analyzed using univariate and bivariate modeling.

Results. Genetic factors explained more than 20% of the variance across all four forms of parental emotion socialization (supportive and non-supportive; current and recalled). The unique environment accounted for 47–72%, and the shared environment showed a minimal effect. Bivariate model testing of the covariation between current and recalled emotion socialization showed that a model with only genetic and unique-environmental effects provided the best fit for both supportive and non-supportive emotion socialization practices.

Conclusions. Parental emotion socialization shows moderate heritability and is primarily influenced by unique environmental factors, whereas shared family background has limited impact. This highlights the importance of considering both genetic factors and individual life experiences in understanding how parenting practices are developed and transmitted across generations.

Subject classification codes: Parenting style/process; Family, Parenting; Emotion regulation; Socialization.

Introduction

Over the past few decades, parental emotion socialization (ES) has proven to be an important factor in predicting child outcomes across various domains (for reviews, see e.g., Hajal & Paley, 2020; Spinrad & Eisenberg, 2023). Therefore, understanding the sources of variation in how parents socialize their children's emotions is essential. According to Eisenberg and colleagues' process model (Eisenberg, 2020; Eisenberg et al., 1998), a range of characteristics related to the parent, the child, and the context serve as predictors of parental ES, and research has identified various psychological and environmental predictor variables, generally showing small effect sizes (Eisenberg, 2020; Hajal & Paley, 2020). A new area of inquiry examines how parents' experiences with ES in their family of origin influence their current parental ES within an intergenerational framework, with recent studies indicating that parents' recollections of their own parents' ES behavior are a central predictor of their current ES toward their children (Leerkes et al., 2020; Severinsen et al., 2026).

Studies of parental ES have identified a range of predictors, but have generally not used genetically informative designs, leaving the relative contributions of genetic and environmental influences to

individual differences in parental ES largely unknown. Behavioral genetics offers a complementary perspective to traditional developmental psychology research by estimating the sources of individual variation. The present study applies behavioral genetic methods to decompose variation in parental ES into genetic and environmental components. Additionally, we examine parents' recalled experiences of parental ES in their family of origin, thereby providing an intergenerational perspective on ES, and estimate the extent to which the association between current and recalled ES reflects genetic versus environmental factors.

By using an extended twin design and a large population-based sample, we address several gaps in the current literature. First, heritability estimates for parental ES have not been previously published. While strong evidence indicates that genetic heritability is important for most human traits (Polderman et al., 2015), parents' genetics are notably absent from influential process models of parenting (Belsky, 1984; Taraban & Shaw, 2018), as well as from process models specific to parental ES (Eisenberg, 2020; Eisenberg et al., 1998). This likely reflects both a historical emphasis on modifiable environmental factors in parenting research and the assumption that genetics are more relevant to stable traits (e.g., personality or temperament). In addition, genetically informative studies are logistically demanding, which may have limited their use in emotion socialization research. Second, much existing research on parental ES has relied on relatively small samples or on convenience or clinically selective samples, and third, fathers remain underrepresented. A behavioral genetics approach can deepen our understanding of parenting by uncovering the combined roles of genetics and environment (Grigorenko, 2024; Kretschmer, 2023). This understanding can, in turn, guide the development of theoretical models as well as interventions aimed at promoting beneficial parenting methods. To the best of our knowledge, no previous studies have been published on parental ES using an extended twin design and a large population-based sample.

Parental Emotion Socialization

Parental ES is a specific aspect of parenting and can be defined as behaviors that shape children's experience, regulation, expression, and understanding of emotions (Zahn-Waxler, 2010), aiming to help individuals successfully adapt and function within their social environment (Hofmann & Doan, 2018). Emotion socialization theory suggests that children learn about emotions through interactions with parents, including how parents respond to their emotions, family discussions about feelings, and how parents express their own emotions (Eisenberg et al., 1998).

Parental ES behaviors are generally categorized as supportive (e.g., validating and encouraging emotional expression,) or non-supportive (e.g., punitive or dismissive responses to emotions (De Raeymaecker & Dhar, 2022; Fabes et al., 2001). Supportive ES is thought to help children develop emotional understanding, emotion regulation, and the ability to express emotions appropriately in social settings (i.e., emotional competence); conversely, dismissive or punitive responses are thought to limit children's opportunities to learn emotional competence (Gottman et al., 1996). Research on emotion socialization has focused on how parents respond to children's negative emotions, acknowledging that managing negative emotions is a major challenge for both parents and children (Kiff et al., 2011; Nelson et al., 2009).

Intergenerational Transmission of Emotion Socialization

A key aspect of parenting research is studying how parental behavior is passed down through generations, known as intergenerational transmission. This phenomenon is familiar in everyday language, e.g., "the apple does not fall far from the tree," and recognized in parenting programs, where emphasis is placed on parents' recollections of their own upbringing and how these influence current parenting (Buston et al., 2022; England-Mason & Gonzalez, 2020). A growing body of evidence documents moderate but robust continuity in parenting practices across generations (for recent reviews, see e.g. Belsky et al., 2009; Kerr & Capaldi, 2019; Rothenberg, 2019).

Retrospective reports of parenting may be prone to a variety of biases; however, the continuity of parenting across generations persists when examining recollections of parenting in one's family of origin (Huffhines et al., 2024; Leerkes et al., 2020) and has been documented using both self-reports (Capaldi et al., 2008) and observations (Conger et al., 2013). Research on intergenerational transmission has mainly focused on maltreatment and harsh parenting. In the field of emotion socialization, a history of parental emotional rejection has been found to correlate negatively with accurate identification of emotions and positively with negative attributions, amusement, and neutral responses to child distress (Leerkes & Siepak, 2006). A longitudinal study also found that mothers' recollections of non-supportive ES were negatively associated with self-efficacy in managing toddlers' distress (Cao et al., 2023). Taken together, these findings may suggest that parental ES partly reflects the impact of early experiences within the family of origin, potentially transmitted through social learning and modeling (Morris et al., 2007). However, behavioral genetic research suggests that individual differences in parenting are substantially shaped by genetic influences (Klahr & Burt, 2014), underscoring the need to consider both environmental and genetic pathways when studying continuity in parental ES.

The Nature and Nurture of Parental Emotion Socialization

Behavioral genetics seeks to understand the sources of variation in human behavior and can provide important insights into whether there is a genetic effect on parental ES and the relative strength of genetic and environmental influences. Twin and extended-family designs compare relatives who differ in their degree of genetic relatedness (e.g., monozygotic (MZ) twins, dizygotic (DZ) twins, full siblings (FS), cousins (C)) to decompose phenotypic variance into additive genetic and environmental components. Under standard assumptions (including equal environments for MZ and DZ twins), greater similarity in parental ES among MZ twins than among DZ twins indicates genetic influence, whereas comparable correlations point to a predominant role for environmental factors (including measurement error), and DZ correlations greater than half of the correlation between MZ twins indicate shared environmental influences (Jaffee et al., 2013; Knopik et al., 2017; Mayhew & Meyre, 2017; Samek et al., 2013).

Genetically informative studies typically estimate the additive genetic effect (A) and decompose the environmental effect into shared (or common) (C) and unique (or non-shared) (E) components, with the estimate of E also including measurement error. Using biometric modeling techniques that assume

correlations between genetic and environmental similarities among siblings, the effects of the latent components (A, C, and E) can be estimated for a specific phenotype (univariate decomposition), and bivariate and multivariate models allow for the simultaneous decomposition of two or more phenotypes and enables the estimation of genetic and environmental sources of covariance between them (Knopik et al., 2017). Analyzing the cross-variance in relatives can explain the environmental versus genetic etiology of the association between two phenotypes, i.e., why there is an association between current parental ES and ES in the family of origin.

Genetically informed designs are usually child- or parent-based, depending on whether the sampling is based on the child being twins (child-based) or the parent being twins (parent-based). Given the difficulty of recruiting large samples in a parent-based design (one must recruit two families to complete a pair of twins, in contrast to recruiting just one family in a child-based design), the literature has comparatively fewer parent-based designs (Klahr & Burt, 2014). One way to increase the sample size is to extend the classical twin design to include relatives beyond the nuclear family with different levels of genetic relatedness, and thus different correlations for A. Expanding the design to include other relatives can improve the accuracy of estimates and make them less sensitive to violation of assumptions (Keller et al., 2010; Zyphur et al., 2013). However, an extended twin family design comes with the cost of increasing the complexity of the theoretical model (Keller et al., 2010). For example, the correlation coefficient of C is typically assumed to be 1 for all siblings growing up in the same nuclear family and 0 for other relatives. However, the assumption of C for full siblings and cousins is not straightforward; the within-family environment can change over time due to various factors, such as the age of the parents, changing socioeconomic status, illnesses, conflicts, etc., and therefore, C might vary as a function of the age difference between siblings. Furthermore, because of kinship, cousins may share some environmental influences, even though they do not grow up in the same nuclear family.

The application of behavioral genetics to the study of parenting is a relatively novel approach (Kendler, 1996; Lytton et al., 2002; McGuire, 2003). Overall, genetic studies of parenting have found evidence of substantial heritability in parenting practices (Bornstein, 2016; Kretschmer, 2023; McGuire et al., 2012). Two meta-analyses found that the parental warmth dimension has the highest heritability estimates (ranging from 30% to 40%), while the parental control dimension has the lowest estimates (ranging from 10% to 20%; Kendler & Baker, 2007; Klahr & Burt, 2014). Other work, particularly using child-based designs, suggests that negative aspects of parenting (e.g., harshness, negativity) show higher heritability than positive dimensions (Oliver et al., 2014). The environmental effects on parenting have been found to be mainly or entirely due to unique factors, including measurement error, with no notable effect of the shared environment (Klahr & Burt, 2014). Studies have also indicated genetic effects on the recollection of parenting, although the mechanisms are unclear (Lichtenstein et al., 2003). There is solid evidence that parenting in general (Root & Denham, 2010) and parental ES specifically (Baker et al., 2011; Garside & Klimes-Dougan, 2002; McKee et al., 2022; Wong et al., 2009) differ as a function of parent gender; however, studies using parent-based designs showed no evidence for differences in the gene-environment etiology of parenting between mothers and fathers (Klahr & Burt, 2014).

Behavioral genetic research has led to the conclusion that the unique environment holds a greater significance for psychological development than the effect of environmental factors shared by siblings growing up in the same family (Plomin & Daniels, 2011). These unique experiences can be random exogenous events, such as accidents or illness, but also internal and idiosyncratic individual psychological characteristics (Plomin, 2023), as when siblings are reacting uniquely to a shared event (e.g., a divorce). In summary, research indicates that heritability influences parenting and that the unique environment remains the primary factor, but so far, no genetic studies have been published that specifically examine the etiology of parental ES.

The Present Study

The present study addresses two questions. First, what are the relative contributions of genetic and environmental influences to individual differences, both in parents' current ES and in their recalled ES in the family of origin? Second, to what extent is the covariation between parents' current and recalled ES attributable to genetic versus environmental influences? For both questions, we divided environmental influences into those shared among all siblings (both twins and non-twins), those shared only between twins, and those unique to each individual.

Based on previous genetic research on parenting, we anticipated that unique environmental factors would account for the largest proportion of variance in both current and recalled emotion socialization, with genetic factors also explaining a substantial portion, whereas shared environmental effects were expected to be minor or negligible. We further hypothesized that a substantial portion of the observed covariation would be attributable to genetic factors rather than the shared environment, providing a potential genetic explanation for the observed continuity across generations despite the limited influence of the shared environment on sibling similarity.

Method

Procedures and Sample Selection

This study is part of the extensive "Parenting Practices in Norway" (PPN) research project (Bekkhus et al., 2023). Data collection took place from September to December 2022. Parents were drawn by stratified random selection from the National Register of Citizens using a family-based study design, according to the following criteria: They were parents with at least one child aged 3–12 years and additionally either a) twins or siblings to twins sharing the same mother, b) born to twins, c) siblings born abroad, or d) partners to any of the mentioned categories. To maximize the chances of a diverse sample, we intentionally oversampled full-sibling parents with cultural backgrounds from countries other than Norway. Participants received invitations via email and SMS dispatches, providing a link to an online consent form and questionnaire. The final sample of the PPN project (N = 5419) consisted of parents, of whom approximately 19% were twins, 29% were born to twins, and 52% of the parents were neither twins nor born to twins. In instances of multiple children within the target age range, we requested parents to provide

responses pertaining to the child nearest to eight years of age. The parents' family membership was retrieved from the National Population Register. For this study, we extracted a combination of a parent-based sample, comprising parents who were part of an MZ or DZ twin dyad and their full siblings (FS), and a child-based sample, consisting of parents who were born to a twin and cousins through MZ or DZ twins (total *N* = 1803).

Ethical considerations

The PPN project has undergone pre-registration and has received approval from the Regional Committees for Medical and Health Research Ethics (reference number: REK-2022/346141). All participants provided electronic informed consent and received a universal gift card valued at 400 NOK (approximately 33 EUR) as compensation for their time.

Measures and Instruments

Demographics

Parents reported their age in years, current relationship status (either in a relationship or not), education level, annual personal income, number of children in the household, and country of birth. Information on parent gender and kinship was obtained from the National Register of Citizens. Education was reported in six categories: no education, primary school, high school, vocational college, college or university with less than 4 years of education, and college or university with more than 4 years of education. Parents reported their financial status in terms of annual personal income in increments of 100 000 NOK, ranging from '0' to 2 000 000 NOK, with the final domain exceeding 2 000 000 NOK. Parents also provided information for the targeted child´s gender (girl, boy, other), age (year of birth), and birth order. The final sample is detailed in Table A1 in the appendix.

Parent emotion socialization

Parent emotion socialization was assessed using the *Coping with Children's Negative Emotions Scale* (CCNES; Fabes et al., 2001). The CCNES measures parental reactions to children's negative emotions by presenting a series of vignettes that depict children displaying negative emotions, such as "If my child falls off their bicycle and breaks it, and then becomes upset and cries, I would…". The original CCNES instrument comprises 12 vignettes with six response categories and 72 items. For the present study, we used a previously validated abbreviated version (Amundsen et al., 2023). This version includes items and subscales from the original instrument to represent supportive and non-supportive parental ES. The subscale Expressive Encouragement (EE; seven items), with response items like "Encourage my child to express his/her feelings of anger and frustration" was used to measure supportive ES. As a measure of non-supportive parental ES, we used a combination of items from the Minimizing Reactions (MR) scale (four items) and the Punitive Reactions (PR) scale (three items). An example of a minimizing response was, "Tell my child that he/she is overreacting," an example of a punishing response was, "Tell my child he/she must behave, or we must go home immediately." Parents reported the likelihood of employing specific responses to these scenarios, each response item representing a particular emotion socialization

parenting strategy, rated on a 7-point Likert scale from 1 (“very unlikely”) to 7 (“very likely”), with a higher mean score indicating more use of supportive or non-supportive ES. The Cronbach’s alphas were .84 and .66 for the supportive and non-supportive scales, respectively.

Emotion Socialization in the family of origin

As a measure of parents' developmental history, we modified the version of the CCNES described above to measure parents' recollection of their own parents’ use of ES in the family of origin. The modification involved changing the instructions for parents from rating themselves to rating how likely it was for their parents to respond with specific ES behaviors towards them during their upbringing. The respondent was asked to indicate if they based their rating on experiences with both parents, the mother, the father, or another caregiver. In cases where parents used very different ES strategies, they were instructed to rate based on the parent who had the most influence on them. The modification further involved changing the subject of the items, e.g., from “If my child falls off his/her bike and breaks it, and then gets upset and cries, I would…” to “If you fell off your bike and broke it, and then got upset and cried, your parent would…”. Apart from this modification, the instrument and the scales remained identical to the shorter version described above. This ensures that the accuracy and validity of the instrument is maintained while still allowing for the necessary adjustment. Cronbach's alpha coefficient for experienced ES in the family of origin was .94 for supportive and .84 for non-supportive ES.

Zygosity assessment

All parents were administered the item, ”Are you a multiple birth?” (“yes”, “no”, “uncertain”). A confirmative or uncertain answer elicited two new items; “What gender do you and your co-twin have?” (“twin brothers”, “sister and brother”, “twin sisters”) and “Have you examined what kind of twin you are? (“yes, identical”, “yes, fraternal”, “no, not examined”)”. If the response was uncertain on the latter item, zygosity was determined by applying a scale used by the Norwegian Twin Registry (Harris et al., 2006). Using a latent class analysis, which included the twins' questionnaire items, achieved a classification accuracy with entropy = 95%.

Missing Data

Participants were excluded if they had missing values on the age of the child or if the child was outside the desired age range, < 3 years or > 13 years of age (n = 50). Inspecting the missingness for the four outcome variables, current supportive and non-supportive ES and recalled supportive and non-supportive ES in the family of origin, revealed that approximately 95% of the parents completed all items. Prioritizing accuracy over maximizing sample size, we decided to exclude participants with three or more missing items on one of the four seven-item outcome scales were excluded (n = 85), resulting in a final sample of n = 1803.

Statistical Analyses

All analyses were conducted using R, version 4.4.3 (R Core Team, 2025). Pearson's correlation coefficients were used to calculate bivariate correlations among all variables, and associations were examined to assess the degree of overlap and multicollinearity. Table A1 in the appendix presents descriptive statistics for the final sample of 1,803 parents.

To address the first aim, namely estimating the heritability of ES, the subsamples of MZ twins, DZ twins, full siblings, cousins of MZ twins, and cousins of DZ twin pairs were identified. The first step was to calculate and examine the pattern of intraclass correlations for each of the four outcome measures. Next, a series of univariate models was tested to estimate the relative contribution of genetic and environmental factors for each measure. To account for the possibility that twin pairs might share more similar environments because they experience their parents at the same time point, our full model included a specific twin environment (T), which is shared between twins but not between other full-sibling pairs. Thus, as shown in Fig. 1, the observed variance was parsed into four latent factors: additive genetic (A), shared "household" environment (C), twin-specific environment (T), and unique environment (E), with the estimates of E accounting for measurement error. In each case, the full model was an ACTE model. The genetic factor was correlated among relatives according to their coefficient of relatedness, the shared ("household") factor was perfectly correlated among all siblings and twins, the twin-specific factor was perfectly correlated among all twins and uncorrelated among other relatives, and the unique-environmental factor was uncorrelated among all relatives. Parent gender and age were included as covariates. The best model was selected using the maximum-likelihood criterion of fit and Akaike´s Information Criterion (AIC) for parsimony (i.e., maximum fit with minimum number of factors).

For our second goal, examining the genetic and environmental contributions to the covariation between current parental ES and recalled parental ES from the family of origin, we fitted bivariate Cholesky models for both supportive and non-supportive ES, as illustrated in Fig. 1. The models were fitted using the svcm-package in R (Eilertsen, 2021).

Note

Measured variables are depicted as rectangles, and a latent variable as an oval. A = Additive genetic factors, C = Shared environmental effects, T = twin-specific shared environment, E = Unique environmental effects. *a*11, *c*11, t11, *e*11... = path coefficients for additive genetic influence, shared environmental influence, twin-specific shared environment and unique environmental influence, respectively.

Results

Descriptive Statistics

Parents' mean age was 39.9 years, 61.2% of the participants were mothers, and 28.6% were twins. The mean age of the children was 7.4 years, 34.2% were firstborn, and 86.9% of the households had 1–3 children. Most parents were in a relationship (92%), fully employed (74.3%), had a college or university education (68.7%), managed well financially (74.4%), were the biological parents of the child (99.6%), and were born in Norway (95.9%). All descriptive statistics are shown in Table A1 in the appendix. Correlations between all variables are shown in Table 1. Both supportive and non-supportive ES showed positive correlations between current and recalled ES, indicating continuity in parenting practice.

Table 1

Bivariate correlations among study variables.

Variable	1	2	3	4	5	6	7	8	9
1. Supportive ES									
2. Non-sup. ES	− .26***								
3. Rec. Sup. ES	.24***	.06*							
4. Rec. Non-sup. ES	.07**	.27***	− .50***						
5. Parent age	− .06*	.13***	.01	.01					
6. Parent gender	− .27***	.31***	.06*	− .04	.16***				
7. Education	− .04	− .12***	− .04	− .09***	.16***	− .13***			
8. Income	− .14***	.17***	.06**	− .04	.28***	.34***	.34***		
9. Child age	− .02	.12***	.00	.03	.43***	− .01	− .04	.04	
10. Child gender	.00	.04	.02	.01	− .08**	.00	− .04	− .03	− .02

Note. Non-sup. ES = Non-supportive ES, Rec. Sup. ES = Recalled Supportive ES, Rec. Non-sup. ES = Recalled Non-supportive ES, *p < .05. **p < .01, ***p < .001

Intraclass correlations (ICC), calculated as Pearson's *r*, for the dyads of different genetic relatedness, along with 95% confidence intervals, are presented in Table 2. As shown, all ICC of parental ES were higher for MZ pairs compared to DZ pairs. Additionally, the estimated ICC were consistently higher for DZ twin pairs compared to full siblings. Notably, ICC were weaker for cousins, particularly for those through DZ twins, for whom no significant ICC were observed.

Table 2

Intraclass correlation for parents' reports on current and recalled supportive and non-supportive ES by type of relationship (dyad) with confidence intervals

Variable	Type of relationship, *n* of dyads				
	MZ	**DZ**	**FS**	**C/MZ**	**C/DZ**
	n = 131	*n* = 132	*n* = 504	*n* = 161	*n* = 344
Current Sup. ES	.41***	.23**	.09*	.-.06	.10
	[.26, .54]	[.06, .39]	[.01, .18]	[-.21, .09]	[-.01, .20]
Current Non-sup. ES	.34***	.16	.13**	.20*	− .05
	[.18, .48]	[-.02, .32]	[-.05,.22]	[.05, .34]	[-.16, .05]
Recalled Sup. ES	.50***	.35***	.17***	.14	.08
	[.36, .62]	[.19, .49]	[.09, .26]	[-.01, .29]	[-.03, .18]
Recalled Non-sup. ES	.52***	.36***	.28***	.19*	.07
	[.39, .64]	[.20, .50]	[.20, .36]	[.03, .33]	[-.04, .17]

Note. MZ = monozygotic twins, DZ = dizygotic twins, FS = full siblings, C/MZ = cousins through MZ, C/DZ = cousins through DZ. Sup. ES = Supportive ES, Non-sup. ES = Non-supportive ES. Values in square brackets indicate 95% confidence interval for each correlation. *p < .05. **p < .01, ***p < .001

Model fitting

Decomposing the sources of variance in current and recalled supportive and non-supportive ES

A series of alternative univariate models were fitted to estimate genetic (A), shared environmental (C; "household effect"), shared twin-specific environmental (T), and unique environmental (E; including measurement error) contributions to variation in the ES measures. The best-fitting models were selected using the maximum-likelihood criterion of fit and Akaike´s Information Criterion (AIC) for parsimony (i.e., maximum fit with the minimum number of factors). Estimates for all tested models are presented in Table 3, and confidence intervals (CIs) for the best-fitting models are reported in Table A2 in the appendix. For supportive ES, the ATE model provided the best fit for both current and recalled ES, with no improvement achieved by adding a general shared environment (C). For current supportive ES, the additive genetic component (A) explained 20% of total variance, the twin-specific environmental factor (T) explained 14%, and the unique environment (E) explained 66%. Corresponding variance components for recalled supportive ES were 38% for A, 15% for T, and 48% for E.

For non-supportive ES, the AE model was the most parsimonious for both current and recalled ES. The variance components were 28% for A and 72% for E for current non-supportive ES, and 53% for A and 47% for E for non-supportive recalled ES. Estimates remain essentially unchanged if we adjust for positive

skew in current non-supportive ES (through a log-transform) and negative skew in current supportive ES (through reflecting scores, log-transforming them, and then reflecting back). In summary, all models indicated a substantial genetic contribution with heritability estimates (h^2) above .20.

Differences in heritability estimates were examined by comparing confidence intervals (CI). Heritability was generally higher for non-supportive compared to supportive, across both current ES and recalled ES from the family of origin. For current ES, the 95% CI for current supportive (h^2 = .20, 95% CI [.07, .36]) and current non-supportive (h^2 = .28, 95% CI [.16, .35]) overlapped, containing each other's point estimates. For recalled ES, the CI for supportive (h^2 = .38, 95% CI [.23, .56]) and non-supportive also partly overlapped (h^2 = .53, 95% CI [.43, .63]), although the point estimate for recalled supportive fell outside the CI of recalled non-supportive.

When comparing current and recalled ES measures, heritability was consistently higher for recalled than for current ES. For non-supportive ES, the CIs for current and recalled estimates showed no overlap, indicating a clear distinction. For supportive ES, the CIs for current and recalled partly overlapped; however, their point estimates did not fall within each other's intervals.

Table 3

Estimates from univariate models of current and recalled; supportive and non-supportive parental ES.

Variable	Model	h^2	c^2	t^2	e^2	df	$\Delta\chi^2$	p	AIC
Current Sup. ES	ACTE	.204	.000	.136	.660	7		-	4963.9
	ACE	.295	.000		.705	6	3.00	.083	4964.9
	ATE	**.204**		**.136**	**.660**	**6**	**0.00**	**.999**	**4961.9**
	AE	.295			.705	5	3.00	.223	4962.9
Current Non-sup. ES	ACTE	.233	.030	.000	.736	7		-	4918.6
	ACE	.233	.030		.736	6	0.00	.999	4916.6
	ATE	.276		.000	.724	6	0.11	.744	4916.7
	AE	**.276**			**.724**	**5**	**0.11**	**.948**	**4914.7**
Recalled Sup. ES	ACTE	.376	.000	.146	.479	7		-	5051.6
	ACE	.494	.000		.506	6	3.23	.072	5052.8
	ATE	**.376**		**.146**	**.479**	**6**	**0.00**	**.999**	**5049.6**
	AE	.494			.506	5	3.23	.198	5050.8
Recalled Non-sup. ES	ACTE	.398	.091	.025	.486	7		-	5025.8
	ACE	.432	.080		.488	6	0.09	.768	5023.9
	ATE	.533		.000	.467	6	0.95	.329	5024.8
	AE	**.533**	**-**		**.467**	**5**	**0.95**	**.620**	**5022.8**

Note. A = additive genetic factors, C = shared environment factor, T = specific twin shared environment factor, E = unique environment factor; a^2, c^2, t^2, e^2 = variance attributed to A, C, T and E, df = degrees of freedom, $\Delta\chi^2$ = Delta Chi-square statistic, p = significance of $\Delta\chi^2$, AIC = Akaike's Information Criterion. Model with the best fit to the data in bold.

Decomposing the covariation between current ES and recalled ES in the family of origin

We tested a series of bivariate models to examine the extent to which covariation between current and recalled ES is attributable to genetic, shared-, twin-specific-, or unique environmental factors. Table 4 shows the estimates from model fitting, and Fig. 2 displays the best-fitted models and their corresponding path coefficients for covariation between current and recalled ES for both supportive and non-supportive ES.

Table 4

Estimates derived from bivariate models of the covariance between current and recalled measures of supportive and non-supportive parental ES

Variable	Model	df	Δχ²	p	AIC
Supportive ES	ACTE	18		-	9872.7
	ACE	15	9.44	.024	9876.2
	ATE	**15**	**0.02**	**.999**	**9866.7**
	AE	12	9.44	.150	9870.2
Non-sup. ES	ACTE	18		-	9792.3
	ACE	15	0.83	.842	9787.2
	ATE	**15**	3.07	.380	9789.4
	AE	**12**	**3.30**	**.771**	**9783.6**

Note. A = additive genetic factors, C = shared environment factor, T = specific twin shared environment factor, E = unique environment factor; df = degree of freedom, Δχ² = Delta Chi-square statistic, p = significance of χ², AIC = Akaike's Information Criterion. Model with the best fit to the data in bold.

As shown in Table 4, examining the sources of covariation between current and recalled supportive ES, the best-fitted model was an ATE model, with approximately 30% of the covariation explained by genetic factors and roughly 70% attributed to unique environmental factors, and with a minor and non-significant influence from a twin-specific shared environment. For non-supportive ES, the model testing also estimated the AE model to be the best fit, but the relative influence of genetic versus environmental sources shifted; genetic and unique environmental factors were equally important, accounting for 52% and 48% explained variance, respectively. Both best-fitting models omitted the shared environment factor; the addition of C and T did not cause a significant change in Chi-squared values. Compared to the best-fitting univariate models, the bivariate models showed a larger genetic effect for non-supportive ES. Figure 2 displays the path coefficients of the best-fitted models (see Table A3 in the appendix for path coefficients for all models). For supportive ES, there is a correlation between current and recalled ES at .24. For non-supportive ES, this correlation is .27. Figure 3 illustrates how these correlations can be partitioned into genetic and environmental sources, according to the results from our bivariate extended family models.

Note

A1 = genetic effects on recalled and current ES, A2 = genetic effects on current ES, E1 unique environmental effects on current and recalled ES, E2 unique environmental effects on current ES, T1 = twin-specific effect on recalled and current ES, T2 = twin-specific effect on current ES, numbers reflect estimated path coefficients.

Figure 3

Proportions of covariance explained between current and recalled ES; for supportive and non-supportive ES, respectively

Note

The additive genetic contributions are calculated by multiplying the path from the A1 factor to recalled ES with the path from A1 to current ES, while unique-environmental contributions are calculated by doing the same with the paths from E1. The resulting numbers are then divided by the total implied covariance to make percentages. In the case of supportive ES, there is also an influence on covariance from the twin-specific environmental factor, T1. Because T1 reduces covariance, it is not part of the explanation for positive covariance and is therefore not shown in the figure. The unique environmental component includes measurement error.

Discussion

There are three key findings on parental ES in this study: modest to moderate heritability, limited influence of shared environment, and a substantial role for unique environmental factors and measurement error. Genetic factors accounted for more than 20% of the variance in all four forms of parental ES; supportive and non-supportive, and both current and recalled. Testing bivariate models to estimate the sources of the links between current and recalled ES showed that a model with only genetic and unique environmental effects provided the best fit for both supportive and non-supportive ES practices. Our findings align with reviews documenting a substantial role of genetics in parenting (Grigorenko, 2024; Klahr & Burt, 2014), and integrating genetic perspectives into the theoretical framework of parental ES could improve their explanatory precision and power.

The Heritability of Current Parental ES

Within a parent-based design, genetic influences can be interpreted primarily as reflecting the influence of parents' own genes on their parenting behavior (Klahr & Burt, 2014; Kretschmer, 2023). These genetic effects may operate through psychological traits such as temperament, cognition, personality, emotion regulation, or liability for psychopathology, all of which are shown to predict parenting behavior (for a review, see Taraban & Shaw, 2018). More specifically, research has demonstrated links between parental ES and emotion regulation (Hajal & Paley, 2020), as well as personality (Hughes & Gullone, 2010). However, the observed genetic effect may also reflect indirect mechanisms. Genes and environment are intertwined in complex processes involving both gene-environment correlations (*r*GE) as well as gene-environment interactions (GxE) (Plomin et al., 1977). Even intuitively "environmental" factors, like income, education, and employment, can be genetically confounded (K. S. Kendler & Baker, 2007). From a lifespan developmental perspective, small genetically based individual differences in psychological traits can be reinforced and become larger differences, partly attributed to the interplay between genes and the environment (Knafo & Jaffee, 2013).

The genetic effect observed in current parental ES may partly reflect an active *r*GE process, whereby an individual seeks out environments that align with their genetic dispositions. For example, a parent with genes predisposed to warmth may seek out friends and partners who are high in warmth, thereby facilitating the development of a supportive parenting style. Furthermore, a parent high in negative emotionality may have a child who inherits the same disposition, evoking more non-supportive parenting behavior (evocative *r*GE). Such “child effect” (Kiff et al., 2011) on parenting has been documented in meta-analysis of genetic studies of children and mothers (Avinun & Knafo-Noam, 2014; Knafo & Jaffee, 2013), and illustrates indirect genetic effect, whereby parental ES is shaped by another person's genotype through an environmental pathway (McAdams et al., 2023). In this parent-based twin design, child-specific evocative effects that make MZ parents differ in their parental ES would contribute to the unique environment component, whereas child effects that systematically covary with parental genetic similarity may contribute to the genetic component; the present design cannot disentangle these processes.

Consistent with prior studies (e.g., Euser et al., 2020; Oliver et al., 2014), our findings showed a tendency toward higher heritability estimates for non-supportive parenting compared to supportive parenting, across both current and recalled ES. This pattern could suggest differences in the relative contributions of genetic and environmental influences to supportive versus non-supportive ES; for instance, non-supportive ES could be more closely linked to genetically influenced dispositions, such as temperament and personality, whereas supportive ES may be relatively more influenced by environmental experiences. However, as confidence intervals overlapped, the findings must be interpreted with caution. Notably, Klahr and Burt's (2014) meta-analysis reported the opposite pattern, with a stronger genetic basis for supportive compared to non-supportive parenting. Future studies are needed to reconcile these discrepancies and clarify whether the etiology of parental ES varies across supportive and non-supportive ES.

Different Heritability Patterns for Current versus Recalled Parental ES

The differing relative contributions of genetic versus unique environmental factors to current versus recalled ES suggest that individual differences in recalled ES have higher heritability than individual differences in current ES. Twin correlations were also generally higher for reports of childhood experiences of parenting than for reports of their own parenting. This pattern aligns with Klahr and Burt's (2014) review, which concluded that genetic effects tend to be larger and shared environmental effects smaller when heritability is estimated from adult twins´ retrospective reports on the parenting they received as children, compared to estimates based on reports on their current parenting (Klahr & Burt, 2014).

The interpretation of this finding depends on how recalled ES is conceptualized. One possibility is to treat a retrospective report as a summary of essential aspects of actual exposure to parenting behavior, capturing broader trends in parenting behavior over time (Klahr & Burt, 2014). Under this assumption, both current and recalled ES reflect actual parenting behavior, and the findings could indicate a partially different etiology of ES received in the family of origin compared with current ES, potentially involving child effects such as evocative gene-environment correlations.

However, the difference in heritability between current and recalled ES reports may reflect the nature of retrospective reports rather than true differences in the etiology of parenting behavior. Reports on parenting behavior are grounded in fundamentally different perspectives: recalled ES refers to parenting received as a child (child perspective), whereas current ES refers to one's own behavior toward a child (parent perspective). Memories of childhood experiences are reconstructions of events and can change with later experiences, new information, and subjective interpretation (Fivush, 2008). Such interpretive processes may themselves be affected by genetically influenced personality traits. In contrast, reports of current behavior are anchored in parents' behavior toward their child in the present and may be more directly influenced by contemporary contextual factors (e.g., stress, momentary internal states). From this perspective, the higher heritability of recalled ES may partly reflect genetic influences on how participants remember, perceive, and interpret parenting, rather than differences in actual parenting behavior alone.

Together, these considerations suggest that the different patterns in the etiology of current versus recalled ES should not be interpreted simply as evidence that parental ES in the family of origin is more strongly genetically determined. Rather, they highlight the need to distinguish between genetic influences on actual parenting and genetic influences on perception and recall of parenting, and point to the need for further examination and methodological triangulation.

The Significance of the Unique Environment

While a genetic factor was evident, unique environmental influences accounted for the largest proportion of variance, particularly for current supportive and current non-supportive ES (66% and 72%, respectively). These estimates must be interpreted with caution, as we do not know to what extent these estimates reflect measurement error. Nonetheless, the findings are supported by consistency with similar patterns observed in other parenting dimensions, such as warmth, control, and negativity (Klahr & Burt, 2014), suggesting a critical role for unique environmental experiences in shaping parental behavior beyond genetics and shared environmental influences. In a parent-based twin design, unique environmental factors encompass individual life experiences (e.g., work conditions, romantic relationships, health events), and the characteristics and behaviors of their children. These factors can shape how parents respond to children's emotions and may differ even between genetically identical twins.

As discussed above, child effects on parenting are well-documented. Genetically influenced differences between children may elicit distinct parenting responses, either amplifying or reducing the similarity between adult twins. Some studies have documented differential parenting for monozygotic twins. For instance, aiming to explore the unique environment for twins, Deater-Deckard and colleagues (2001) observed the parenting of 62 pairs of MZ twins and found significant differences in maternal negativity and harsh discipline within the twin pairs. While not addressing causality, such findings illustrate that even genetically identical twins may experience different parenting, which may contribute to unique environmental influences.

A Minor Effect of the Shared Environment

Observing a minor effect of the shared environment in the form of a twin-specific factor for current and recalled ES but limited to supportive ES, requires careful interpretation. First, it suggests a small twin-specific shared environmental effect that can be distinguished from a general household effect. One possible explanation is that being in the same place at the same time may drive some similarity in the development of supportive ES, possibly reflecting that parents change over time. Second, considering that different patterns for supportive and non-supportive ES have been frequently observed in prior studies (Eisenberg, 2020), the finding of separate patterns for supportive and non-supportive across both current and recalled ES, is consistent with the idea that these two constructs are not merely opposite ends of a bipolar spectrum but are instead separate constructs. Third, given that it is only a minor effect, it could suggest that the environments shared by siblings growing up in the same family have minimal influence on the development of parental ES. However, this does not mean that parenting and shared family factors (e.g., divorce or parenting behavior) have no impact on child development. Shared family experiences can markedly influence development, but if they are uniquely experienced, they may increase differences between siblings rather than similarity and thus contribute to the unique environmental component (Plomin, 2023; Plomin et al., 2016). Gene-environment interactions (GxE) may help explain how the impact of the same environmental factor can differ across genotypes, reflecting genetic sensitivity to the environment (Plomin et al., 1977). As proposed by differential susceptibility theory (Belsky et al., 2022; Belsky & Pluess, 2009), some children may, due to their genotype, be more sensitive to dismissive and punitive parental responses than others. These processes help explain how similar childhood events may lead siblings down different developmental paths, resulting in differences in parenting as adults.

The findings thus suggest a high degree of individualization in how parents develop ES, reflecting differences in individual experiences and reactions to similar family events and daily parenting routines. The notion of subjective environments highlights the complexity of environmental influences on parental behavior.

The Intergenerational Transmission of Parental ES

The study's findings align with previous research on the continuity of parenting practices across generations (Kerr & Capaldi, 2019); both supportive and non-supportive ES showed a modest but positive correlation between current and recalled ES practices. As illustrated in Fig. 3, genetic influences accounted for a moderate portion of the covariation. However, the unique environmental factor was the primary contributor to covariation in supportive ES, whereas for non-supportive ES, genetic and unique environmental factors were equally influential.

Observing a genetic factor explaining parental ES in the univariate models, it might appear self-evident or even tautological to find a genetic factor that explains the covariance between current parental ES and the same parental ES experienced in childhood. However, although it seems likely that this covariation also has a genetic component, it is not necessarily the case. The covariance of current and recalled ES can have a different etiology than the heritability of each trait considered separately (Knopik et al., 2017).

Recalled ES is a measure of the parents' recollection of parental ES in the family and is not an objective measure of parental ES in the family of origin for all children in the family, as illustrated by the difference in

ratings between MZ twins. Parenting is the outcome of a reciprocal process between the parent and the specific child rather than a fixed characteristic of the parent. From this perspective, one possible interpretation of the genetic component explaining the covariance is that the overlapping genetic influences may shape both their perceptions of how they were parented and their own parenting practices. The differential-susceptibility theory (Belsky & Pluess, 2009) further explains how children can vary in their sensitivity to parenting, both positive and negative. For example, a sensitive child might perceive parents as harsher than a less sensitive sibling and be more strongly affected by these experiences. Such genetically influenced sensitivity may contribute to later differences in parenting behavior. Thus, a genetic factor does not imply that parenting behavior is directly passed down, but rather that underlying traits may contribute to continuity across generations. The genetic effect could also reflect a genetically influenced response style, in which a parent tends to respond similarly across questionnaires and over time (for example, consistently endorsing items more negatively), thereby inducing a correlation between current and recalled ES rapport independently of any actual continuity in ES practices.

For supportive ES, genetic influences were not the primary source of covariation; rather, the unique environment was the primary factor explaining the link between recalled and current ES practices. The unique environmental sources of covariance, as shown in Fig. 3, must, however, be interpreted with caution, as they might partly reflect correlated measurement error.

In summary, these findings suggest that underlying genetic dispositions play an important role in explaining the observed continuity of parental ES strategies across generations. Genetic dispositions in temperament, personality, and emotion regulation may shape both how parents experienced and recalled the parental ES practice they received and how they themselves practice ES later in life.

Strengths and Limitations

The present study expands the literature on parental ES by examining genetic influences using an extended twin design. The findings are strengthened by a population-based sample and the inclusion of both mothers and fathers. However, equally important as interpreting what the study indicates is recognizing what it does not imply. First, the heritability estimates do not provide information about the genetic influence on an individual level. For example, when current supportive ES has a heritability estimate of 0.20, it does not mean that a parent's current ES is 20% genetically determined; it means that 20% of the individual variation within the sample is attributable to genetic differences between individuals in that population. Second, heritability and environmental estimates are specific to a particular sample at a particular time; they cannot be easily generalized to other populations (McGuire et al., 2012). Their precision varies depending on sample size and measurement error (Knopik et al., 2017). Currently, research on parenting and genetics mainly involves samples from WEIRD populations (Western, Educated, Industrialized, Rich, and Democratic countries) (Grigorenko, 2024; McAdams et al., 2023).

Third, the confidence intervals for the intraclass twin correlations indicate uncertainty about the observed differences between MZ and DZ twins and, consequently, about the estimated variance, suggesting that the findings should be interpreted with caution. Further, the heritability estimates in twin studies are generally based on several assumptions that may affect the results, including the equal environments

assumption for MZ and DZ twins and the absence of assortative mating. Unmodeled gene-environment correlation or interaction may also influence the partitioning of genetic and environmental influences (for discussions on the assumptions underlying twin design in general, see e.g. Conley et al., 2013; Knopik et al., 2017; McAdams et al., 2023) along with methodological challenges associated with extended designs in particular (Keller et al., 2010).

Fourth, all measures in this study rely on self-report, which may limit the breadth of insights (Morris et al., 2022), and introduce bias related to individuals' ability to summarize behavior that varies across situations, as well as social desirability effects (Paulhus, 1984). The measurement of parental ES in the family of origin is especially vulnerable to distortion because it requires recalling events from many years ago. Memories are not snapshots of the past, but reconstructions influenced by the person's personality and interpretation, as well as the mood state at the time of reporting (Fivush, 2008). Fifth, a narrow operationalization of supportive ES and a relatively large span in child age may affect comparisons with other studies on supportive ES and the generalizability of the results. Sixth, the heritability estimates should not be misunderstood as suggesting that a certain portion of parental behavior is predetermined, nor as providing evidence about underlying causal effects. Genetic influences on parenting can vary depending on environmental factors, such as gene-environment interactions, which can either enhance or reduce their expression.

Implications and Future Research

The findings of this study suggest that theoretical models of parental ES should be expanded to explicitly incorporate genetic factors as an important source of individual differences. At the same time, the important role of unique environmental influences highlights the importance of exploring psychological aspects, particularly individuals' specific experiences and subjective perceptions of past and present events (Plomin, 2023). Additionally, distinct patterns observed for supportive and non-supportive ES support studying them as separate dimensions, improving our understanding of the specific impacts and mechanisms associated with each behavior.

Finding a combination of minimal shared environmental influence and moderate genetic effects relates to Plomin's (2011) description of the implications of behavioral genetic research as a "gloomy prospect". The "gloomy prospect" refers to the idea that the vast majority of environmental influences on an individual's development likely to be idiosyncratic, unpredictable, or even random (Plomin & Daniels, 2011). However, rather than adopting a bleak view, this perspective may reorient research efforts toward more personalized approaches to development, capturing the totality of rare and idiosyncratic environments within whole environmental domains (Plomin, 2023). Highlighting the influence of unique environmental factors on parental ES can support the relevance of parenting interventions aimed at modifying behaviors. Effective parental interventions should help parents understand how individual experiences and characteristics shape their parenting, enabling them to identify strategies that foster adaptive emotional interactions with their children. This perspective counters pessimistic views of genetic determinism, advocates for more individualized and potentially impactful strategies (Plomin, 2023), and aligns with the emerging field of "personalized medicine" (Goetz & Schork, 2018), suggesting the potential for more personalized parenting

support. Additionally, recognizing that some parents may face greater challenges related to genetically influenced dispositions strengthens the case for a differentiated approach and may reduce parent blame while shifting focus to building skills and support systems that compensate for individual vulnerabilities.

Future research on the origins and intergenerational transmission of parental ES should address the content of unique environments and clarify the impact of measurement error. To understand the underlying mechanisms of gene-environment interplay, researchers need to incorporate different and repeated measures with longitudinal data spanning multiple generations. Creating research with parent-based twin designs and large population-based samples is logistically challenging and expensive, but provides valuable opportunities to understand the origins of parenting behavior (e.g. Branje et al., 2020). Triangulating findings across methods remains essential. The complex interplay between genes and environment makes a precise calculation of the heritability of parenting seem unrealistic (Scarr & McCartney, 1983). However, recent advances that integrate extended-family designs with genomic data now provide increasingly sophisticated tools for disentangling the genetic and environmental contributions to parental ES (McAdams et al., 2023). Future research might also broaden the scope to include other family members (such as partners and in-laws) to account for assortative mating and examine how parenting is shaped by family dynamics and diverse environmental factors (e.g., public services and neighborhood conditions).

Declarations

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Article information

Conflict of Interest Disclosures

Each author signed a disclosure form regarding potential conflicts of interest. None reported any financial or other conflicts related to the work.

Ethical Principles

The authors confirm they followed established professional ethical guidelines in the preparation and conduct of this study. These guidelines include securing informed consent from all participants, treating subjects ethically, respecting their rights, and safeguarding participant privacy and data security. Specifically, ethical approval for the PPN project was granted by the Regional Committees for Medical and Health Research Ethics (REK-2022/346141). An evaluation of privacy implications was conducted in collaboration with the Norwegian Center for Research Data (NSD-2022/584188) and received approval

from the University of Oslo's Head of Department and Data Protection Office. All participants provided electronic informed consent prior to their involvement. The data were anonymized and securely stored within the Services for Sensitive Data (Tjeneste for Sensitive Data—TSD), a dedicated cloud storage and computing environment owned and managed by the IT Department of the University of Oslo, designed for handling sensitive research data. Participants were compensated with a universal gift card for their participation.

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Data Availability Statement

The data, materials, and codes necessary to reproduce the analyses presented are available from the corresponding author upon request and ethical approval. Analyses presented here were not preregistered, but preregistration for the main project is available here: https://osf.io/k7ude.

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Figures

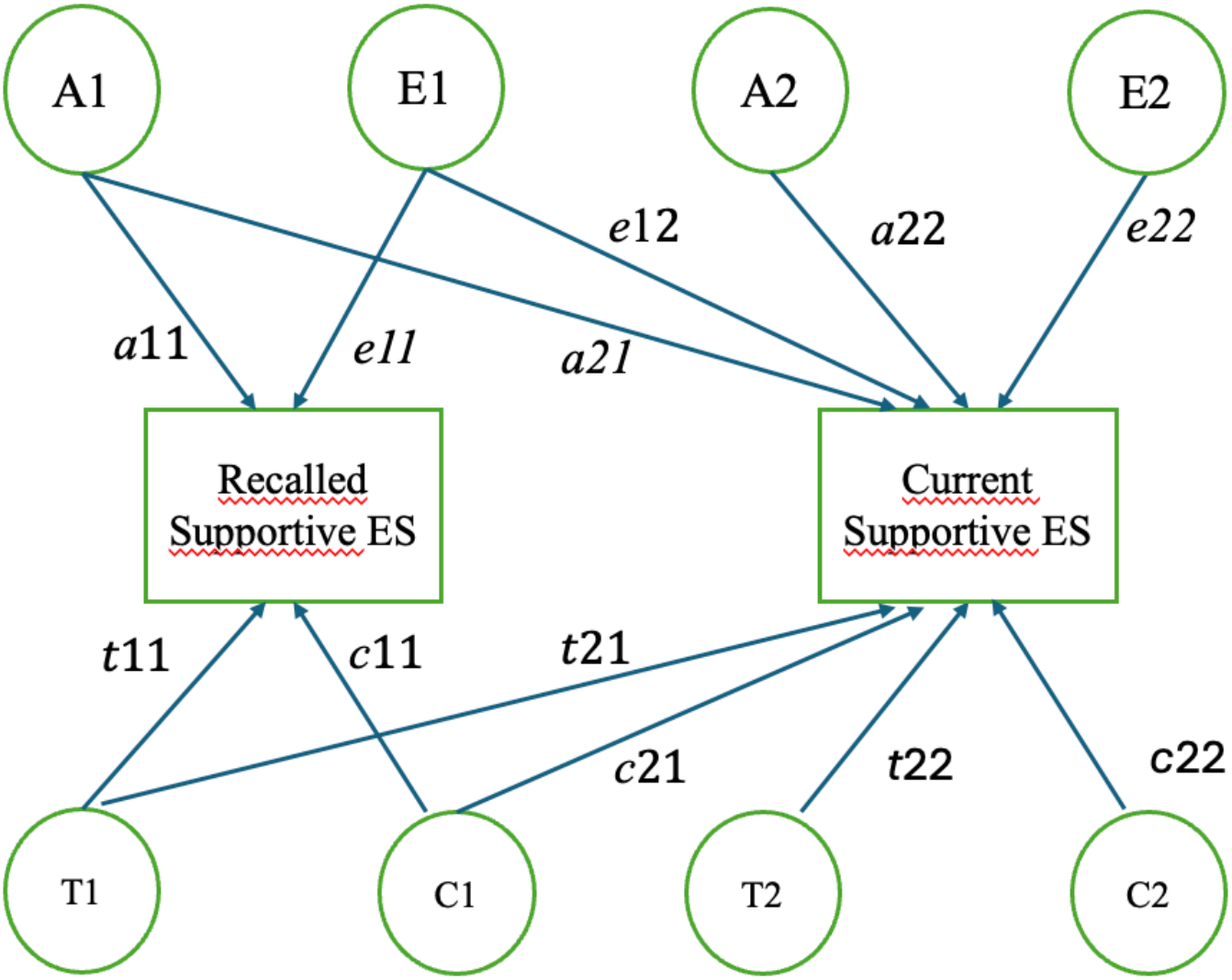


Figure 1

An illustration of the bivariate ACTE model with path coefficients used in the current study.

Note: Measured variables are depicted as rectangles, and a latent variable as an oval. A = Additive genetic factors, C = Shared environmental effects, T = twin-specific shared environment, E = Unique environmental effects. *a*11, *c*11, t11, *e*11... = path coefficients for additive genetic influence, shared environmental influence, twin-specific shared environment and unique environmental influence, respectively.

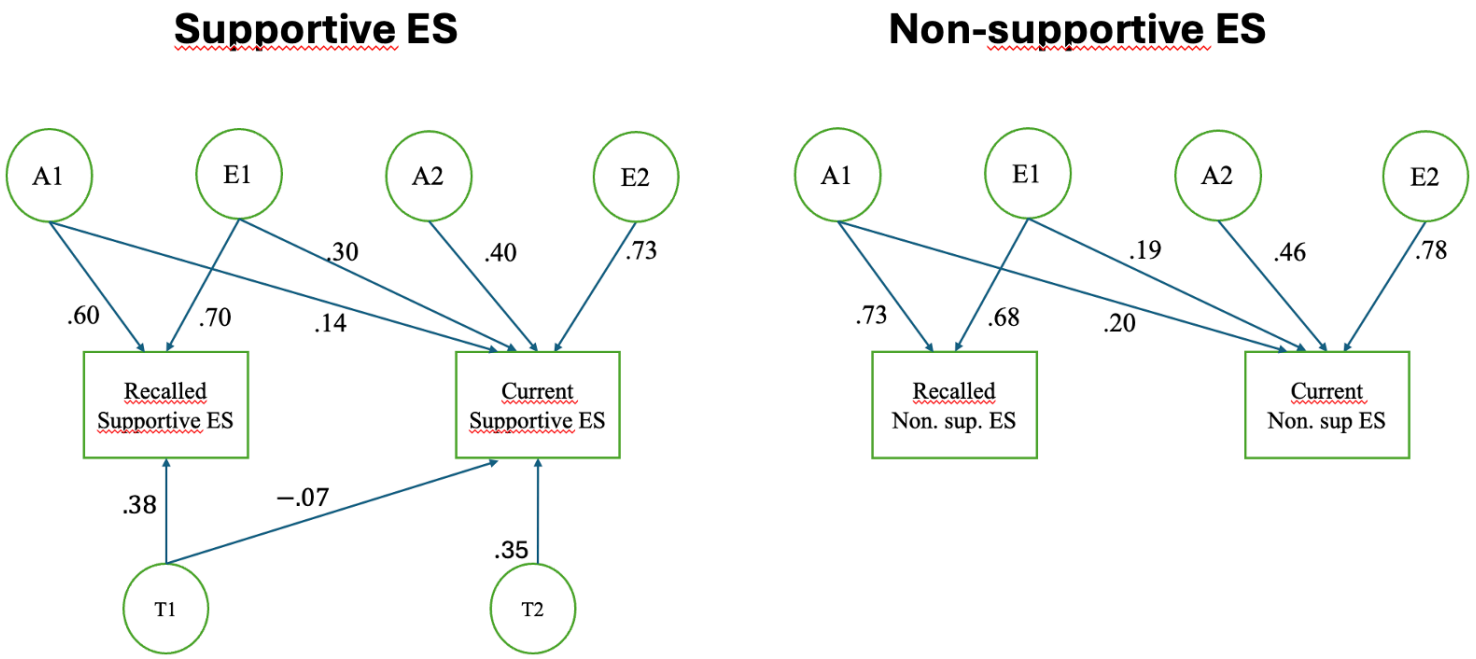


Figure 2

Estimates for best-fitting bivariate models for the association between recalled and current ES, for supportive and non-supportive ES

Note: A1 = genetic effects on recalled and current ES, A2 = genetic effects on current ES, E1 unique environmental effects on current and recalled ES, E2 unique environmental effects on current ES, T1 = twin-specific effect on recalled and current ES, T2 = twin-specific effect on current ES, numbers reflect estimated path coefficients.

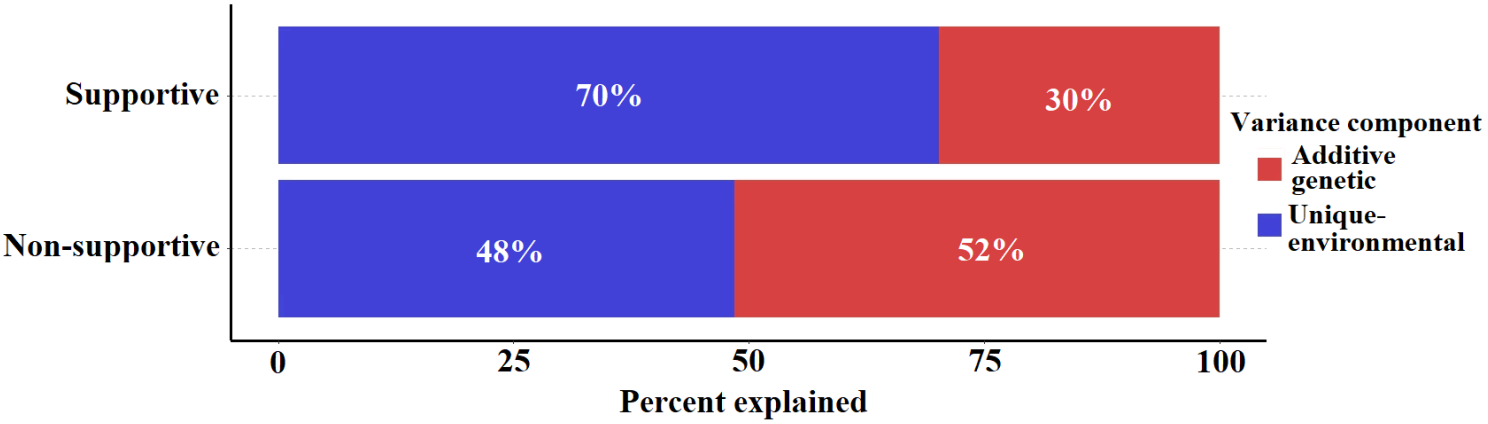


Figure 3

Proportions of covariance explained between current and recalled ES; for supportive and non-supportive ES, respectively

Note: The additive genetic contributions are calculated by multiplying the path from the A1 factor to recalled ES with the path from A1 to current ES, while unique-environmental contributions are calculated by doing the same with the paths from E1. The resulting numbers are then divided by the total implied covariance to make percentages. In the case of supportive ES, there is also an influence on covariance from the twin-specific environmental factor, T1. Because T1 reduces covariance, it is not part of the explanation for positive covariance and is therefore not shown in the figure. The unique environmental component includes measurement error.

Supplementary Files

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- Appendix.docx