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Special Issue Article

The role of parenting in the intergenerational transmission of executive functioning: A genetically informed approach

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Abstract

Deficits in executive functioning both run in families and serve as a transdiagnostic risk factor for psychopathology. The present study employed twin modeling to examine parenting as an environmental pathway underlying the intergenerational transmission of executive functioning in an at-risk community sample of children and adolescents ($N = 354$ pairs, 167 monozygotic). Using structural equation modeling of multi-informant reports of parenting and a multi-method measure of child executive functioning, we found that better parent executive functioning related to less harsh, warmer parenting, which in turn related to better child executive functioning. Second, we assessed the etiology of executive functioning via the nuclear twin family model, finding large non-shared environmental effects ($E = .69$) and low-to-moderate heritability ($A = .22$). We did not find evidence of shared environmental effects or passive genotype–environment correlation. Third, a bivariate twin model revealed significant shared environmental overlap between both warm and harsh parenting and child executive functioning (which may indicate either passive genotype–environment correlation or environmental mediation), and non-shared environmental overlap between only harsh parenting and child executive functioning (indicating an effect of harsh parenting separable from genetic confounds). In summary, genetics contribute to the intergenerational transmission of executive functioning, with environmental mechanisms, including harsh parenting, also making unique contributions.

Keywords: harsh parenting; nuclear twin family model; warm parenting

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Deficits in executive functioning, a set of top-down processes that involve regulation of goal-directed behavior, including processes like inhibitory control, working memory, and cognitive flexibility (Diamond, 2013), have long been linked to various forms of psychopathology (Bloemen et al., 2018; Wright et al., 2014), including attention-deficit/hyperactivity disorder (Castellanos et al., 2006; Cherkasova et al., 2013), antisocial behavior (Moffitt, 2018), substance abuse (Smith et al., 2014; Verdejo-Garcia et al., 2008), and depression (Gotlib & Joormann, 2010). Executive functioning runs in families, such that parents with better executive functioning skills tend to have children with better executive functioning skills (Deater-Deckard, 2014; Jester et al., 2009). However, much observational work does not disentangle the genetic and nongenetic influences on individual differences in executive functioning (Cuevas, Deater-Deckard, Kim-Spoon, Wang, et al., 2014), and genetically informed work is needed to clarify the mechanisms that contribute to its intergenerational transmission.

Parenting behaviors are one proposed mechanism underlying the intergenerational transmission of executive functioning. There is substantial evidence that parenting behaviors like warmth and scaffolding promote better executive functioning development, whereas behaviors like harshness and negative control impede executive functioning development (Fay-Stammbach et al., 2014; Hughes & Devine, 2019; Li et al., 2019). Parent executive functioning is thought to impact parenting (and the broader home environment), which in turn, impacts child development (e.g., Distefano et al., 2018; Kao et al., 2018; Korucu et al., 2020). For example, a parent with better executive functioning skills may have a better ability to utilize warmer and less harsh parenting behaviors, promoting better executive functioning development in their child. However, with some notable exceptions (Bridgett et al., 2018; Cioffi et al., 2020), the majority of developmental studies linking parent executive functioning to child executive functioning via parenting behaviors are not genetically informed.

A separate line of research implicates genetic influences as a primary contributor to the intergenerational transmission of executive functioning. Performance on executive functioning tasks is moderately heritable, though with evidence of moderate non-shared environmental influences (Gagne & Saudino, 2010; Li & Roberts, 2017; Vasilopoulos et al., 2012). The heritability of task-general executive functioning, often indexed via latent modeling, is thought to be much higher (Friedman & Miyake, 2017), with estimates as high as 99% (Friedman et al., 2008) or 100% (Engelhardt et al., 2015). The high heritability of latent executive functioning also appears to be consistent across development, from childhood into adulthood (Friedman & Miyake, 2017).

Given these heritability estimates, it is possible that observational studies linking parenting to executive functioning are

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actually capturing unmeasured genetic processes. That is, because parents and children share genes, associations in which parenting mediates the relationship between parent and child executive functioning could be driven by *genotype–environment correlation,* or the correlation between the parents' (and thus children's) genetic tendencies and the environments the children encounter (Deater-Deckard, 2014; Knopik et al., 2017; Manuck & McCaffery, 2014). Two types of genotype–environment correlation are particularly important for this issue: *passive* and *evocative genotype–environment correlation* (see Manuck & McCaffery, 2014 for a discussion). First, passive genotype–environment correlation emerges because the environment parents provide to their biological children is influenced by the parents' genotype. Because parents share genes with their biological children, the child's genes are necessarily correlated with their environmental experiences. In this particular case, parent executive functioning affects the family environment via parenting (Deater-Deckard, 2014). For example, parents with worse executive functioning react more harshly to misbehavior (Bridgett et al., 2017; Deater-Deckard et al., 2010). Thus, though parents may have passed down genetic risk for executive functioning, it may appear as though parenting (which is correlated with parent and child executive functioning) is the cause of poor executive functioning in the child. Second, evocative genotype–environment correlation includes the ways in which children elicit reactions from their environment that are consistent with their genetic tendencies. In this case, children with a genetic predisposition for lower executive functioning could exhibit behaviors that evoke harsher parenting. Put another way, parenting would be a consequence of child executive functioning, such that the genetic influences on executive functioning would correlate with those on parenting. Consistent with this idea, previous adoption work has found evidence for an evocative genotype–environment correlation in which higher levels of child anger evokes harsher parenting from adoptive mothers (Bridgett et al., 2018). Thus, some nongenetically informed studies may implicate parenting as a causal mechanism when these associations are actually a function of passive or evocative genotype–environment correlation.

Fortunately, there are an array of twin modeling approaches that can leverage the genetic relatedness of twins to elucidate the role of genotype–environment correlation. First, the nuclear twin family model (Burt & Klump, 2012; Keller et al., 2010) leverages similarities between twins and their biological parents to determine the etiology of a given phenotype. This model includes parent measures of the phenotype of interest to provide several advantages over the classical twin design, including the ability to divide shared environmental influences (C in the classical twin model) into those shared by siblings (S; "sibling-level shared environmental influences") and those shared by twins and their parents (F; "family-level shared environmental influences"; Burt & Klump, 2012; Keller et al., 2010). Sibling-level shared environmental influences (S) include anything that makes siblings more similar to each other, but not to their parents, such as shared school environments, shared parenting experiences, and shared peers (Burt & Klump, 2012; Keller et al., 2010). Family-level shared environmental influences (F), on the other hand, include influences that make twins and their parents more similar to each other, such as shared cultural influences, family socioeconomic status, and broader societal norms (Burt & Klump, 2012; Keller et al., 2010). Additionally, this model can quantify passive genotype–environment correlation, calculated as the covariance between additive genetic (A) and family shared environmental effects (F; Burt & Klump, 2012; Keller et al., 2010). To complement the use of the nuclear twin family model, a bivariate ACE model can decompose the association between parenting and child executive functioning into genetic (A), shared environmental (C), and non-shared environmental (E) components, providing insight into the genetic and environmental origins of their association. Because genetic influences (A) on parenting in a child twin design index the extent to which twins' genetic tendencies affect the parenting they receive (Klahr & Burt, 2014), the presence of a genetic correlation would likely index a role of evocative genotype–environment correlation. Shared environmental influences (C) on parenting in a child twin design could index either shared environmental influences or passive genotype–environment correlation, but a limitation of this modeling approach is that the two cannot be distinguished (Neiderhiser et al., 2004). Moreover, the bivariate modeling approach can test whether there are shared or non-shared environmental influences that overlap between parenting and child executive functioning, which would support the notion that some of the association between parenting and child executive functioning is environmental in origin. Thus, between these two modeling approaches, both passive and evocative genotype–environment correlation can be directly measured. We are also able to clarify whether there are environmental mechanisms through which parenting impacts child executive function.

In sum, these complementary modeling approaches can clarify the role that various forms of genotype–environment correlation play in the complex relationship between parent executive functioning, parenting, and child executive functioning. In doing so, however, it would be important to attend to a few key issues: First, it is important to consider the reliability and validity of frequently used executive functioning measures (Eisenberg et al., 2019; Karr et al., 2018). The metrics traditionally extracted from behavioral executive functioning tasks suffer from test–retest reliability issues, do not consistently relate to real-world outcomes such as mental health, physical health, or income, and do not load into a consistent factor structure (Eisenberg et al., 2019; Enkavi et al., 2019; Hedge et al., 2018; Karr et al., 2018; Rouder & Haaf, 2019; Weigard et al., 2021). One emerging and promising alternative to traditional behavioral measures is to use computational cognitive models to capture the processes underlying performance across a variety of tasks that engage executive functioning skills (Weigard & Sripada, 2021). Evidence accumulation models (Brown & Heathcote, 2008; Evans & Wagenmakers, 2020; Ratcliff et al., 2016), which are widely used to model choice response time tasks (including many executive functioning measures) suggest that a single higher-order individual difference dimension drives performance across many tasks: the efficiency with which individuals can gather evidence for correct choices in the context of background noise (Lerche et al., 2020; Schmiedek et al., 2007; Weigard et al., 2021). "Efficiency of evidence accumulation", as indexed by these computational models, exhibits good test–retest reliability (Lerche & Voss, 2017; Schubert et al., 2016) and outperforms traditional executive functioning metrics both in temporal stability and in prediction of individuals' self-regulation (Weigard et al., 2021). In addition to using computational models of behavioral performance, behavioral measures can be complemented via the inclusion of survey measures. Though often weakly correlated with these behavioral measures of executive functioning (Dang et al., 2020), self-report, survey measures of executive functioning show stronger associations with real-world outcomes than do behavioral measures (Eisenberg et al., 2019). Thus, to maximize relevance to real-world outcomes, the present study combines computational and self-report measures of child executive functioning.

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Second, much of the extant work focuses on infants and young children (e.g., Bernier et al., 2010, 2012; Blair et al., 2014; Bridgett et al., 2018; Broomell et al., 2020; Cioffi et al., 2020; Distefano et al., 2018; Hammond et al., 2012; Helm et al., 2020; Hughes & Devine, 2019; Kao et al., 2018; Korucu et al., 2020; Zeytinoglu et al., 2017). Early childhood is indeed an important time period to study, as parenting influences may be especially important for young children (Gee, 2016; Shaw & Bell, 1993) and the prefrontal cortex and associated executive functioning skills are rapidly developing during this time (Kolb et al., 2012; Zelazo & Carlson, 2012). However, prefrontal cortex development continues through later childhood and adolescence (Kolb et al., 2012) and adolescence is a peak period for the onset of disorders associated with executive functioning difficulties (Cherkasova et al., 2013; Moffitt, 2018). Given evidence that parenting is related to executive functioning and related constructs during adolescence (Li et al., 2019), work is needed that considers the role of parenting on the intergenerational transmission of executive functioning in later childhood and adolescence as these skills are refined and begin to approach adult levels.

Thus, the present study examined the roles of shared genes and parenting in the intergenerational transmission of executive functioning in a large, socioeconomically diverse sample of children and adolescents. We utilized behavioral and self- and parent-report measures of executive functioning for twins and their parents from the Michigan Twin Neurogenetics Study (MTwiNS), a unique longitudinal study of twins ($N = 708$ in 354 pairs) that were oversampled for residence in low-income neighborhoods. For our first aim, we assessed whether harsh or warm parenting were phenotypically related to a computationally derived, task-general measure of executive functioning. We then tested whether parent executive functioning was associated with child executive functioning via parenting behaviors. We hypothesized that warmer, less harsh parenting would be associated with better child executive functioning. For our second aim, we assessed the etiology of executive functioning via the nuclear twin family model. Using the nuclear twin family model, we assessed the role of sibling-level and family-level influences on executive functioning and quantified the role of passive genotype–environment correlation. We hypothesized that executive functioning would be moderately heritable, and that there would be evidence of passive genotype–environment correlation, reflecting that the genes underlying parent executive functioning relate to the general family environment, including the overall parenting environment. For our third aim, we employed a bivariate twin model to examine the etiology of the phenotypic association between parenting and executive functioning. Our hypotheses for this aim were informed by results of Bridgett et al. (2018), an adoption study which, by design, eliminated the impact of passive genotype–environment correlation. This study found evidence of evocative genotype–environment correlation (i.e., child anger evoked harsher parenting) and an environmental effect of parenting on child self-regulation after controlling for evocative genotype–environment correlation. Thus, we hypothesized that there would be some environmental influence of parenting on child executive functioning, as well as evidence of evocative genotype–environment correlation.

Method

Participants

The present study included data from families in the MTwiNS, a project within the Michigan State University Twin Registry (MSUTR; Burt & Klump, 2019). The 354 families participating in MTwiNS were originally identified through birth records as part of the Twin Study of Behavioral and Emotional Development in Children (TBED-C; for details see Burt & Klump, 2019) and recruited into one of two cohorts – a population-based cohort that represented families living within 120 miles of Michigan State University and an at-risk cohort recruited from the same area, but that was restricted to families living in U.S. Census tracts where at least 10.5% of families lived below the poverty line (the mean for the state of Michigan at the time; Burt & Klump, 2019). MTwiNS participants were re-recruited from the original participant pool based upon criteria for the second, "at-risk" cohort. Thus, MTwiNS includes families from the "at-risk" cohort as well as those from the population-based cohort that would have qualified for the "at-risk" cohort. This re-recruitment strategy yielded a sample representative of families living in neighborhoods with above average levels of poverty, a unique sampling frame within both the behavioral genetics and neuroimaging literatures (Burt et al., 2021).

The 708 twins (354 pairs, 167 monozygotic [MZ]) included in the present study were 7–19 years old (Mean = 14.6, $SD = 2.2$; 54.5% male; only 3.3% of the present sample was 10 or younger). The breakdown of twins' parent-reported ethnicity reflected the surrounding area (78% White/Caucasian, 13% African-American, 5% Other, 1% Latino/Latina, 1% Pacific Islander, 1% Native American, <1% Asian). Median reported family annual income for this sample was $70,000 to $79,999 and ranged from less than $4999 to greater than $90,000. 9% of included families reported an annual income below the 2020 federal poverty line of $26,246 per year and 33% reported annual income below the living wage for a family of 4 in Michigan (http://livingwage.mit.edu/states/26). Zygosity was established using physical similarity questionnaires administered to the twins and/or their parents (Bouchard et al., 1990; Iacono et al., 1999; Peeters et al., 1998). Discrepancies were resolved through review of zygosity items or by DNA markers. Parents provided informed consent and children provided assent.

Procedure

Primary caregivers completed executive functioning self-report measures during their preliminary visit to Michigan State University as part of their participation in the TBED-C (twin ages ranged from 6 to 11 at the time; Mean = 8.05; $SD = 1.43$). All other measures were collected in a day-long return visit to the University of Michigan (twin ages at follow-up ranged from 7 to 19 as described above). Each twin completed a variety of behavioral tasks, some of which occurred during an magnetic resonance imaging (MRI) scan, including a go/no-go task (during imaging) and a stop-signal task (in the lab). Twins also completed a battery of child-report questionnaires. Primary caregivers completed a demographic interview with an examiner and a battery of self- and parent-report questionnaires.

Measures

Child executive functioning

Go/no-go task. The child friendly go/no-go task used in this study was adapted from Casey et al. (1997) and assesses inhibition via a "whack-a-mole" game (stimuli courtesy of Sarah Getz and the Sackler Institute for Developmental Psychobiology; task downloaded from http://fablab.yale.edu/page/assays-tools). In the present task, participants were instructed to press a button as quickly as possible in response to one stimulus ("go", a mole)

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and avoid responding to a less frequent nontarget ("No-Go", a vegetable). The target stimuli (moles) were modified with various "disguises" to make the task more interesting and difficult given the relatively slow speed of stimuli used to accommodate functional magnetic resonance imaging (fMRI) data acquisition. The task consisted of four runs, each with approximately 55 trials, for a total of 255 trials of which 55 were no-go (21.6% no-go). Each no-go trial was preceded by 1–5 go trials. Each trial lasted 2300 ms, including a maximum of 1800 ms stimulus presentation, 400 ms feedback, and 100–1000 ms fixation to account for individual differences in reaction time. Participants practiced the task briefly in an MRI simulator before the MRI scan. Participants with below-chance "go accuracy" (correct responses on <55% of "go" trials) were not considered to be meaningfully participating in the task ($N = 2$), leaving $N = 589$ individuals with viable go/no-go data.

Stop-signal task. The child friendly stop-signal task used in this study was a 10 minute, 150 trial task adapted from Bissett & Logan (2012) as described previously (Begolli et al., 2018). Participants were presented with a fish for 850 ms and told to push the "a" or "l" keys as quickly as possible based on the orientation of the fish, unless a visual stop-signal stimulus (Martin the Manta Ray) appeared on the screen, which occurred on 50 of the task trials (33%; "stop" trials). This stop-signal was presented following a stop-signal delay (SSD) that was determined through a standard "staircase tracking" algorithm (Logan, 1994) designed to lead to a roughly 50% probability of inhibition on "stop" trials for each participant. This algorithm began with a 250 ms SSD and was thereafter increased or decreased by 50 ms on each subsequent "stop" trial based on whether the participant was able to successfully inhibit. The stop-signal task was added to the protocol partway through data collection and was therefore only available for a subset of participants ($N = 332$ individuals).

Evidence accumulation model analyses. Parameters of the linear ballistic accumulator (LBA) model were estimated in an individual-level Bayesian framework for the go/no-go task and "go" choice trials from the stop-signal task using the Dynamic Models of Choice functions (Heathcote, 2019), an adaptation of the differential evolution Markov chain Monte Carlo model (Turner et al., 2013), in R version 4.1.0 (R Core Team, 2021). Plots of model fit (Gelman et al., 1996) and parameter recovery studies (Heathcote, Brown, et al., 2015) indicated good model fit to the data in each task and acceptable parameter recovery given the task designs. Following previous work (Heathcote, Suraev, et al., 2015; Weigard et al., 2020), efficiency of evidence accumulation (hereafter, "efficiency") was estimated as the difference in accumulation rates between the evidence accumulator for the correct choice (*vc*) and that for the incorrect choice (*ve*) scaled by the between-trial variability in accumulation rates pooled across both choices (*sv*): (*vc*-*ve*)/*sv*. It was estimated separately for "go" and "no-go" trial types within the go/no-go task due to previous evidence that it may systematically differ across the two conditions (Huang-Pollock et al., 2017; Ratcliff et al., 2018).

Self-report of executive functioning. Twins reported on their EF abilities via the attention, activation control, and inhibitory control subscales of the Early Adolescent Temperament Questionnaire (EATQ; Capaldi & Rothbart, 1992). These scales consist of six, five, and five items, respectively, and can be combined to make up an "effortful control" superscale, though for the purposes of this study each scale was used as a separate indicator (in a latent factor) of executive functioning (Snyder et al., 2015). The attention scale measures the ability to focus and shift attention, the activation control scale measures the ability to begin and complete tasks, and the inhibitory control scale measures the ability to suppress unwanted behaviors (Snyder et al., 2015). Within our sample, internal consistencies for the attention and activation subscales were borderline to acceptable ($\alpha = .674$ and .748) while internal consistency of the inhibitory control subscale was poor ($\alpha = .355$).

Executive functioning factor score. Individual twins' executive functioning scores were calculated as latent factor scores combining the three efficiency parameters (go, no-go, and stop-signal) with the three self-report executive functioning scores (attention, activation control, inhibitory control) using maximum likelihood estimation with bootstrapping in Mplus version 8.6 (Muthén & Muthén, 1998–2017) via R, tidyverse (Wickham et al., 2019), and the *MplusAutomation* package (Hallquist & Wiley, 2018). This modeling approach with full information estimation is robust to missing data, and executive functioning scores were therefore available for all participants ($N = 708$ individuals). Correlations between all executive functioning measures are available in Table S8.

Parent executive functioning

Multidimensional personality questionnaire. Parents reported on their own self-control abilities via the self-control scale of the Multidimensional Personality Questionnaire (MPQ), a 24-item scale which measures planfulness versus impulsivity (Tellegen & Waller, 2008). A higher score on this scale indicates better self-control. Within our sample, internal consistency for this scale was acceptable ($\alpha = .743$ and .752 for mothers and fathers, respectively).

Adult Self-Report. Parents reported on their own attention problems via the attention problems scale of the Adult Self-Report (Achenbach & Rescorla, 2001). This scale measures attention problems (e.g., "I have trouble concentrating or paying attention for long") and was reverse-scored such that a higher score on this scale indicated less attention problems. Within our sample, internal consistency for this scale was acceptable ($\alpha = .818$ and .809 for mothers and fathers, respectively).

Parent executive functioning composite. Control and attention scales were z-scored and then averaged for each parent to create an "average" reported executive functioning score. If parents had one measure but not the other ($N = 24$ mothers and 15 fathers), the one remaining report was used. With this approach, almost all families ($N = 339$ families) had parent executive functioning data. Correlations between all parent and child executive functioning measures are available in Table S8.

Parenting

Parental environment questionnaire. The Parental Environment Questionnaire (PEQ; Elkins et al., 1997) was administered to assess involvement and conflict in each parent–child dyad. The involvement subscale (12 items) assesses communication, closeness, and support in the parent–child relationship (e.g., "I praise my child when he/she does something well"). The conflict subscale (12 items) assesses disagreement, tension, and anger in the parent–child relationship (e.g., "I often criticize my child"). Mothers and fathers reported on their relationships with each twin, and each twin reported on their relationship with their parent(s).

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Within our sample, internal consistencies for the PEQ subscales were acceptable (for conflict, $\alpha = .901$, .883, and .873 for twin, mother, and father report, respectively; for involvement, $\alpha = .859$, .794, and .858 for twin, mother, and father report, respectively).

Alabama parenting questionnaire. The Alabama Parenting Questionnaire (APQ; Shelton et al., 1996) was administered to assess parenting in each parent–child dyad. The parental involvement subscale (10 items) assesses closeness and communication in the parent–child relationship (e.g., "You have a friendly talk with your child"). The positive parenting subscale (6 items) assesses positive reinforcement behaviors (e.g., "You praise your child if he/she behaves well"). The inconsistent discipline subscale (6 items) assesses unpredictability in parental discipline (e.g., "The punishment you give your child depends on your mood"). The corporal punishment subscale (three items) assesses physical discipline (e.g., "You spank your child with your hand when he/she has done something wrong"). Some participants received a shortened version of the corporal punishment subscale; these participants' scores were prorated accordingly. Mothers and fathers reported on their relationships with each twin, and each twin reported on their relationship with their mother. Within our sample, internal consistencies for the APQ subscales were acceptable (for inconsistent discipline, $\alpha = .749$ and .701 for mother and father report, respectively; for positive parenting, $\alpha = .785$ and .801 for mother, and father report, respectively). Because some families received only one corporal punishment item, internal consistency was not computed for that scale.

Parenting composite. Consistent with prior work (e.g., Burt et al., 2003), we created a composite of all reporters for each subscale of the PEQ and APQ to assess the overall parenting environment for each child, averaging z-scored twin reports, mother reports, and father reports, and then averaging these three reporters together. Using this method, we created a "harsh parenting" composite including data from all available reporters on the PEQ conflict, APQ inconsistent discipline, and APQ corporal punishment subscales (Table S9); we created a "warm parenting" composite from all available reporters on the PEQ involvement, APQ parental involvement, and APQ positive parenting subscales (Table S10). When any of these reporters were missing ($N = 292$ (harsh) and $N = 325$ (warm) individuals were missing at least 1 informant-report, primarily father), we calculated the composite from the available reports. Using this method, all twins had composite parenting data ($N = 708$ individuals).

Analytic plan

Question 1: Is parenting one pathway through which executive functioning is transmitted intergenerationally?

First, we employed a regression approach to test whether harsh or warm parenting was associated with child executive functioning. We then tested whether parent executive functioning might have an indirect effect on child executive functioning via harsh or warm parenting using structural equation modeling in Mplus version 8.6 (Muthén & Muthén, 1998–2017). We used a maximum likelihood estimator with bootstrapping (bootstrap = 1000). We tested for specific indirect effects of parent executive functioning on child executive functioning via harsh or warm parenting, clustering by family to account for relatedness of twins. In all models, we included child age and sex as controls. We also included twin race/ethnicity (White vs. non-White), a socially constructed category, to control for differences in exposure to systemic racism and related systematic differences by race and ethnicity in exposure to stress, trauma, and opportunity.

Question 2: What is the etiology of executive functioning?

Twin analyses leverage the difference in the proportion of genes shared between MZ twins (who share 100% of their segregating genes) and dizygotic (DZ) twins (who share roughly 50% of their segregating genes) to make inferences about additive genetic (A), shared environmental (i.e., environmental factors that make twins similar to each other; C) and non-shared environmental (i.e., factors that make twins different from each other, including measurement error; E) contributions to a given phenotype (Figure 2b; see Plomin et al., 2012). In the classical twin model, these three estimates are calculated based on just two pieces of information: the covariance between MZ twins and the covariance between DZ twins (along with multiple assumptions; Burt & Klump, 2012; Plomin et al., 2012). To address this second question, we employed the nuclear twin family model, an extension of the classical twin model. The nuclear twin family model incorporates two additional pieces of information, the covariance between parents and the covariance between parents and children, allowing researchers to account for assortative mating and differentiate shared environmental influences from passive genotype–environment correlation (Figure 2a; Burt & Klump, 2012). More specifically, the inclusion of parent data on the phenotype of interest allows for the differentiation of shared environmental influences (C) into those shared between siblings (S; "sibling-level shared environmental influences") and those passed between parents and offspring (F; "family-level shared environmental influences"). Within this model, passive genotypeenvironment correlation (w; Figure 2a) can be represented by the covariance between additive genetic factors (A) and familial environmental variance (F). Given that a major focus of our question is on family-level factors and passive genotype–environment correlation, we first ran a full nuclear twin family model including S and F (i.e., ASFE; Figure 2a). All twin models were run in Mplus version 8.6 (Muthén & Muthén, 1998–2017) via R version 4.1.0 (R Core Team, 2021) and the mplusAutomation package (Hallquist & Wiley, 2018).

Twin analyses included all twins with any relevant data ($N = 708$ individuals, $N = 354$ pairs, $N = 167$ MZ pairs). Data were prepared for twin analysis using tidyverse (Wickham et al., 2019) within R version 4.1.0 (R Core Team, 2021). To eliminate mean age or sex differences, we regressed out age and sex effects from all phenotypes of interest (i.e., extracted the residuals from a regression with age and sex predicting the phenotype of interest); We used the standardized residual from this regression as our phenotype score (Mean = 0, $SD = 1$).

Question 3: Does parenting have a unique environmental effect on executive functioning? Is there evidence of evocative genotype–environment correlation?

To further examine the relation between harsh parenting and child executive functioning, we fit classical bivariate ACE models to decompose the covariance between harsh parenting and child executive functioning. Like the nuclear twin family model, the classical twin model estimates additive genetic (A) and non-shared environmental (E) contributions to a given phenotype; however, as described above, this model estimates broad shared environmental influences (C) rather than differentiating sibling-level (S) or family-level (F) influences. The bivariate classical ACE model

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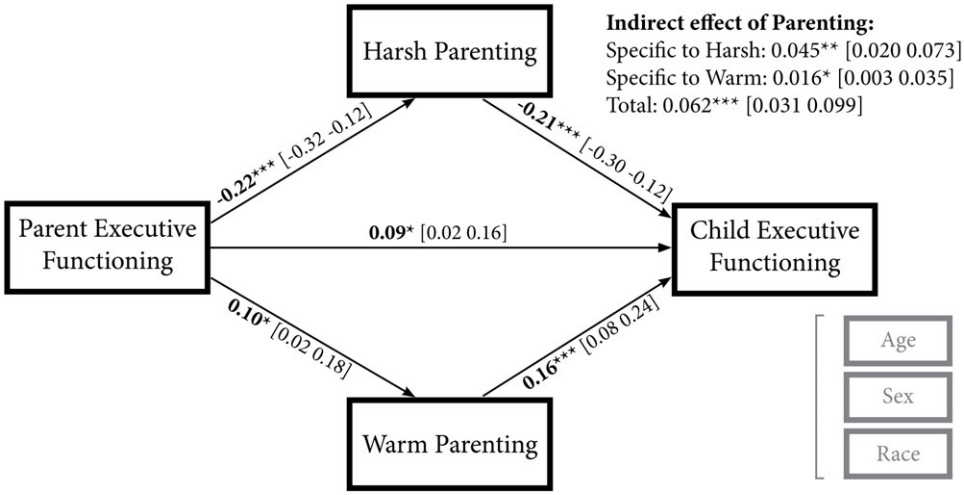

Figure 1. Parent executive functioning relates to child executive functioning via an effect on parenting. *Note.* Structural equation modeling using maximum likelihood estimation with bootstrapping and clustering for family in Mplus revealed an indirect effect of parent executive functioning on child executive functioning via both harsh and warm parenting behaviors. This figure depicts standardized estimates and bootstrapped 95% confidence intervals (bootstrap = 1000). For estimates, $p < .1$, $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$.

incorporates two phenotypes of interest into the same model, parsing the phenotypic covariance into that which is due to genetic (A), shared environmental (C), and non-shared environmental (E) factors (Figure 2c); these covariances can then be standardized on their respective variances to produce genetic and environmental correlations. These correlations can then be used to make inferences about the source of the overlap between parenting and child executive functioning. For example, because twin modeling of parenting indexes the extent to which twins' genetic tendencies influence the parenting they receive, genetic (A) overlap would indicate genetic confounds, and more specifically evocative genotype–environment correlation (Klahr & Burt, 2014). On the other hand, shared environmental overlap (C) would indicate either passive genotype–environment correlation or environmental mediation, while non-shared environmental overlap (E) would indicate environmental mediation of their relationship.

Results

Descriptive statistics are reported in Tables S1 and S2. Harsh parenting and warm parenting were moderately negatively correlated ($r = -.33$) and both harsh and warm parenting correlated with child executive functioning ($r = -.29$ and .26, respectively; Table S1). ICCs for executive functioning were .35 for MZ twins and .23 for DZ twins, while ICCs for harsh parenting were $rMZ = .75$ and $rDZ = .64$, and for warm parenting were $rMZ = .81$ and $rDZ = .70$ (see Table S2 for cross-trait cross-twin correlations). The executive functioning factor score fit was below traditionally accepted metrics for acceptable fit (Table S3). Loadings were substantially higher for self-report measures than for efficiency measures.

Harsh parenting is a phenotypic pathway linking parenting executive functioning to child executive functioning

In a path model that linked parent executive functioning to child executive functioning via both harsh and warm parenting (see Figure 1), links between parent executive functioning and harsh parenting, and between harsh parenting and child executive functioning were both significant (beta = −0.22, 95% CI [−0.32, −0.12]; beta = −0.21, 95% CI [−0.30, −0.12]). Similarly, links between parent executive functioning and warm parenting, and between warm parenting and child executive functioning were both significant (beta = .10, 95% CI [.02, .18]; beta = −.16, 95% CI [.08, .24]). Moreover, there was an indirect effect of parent executive functioning on child executive functioning via parenting (total indirect beta = .062, 95% CI [.03, .10]), and significant specific indirect effects of both harsh (beta = .05, 95% CI [.02, .07]) and warm (beta = .02, 95% CI [.003, .04]) parenting. There was also a small direct effect, such that a portion of the relationship between parent and child executive function was not mediated by parenting. This model clustered by family to account for relatedness of twins and included controls for child age, sex, and race. Simple linear regressions with each set of variables yielded consistent results (Tables S4–S6).

Executive functioning shows substantial non-shared environmental effects with some evidence for sibling-level shared environmental effects

We first ran a full ASFE nuclear twin family model, which differentiated shared environmental influences (C) into those shared among the family unit (F) and those shared by siblings (S; Figure 2a). The full ASFE model estimated large non-shared environmental influences on child executive functioning ($E = .68$, 95% CI [.54, .81]; Table 1). Sibling-level shared environmental influences, by contrast, were small and were not significant ($S = .07$, 95% CI [0, .23]; Table 1), as were the family-level (F) influences ($F = .00$, 95% CI [0, .05]; Table 1). The model also estimated passive genotype–environment correlation, or the correlation between genetic (A) and family (F) influences, to be 0. This correlation of 0 indicates that any family-level influences are unlikely to be a function of the genetically influenced tendencies of the parents. A reduced ASE model, which set F to 0, fit the data better and also indicated low-to-moderate heritability ($A = .22$, 95% CI [.12, .33]; Table 1) as well as a trend toward sibling-level shared environmental influences ($S = .09$, 95% CI [0, .19]; Table 1). The very low F estimate would suggest that any shared environmental influences on executive functioning are more likely to be those that make siblings more similar, rather than those that make children and their parents more similar. This absence of environmentally driven parent–child similarity precludes passive genotype–environment correlation effects on child executive functioning (as also seen in the above point estimate of zero for passive genotype–environment correlation).

Because child and parent executive functioning were measured in different ways (factor score including both behavioral

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Table 1. Model estimates and model fit statistics for univariate and bivariate models

Nuclear twin family models	A	S	F	E	AIC	BIC	ssBIC	RMSEA
Executive functioning ASFE	.26 [0, .53]	.07 [0, 0.23]	.00 [0, .05]	.68*** [.54, .81]	3449.97	3473.19	3454.15	.12
Executive functioning ASE	.22** [.12, .33]	.09 [0, .19]	–	.69*** [.59, .80]	3447.33	3466.67	3450.81	.12
Classical twin models	**A**	**C**		**E**	**AIC**	**BIC**	**ssBIC**	**RMSEA**
Executive functioning	.29 [0, .50]	.08 [0, .32]		.63*** [.49, .79]	1979.14	1994.62	1981.93	.00
Harsh parenting	.17 [0, .41]	.57*** [.37, .72]		.26*** [.19, .33]	1785.18	1800.66	1787.97	.05
Warm parenting	.26** [.07, .47]	.56*** [.37, .72]		.18*** [.13, .24]	1725.03	1740.50	1727.81	.02
Bivariate models	**Axy**	**Cxy**		**Exy**	**CFI**	**TLI**	**SRMR**	**RMSEA**
Harsh parenting / executive functioning	.00 [0, .32]	.08 [0, .19]		.03 [0, 0.09]	.99	.99	.07	.03
	rA = .02	*rC* = 1***		*rE* = .21**				
	prA = .01	*prC* = .70***		*prE* = .29**				
Warm parenting / executive functioning	.02 [0, 0.32]	.05 [0, 0.19]		.00 [0, .09]	1	1	.05	.00
	rA = .27	*rC* = .78*		*rE* = .03				
	prA = .31	*prC* = .65*		*prE* = .05				

Note. This table depicts estimates and model fit statistics for the univariate Nuclear Twin Family Model, which provides a decomposition of shared environmental contributions into those shared between parents and children (F) and those shared between siblings (S). The reduced ASE model fit better than the full ASFE model. Additionally, this table depicts estimates and model fit statistics for the univariate ACE models, which provide a decomposition of genetic and environmental contributions to variance in executive functioning, harsh parenting, and warm parenting ($N = 354$ pairs, 167 monozygotic). Finally, this table depicts relevant estimates from the bivariate ACE model, which provides a decomposition of genetic and environmental contributions to the phenotypic correlation between harsh parenting and executive functioning. Included are the ACE estimates for the overlap between harsh parenting and executive functioning, as well as the genetic (*rA*) and environmental (*rC*, *rE*) correlations, and the proportions of shared variance attributable to each (*prA*, *prC*, *prE*). High C correlations are likely driven by low C estimates overall. For estimates, $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$.

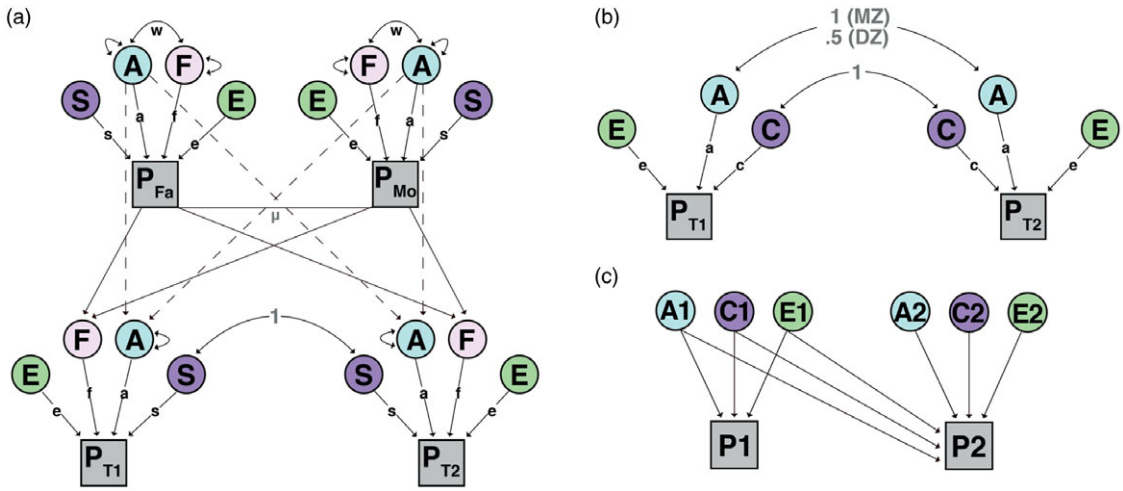

Figure 2. Path diagrams of the nuclear twin family model, the classical univariate twin model, and the bivariate twin model. *Note.* This figure depicts path diagrams of the three twin models employed by the present study. **(a)** The nuclear twin family model incorporates parent measures of a phenotype of interest to allow for estimation of additional parameters. For the purposes of this study, an ASFE model was estimated. A represents genetic influences, S represents sibling-level shared environmental influences, F represents family-level shared environmental influences, and E represents nonshared environmental influences. P represents the phenotype of interest, measured for the father (Fa), mother (Mo), and twins (T1, T2). This model also takes into account assortative mating (μ) and calculates passive rGE (w). Adapted from Burt and Klump (2012). **(b)** The classical univariate twin model estimates genetic (A), shared environmental (C) and nonshared environmental (E) contributions to a given phenotype. Adapted from Burt and Klump (2012). **(c)** The bivariate twin model decomposes phenotypic covariance into genetic (A), shared environmental (C), and nonshared environmental components (E). P1 and P2 represent the two phenotypes of interest. For simplicity, the path model for only one twin is depicted here. Adapted from Carroll et al. (2021).

performance and self-report for youth vs. self-report alone for parents), we tested two supplemental models to confirm that the very low F estimate was not caused by measurement issues. In the first supplemental model, we ran the full ASFE model using twin self-report executive functioning alone as measured by the effortful control superscale of the EATQ, which combines the attention, activation control, inhibitory control scales used in the factor score for the primary model. In the second supplemental model, we ran the full ASFE model using twin behavioral executive functioning alone, as measured by an average of the three computational metrics available (go, no-go, and stop-signal). Both supplemental models also estimated F at 0 (Table S7).

In sum, this series of nuclear twin family models suggested substantial non-shared environmental influences on executive functioning ($E = .69$), which include both measurement error and experiences that make family members more different from each other. There was also evidence for low-to-moderate genetic influences on executive functioning ($A = .22$), and trend-level

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evidence for small shared environmental influences, particularly those that are shared among siblings, but not the whole family unit (e.g., exposure to similar peers, similar parenting style, similar school environment).

The effect of harsh parenting on child executive functioning is due, at least in part, to non-shared environmental influences

We employed bivariate ACE models (Figure 2c) to examine the etiology of the association between both harsh and warm parenting and child executive functioning, finding some overlap between harsh parenting and child executive functioning on the non-shared environmental component ($rE = -.21$, $p = .014$; 29% of observed association explained by E correlation; Table 1). This finding indicates that at least part of the effect of harsh parenting on executive functioning is not due to genetic or family-level confounds, but to unique environmental influences. On the other hand, there was not significant genetic or non-shared environmental overlap between warm parenting and executive functioning, though the proportion of overlap explained by the genetic component, while not significant, was estimated to be moderate ($prA = .31$; Table 1). Both harsh parenting and warm parenting had significant shared environmental overlap with executive functioning (rC; Table 1). Though univariate models estimated very little C for child executive functioning, the bivariate model benefits from the MZ and DZ cross-trait cross-twin covariances for parenting and executive functioning (correlations in Table S2), rather than univariate intraclass correlations. For reference, univariate classical ACE estimates for harsh parenting, warm parenting, and child executive functioning are also included in Table 1. Interestingly, harsh parenting did not have a significant genetic component ($A = .17$, 95% CI [0, .41]; $C = .57$, 95% CI [.37, .72]; $E = .26$, 95% CI [.19, .33]; Table 1), while warm parenting did have evidence of moderate genetic influences ($A = .26$, 95% CI [.07, .47]; $C = .56$, 95% CI [.37, .72]; $E = .18$, 95% CI [.13, .24]; Table 1). On the other hand, both harsh and warm parenting had evidence of large shared environmental influences (C), which is often thought to reflect either family environmental influences or passive genotype–environment correlation in a child twin design (Neiderhiser et al., 2004). In the case of a relationship variable, as presented here, C might also index effects of parent personality, family socioeconomic status, or culture, on the parenting parents provide, as these effects would not vary between twins (Klahr & Burt, 2014).

Discussion

In the present study, we leveraged a genetically informed design to better understand the role of parenting in the intergenerational transmission of executive functioning. Using a latent factor capturing both self-report measures of child executive functioning and computational measures of cognitive processes underlying executive functioning, we found that parent executive functioning was related to child executive functioning via both harsh and warm parenting. Results from nuclear twin family models then revealed large non-shared environmental influences on executive functioning, with some evidence for genetic influences and no evidence of passive genotype–environment correlation. Finally, bivariate twin models indicated overlap in shared environmental influences between both warm and harsh parenting and child executive functioning, which may indicate either passive genotype–environment correlation or environmental mediation. These bivariate twin models also revealed overlap between non-shared environmental influences on harsh (but not warm) parenting and child executive functioning, but little evidence for evocative genotype–environment correlation, suggesting that the effect of harsh parenting on executive functioning is not solely due to genetic confounds. In sum, these analyses suggest that executive functioning in parents is related to executive functioning in children via parenting, and that this association reflects at least in part an environmental effect of harsh parenting.

We found that harsher parenting was associated with worse child executive functioning at the phenotypic level. This finding is in line with substantial evidence within the developmental literature that parenting matters for executive functioning development, such that harsh, controlling behaviors are associated with worse child executive functioning (Cuevas, Deater-Deckard, Kim-Spoon, Watson, et al., 2014; Valcan et al., 2017). Additionally, we found that warmer parenting was associated with better child executive functioning, consistent with a growing body of literature that indexes warm, supportive parenting as a major promoter of executive functioning development (Cioffi et al., 2020; Deater-Deckard, 2014; Distefano et al., 2018; Helm et al., 2020; Hughes & Devine, 2019; Towe-Goodman et al., 2014). We also found evidence that these parenting behaviors play a role in the intergenerational transmission of executive functioning. Better parent executive functioning was associated with less harsh, warmer parenting, and there was an indirect effect of parent executive functioning on child executive functioning via both harsh and warm parenting behaviors. This finding is consistent with other studies suggesting that parenting may be one mechanism linking parent executive functioning and child executive functioning (e.g., Korucu et al., 2020). Much of the extant work regarding parenting and executive functioning focuses on early childhood (see Valcan et al., 2017 for a meta-analysis). Thus, our study extends these findings to adolescence (96.7% of the sample was age 11–17). Additionally, while previous work measured specific and highly relevant parenting behaviors like scaffolding or intrusiveness (Broomell et al., 2020; Distefano et al., 2018; Fay-Stammbach et al., 2014; Hammond et al., 2012; Hughes & Devine, 2019; Valcan et al., 2017), our parenting composites indexed a much broader picture of the parent–child relationship, incorporating multiple reporters and multiple questionnaire measures, thus extending previous research to broad indexes of positive and negative parenting. This robust association between broad parenting and child executive functioning could imply that clinical strategies to generally decrease harshness and increase warmth may support child executive functioning even without teaching more targeted parenting behaviors like scaffolding.

Using twin modeling, we found evidence for large non-shared environmental influences on executive functioning. In contrast to other twin studies of executive functioning (Engelhardt et al., 2015; Friedman & Miyake, 2017; Friedman et al., 2008), we found the heritability of executive functioning to be modest ($A = .29$). A notable difference between the present study and previous work is the measure of executive functioning. While previous work used latent factors of performance across various executive functioning tasks to assess task-general executive functioning (Friedman & Miyake, 2017), the present study instead used computational modeling to index efficiency of evidence accumulation, a task-general cognitive mechanism thought to underlie performance on executive functioning tasks (Weigard & Sripada, 2021) and then combined these computational measures with self-report executive

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functioning data. The task-general efficiency measure is reliable (Lerche et al., 2020; Schubert et al., 2016; Weigard et al., 2021) and has recently shown consistent links with self-regulatory processes and promise as a transdiagnostic risk factor for psychopathology (Sripada & Weigard, 2021; Weigard & Sripada, 2021). Thus, our inclusion of this computational measure of executive functioning may have contributed to different heritability estimates. On the other hand, supplemental models did find that there was evidence of large non-shared environmental (E) influences even when measuring executive functioning with behavioral or self-report measures alone. However, E estimates do include measurement error, and it is possible that measurement error is also contributing to the large E estimates found for executive functioning. Future work could make use of latent modeling of the computational measures across more than two tasks.

Additionally, the lower estimates of heritability in this study could be due to the sample itself. Because residence in low-income neighborhoods is a robust risk factor for externalizing behaviors, this sample was oversampled for families living in lower-income neighborhoods, a novel approach which captures greater levels of risk and subsequent externalizing than most other twin studies, which typically contain fewer families facing substantial adversity. Indeed, very few twin studies have been explicitly sampled for environmental risk (Burt et al., 2021). Because there is evidence that neighborhood disadvantage moderates the heritability of various phenotypes (e.g., Burt et al., 2016), it is possible that heritability is indeed lower within this unique sampling frame, and higher in more advantaged neighborhoods, though genotype by environment interaction models would be necessary to test this hypothesis (and the current sample is under-powered to do so).

When employing the nuclear twin family model to decompose shared environmental effects into those shared by the whole family and those common to siblings, we found no evidence for family-level influences on child executive functioning. These influences could include anything that makes siblings and parents more similar to each other, such as shared culture or the general home environment. Using this model, we also found no evidence of passive genotype–environment correlation, or the correlation between the parents' genetic tendencies and the home environment they provide for their children. This finding is consistent with adoption work, which found an effect of parenting on executive functioning even when eliminating passive genotype–environment correlation (Bridgett et al., 2018). These findings add to a body of literature which implicates parenting practices as an important environmental driver of the development of child executive functioning (Cioffi et al., 2020; Cuevas, Deater-Deckard, Kim-Spoon, Watson, et al., 2014; Deater-Deckard, 2014; Distefano et al., 2018; Helm et al., 2020; Hughes & Devine, 2019; Towe-Goodman et al., 2014; Valcan et al., 2017), providing evidence that the association between parenting practices and child executive functioning are not likely to be due entirely to shared genetics between parents and their children.

To ensure the lack of family-level influences or passive genotype–environment correlation was not due to measurement differences between parents and children, we re-ran the nuclear twin family model separately for self-report of child executive functioning and for the behavioral measure of executive functioning. Even when using only self-report for both parents and children, we still found no evidence of family-level effects. Thus, it appears this finding is not simply due to the inclusion of behavioral data for children and not for their parents. However, parents and children did not complete the exact same measures – parents self-reported their attention problems via the Adult Self-Report and their inhibitory control via the MPQ, while children self-reported attention, inhibitory control, and activation control via the EATQ. Thus, measurement differences even within the self-report data could still play a role in the very small F (family-level influences) estimate, as the use of these different measures might artificially inflate differences between children and their parents. These measurement differences could also lead to our SEM results providing an underestimate of the association between parent and child executive functioning because these different measures may tap slightly different underlying constructs. Future work could re-test these questions using identical measures for parents and children.

Separate from measurement issues, it is important to note that executive functioning is still developing during middle childhood and adolescence (Kolb et al., 2012), and some differences between parents and children may also be due to the large difference in developmental stage between parents and their children. It would be interesting to examine whether the finding of very small family-level influences remains when twins reach adulthood.

We also employed bivariate twin modeling to examine the etiology of the association between parenting and child executive functioning, finding little evidence for evocative genotype–environment correlation (i.e., overlap of genetic components (A) of parenting and child executive functioning) and that harsh parenting had some non-shared environmental overlap with child executive functioning ($rE = -.21$). This finding indicates that the association between harsh parenting and child executive functioning is not entirely attributable to genetic or family-level confounds. Harsh parenting has previously been shown to have a unique environmental impact on other child outcomes, such as callous-unemotional traits (Tomlinson et al., 2021; Waller et al., 2018). Our finding in the present study further underscores the importance of reducing harsh parenting as an intervention target to improve child executive functioning and prevent related psychopathology.

We did find evidence of significant shared environmental (C) overlap between parenting and child executive functioning, which is difficult to interpret given very low C estimates for child executive functioning overall. Regardless, this finding reflects the fact that the association between executive functioning and parenting does not vary by zygosity or across co-twins, aligning with our finding of a main effect of parenting on child executive functioning. Notably, there were relatively strong correlations in parenting received between twins (for harsh parenting $rMZ = .75$ and $rDZ = .64$; for warm parenting $rMZ = .81$ and $rDZ = .70$). Reflecting these twin correlations, C estimates for both harsh and warm parenting were high ($C = .57$ and .56, respectively, in univariate ACE models; Table 1). A child twin design indexes the extent to which influences on twins, not on parents, affect the parenting the twins receive (Klahr & Burt, 2014). Thus, in this child twin analysis of parenting, the large C estimates could reflect family-level environmental influences or passive genotype–environment correlation (Neiderhiser et al., 2004), or other influences that do not vary between twins, such as effects of parent personality, or even parent executive functioning, on the parenting they provide (Klahr & Burt, 2014).

This study has several limitations. First, the current sample size is not sufficient for a GxE analysis. A GxE model could reveal that parenting affects the heritability of executive functioning, separate from any effects of genotype–environment correlation. For

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example, harsh parenting could moderate the heritability of executive functioning, "activating" genetic risk. Future work with larger sample sizes could address this question. Second, we designed our study to incorporate both self-report and computational measures of executive functioning in an effort to use complimentary information about child executive functioning. Our factor score combining these metrics had relatively poor fit, and factor loadings for the computational measures were low. Based on these factor loadings, the extracted factor scores more closely reflected self-report. Thus, the factor contained less influence from our novel and promising computationally derived measures of executive functioning. These low loadings could reflect that the behavioral and self-report executive functioning measures were not very correlated, as expected given the growing body of literature questioning the cohesiveness of the executive functioning construct (Dang et al., 2020; Eisenberg et al., 2019; Karr et al., 2018; Weigard & Sripada, 2021). Third, our sample size was modest for these complex analyses. Studies have shown that shared environmental estimates (e.g., C) require relatively high sample sizes for adequate power; thus our study may have underestimated these effects (Burt et al., 2020). Our sample may have also been underpowered to detect significant A overlap between warm parenting and executive functioning in in the bivariate model despite a relatively large proportion of variance explained ($prA = .31$, Table 1). Finally, internal consistency of the inhibitory control subscale of the EATQ was poor ($\alpha = .355$), though our use of a factor score, which would only reflect shared variance with other constructs, may help to mitigate this issue.

In summary, we used a genetically informed design with multiple quantitative models to dig deeper into the role of parenting in the intergenerational transmission of executive functioning in adolescence. Like many others, we found that harsh parenting was associated with worse child executive functioning, while warm parenting was associated with better child executive functioning. We found little evidence of passive or evocative genotype–environment correlation and instead found that the relationship between harsh parenting and child executive functioning is due, at least in part, to non-shared environmental influences. These findings highlight that parenting may be one environmental mechanism through which executive functioning is passed across generations. More broadly, this work highlights that targeting harsh parenting through interventions is critical to improving adolescent executive functioning and preventing related psychopathology and underscores the importance of genetically informed designs when studying parenting influences on executive functioning.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0954579422000645

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Conflicts of interest. None.

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