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Parenting as a Reaction Evoked by Children's Genotype: A Meta-Analysis of Children-as-Twins Studies

Reut Avinun[1] and Ariel Knafo[1]

Abstract

Parenting has been extensively studied but mostly as a causal factor influencing child outcomes. The aim of the current article is to examine the child's side of the relationship by meta-analyzing studies which used quantitative genetic methods that provide leverage in understanding causality. A meta-analysis of 32 children-as-twins studies of parenting revealed a heritability estimate of 23%, thus indicating that genetically influenced behaviors of the child affect and shape parental behavior. The shared- and nonshared-environmental estimates, which amounted to 43% and 34%, respectively, indicate not only substantial consistency in parental behavior but also differential treatment within the family. Assessment method, age, and parenting dimension were found to be significant moderators of these influences. Our findings stress the importance of accounting for genotype-environment correlations in child-development studies and call into question previous research that interpreted correlational results in unidirectional terms with parenting as the sole causal factor.

Keywords

genotype-environment correlation, evocative, parenting, child influences, twin studies

Parental behavior arguably plays an important role in child development, as it was found to be associated with various child outcomes such as self-reliance, self-control, contentment, and externalizing and internalizing problems (Barber, Olsen, & Shagle, 1994; Baumrind, 1971; Gershoff, Lansford, Sexton, Davis-Kean, & Sameroff, 2012; Piko & Balázs, 2012; Steinberg, Mounts, Lamborn, & Dornbusch, 1991). The bidirectionality that characterizes the parent–child relationship, however, has not been incorporated into most studies within developmental research (Kuczynski, 2003). Even though the idea of the child influencing the family environment was raised a few decades ago (e.g., Anderson, Lytton, & Romney, 1986; Bell, 1968; Plomin, DeFries, & Loehlin, 1977), many studies still deduce causation based on correlational analyses. As a result, our understanding of child effects on family processes in general, and parenting in particular, is partial at best.

Establishing causation in a relationship is a challenging task: It is difficult to determine who affects whom in ongoing interactions. In an attempt to determine directionality, longitudinal studies have endeavored to build a behavioral timeline. However, time precedence is not necessarily indicative of causality (Reiss, 1995). One possible explanation of longitudinal design results may lie in genetic effects that manifest only at certain ages. This may also be true with respect to cross-lagged designs that control for past behavior. One means of disentangling the "who affects whom" conundrum is incorporating genetic methods. The advantage of genes is that their place in time is distinct, and therefore they can provide a clearer path of causation, especially when parents and their biological children are concerned. The current meta-analysis is aimed at aggregating past quantitative genetic studies that evaluated the extent of child effects on parenting to determine whether the child plays a meaningful role in shaping parental behavior.

Genotype-Environment Correlation (rGE)

The phenomenon in which children's genetically influenced characteristics are correlated with the behavior or responses of the environment (e.g., parenting) is called genotype-environment correlation (rGE). Although the concept of rGE was described in psychology more than 30 years ago (Plomin et al., 1977; Scarr & McCartney, 1983), rGE research is in its infancy. Three types of rGE have been proposed (Plomin et al., 1977; Scarr & McCartney, 1983). The first is "passive rGE," which occurs when an association

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between an environment and an individual's phenotype can be at least partly accounted for by a shared genotype between the individual (e.g., the child) and the person providing the environment (e.g., a parent). The second is "evocative rGE," which refers to responses that are evoked from the environment by genetically influenced characteristics. For example, a child characterized by high levels of antisocial behavior—a trait showing substantial heritability—is more likely to elicit harsh discipline from parents than a prosocial child. The third is "active rGE," which refers to instances where individuals select their environment (e.g., friends, university) based on their genetic tendencies. Evidence of rGE may lead scholars to reexamine unidirectional parent → child conclusions and motivate a more elaborate view of causation in the family environment.

Unraveling rGE Through Twin Research

The presence of child effects in the parent–child relationship can be supported by using a quantitative genetic/twin design and demonstrating that parental behavior is affected by children's genotype. To partition the variance of individual differences in a certain trait to environmental and genetic effects, twin studies take advantage of the genetic difference between monozygotic (MZ) twins, who share 100% of their genes, and dizygotic (DZ) twins, who share on average 50% of their genes. The twin design assumes that if MZ twins are more similar than DZ twins, the individual differences in the characteristic examined are influenced by heritability (which usually refers to an additive genetic component—the total influence of independent genetic effects—and is therefore termed A). Similarity beyond this genetic effect is attributed to the environment the twins share (shared or common environment effect, C), and any differences between the twins are ascribed to nonshared environment, which also includes measurement error (E).

Conceptualizing the environment as a characteristic of the individual, similar to IQ or personality, makes it possible to explore the extent to which it is affected by genetic (rGE) and nongenetic influences (Plomin & Bergeman, 1991). The estimation of genetic and nongenetic influences can vary due to measurement issues, one of which is the study design (Plomin & Bergeman, 1991). To examine the genetic influences on parenting, it is possible to use either a child-based sample (children-as-twins) or a parent-based sample (parents-as-twins). These two samples yield different variance components of parental behavior that constitute complementary parts of the same picture.

When the twins are parents, the focus is on influences that stem from the parents, and the estimations will be based on the differences and similarities between them. Consequently, when the characteristic measured is parenting, the heritability component will estimate direct genetic effects of the parents' genotype on the parenting they provide. However, the children-as-twins design, which focuses on the differences and similarities between the children, evaluates influences that stem from the child. In this case, therefore, the heritability component will estimate the genetic influences of the child's genotype on the parenting he or she experiences, that is, evocative rGE. Crucially, it will not be passive rGE that is estimated, because passive rGE will not create differences in the parenting experienced by MZ and DZ twins. Because the current meta-analysis focuses on child effects on parenting, only children-as-twins designs will be discussed. Although in theory the heritability component in children-as-twins studies can reflect active and evocative rGE (Neiderhiser et al., 2004), whenever parenting is concerned, it most likely reflects evocative rGE, because children cannot actively choose their parents. We will refer to the heritability component as representing evocative child effects.

The shared-environmental component in the children-as-twins design may include the effects of culture, socioeconomic status, and parents' characteristics—all of which may cause parents to treat their children similarly. The nonshared-environmental component may comprise nongenetic evocative effects that make children's behavior different and thus also the parenting they experience. Examples include the intrauterine environment, injuries/diseases/infections, or psychosocial factors such as different friends.

Possible Moderators of the Genetic and Environmental Effects on Parenting

Various studies have used the children-as-twins design to examine the environmental and genetic influences on individual differences in parental behavior (e.g., Boivin et al., 2005; Cohen, Dibble, & Grawe, 1977; Deater-Deckard, 2000; Forget-Dubois et al., 2007; Hoffman, 2011; Knafo & Plomin, 2006; Moberg, Lichtenstein, Forsman, & Larsson, 2011). A review of these studies revealed several possible moderators of these influences: parents' sex, children's age, parenting dimension, and assessment method. We describe each of these below.

Children's Age

Age is a known moderator of heritability. Heritability estimates of personality characteristics usually increase with age, whereas shared-environmental effects decrease (Bergen, Gardner, & Kendler, 2007; Haworth et al., 2009; Knafo, Zahn-Waxler, Van Hulle, Robinson, & Rhee, 2008). This phenomenon can be explained by the independence that children gain as they grow older, which makes them more likely to choose their own environment (active rGE; Scarr & McCartney, 1983). Because the genetic effects on personality and behavior become more pronounced with age (Bergen et al., 2007; Haworth et al., 2009; Knafo et al., 2008), the same is likely to hold true for evocative rGE. In other words, evocative rGE will probably increase with age as a result of the child taking a more active role in the family environment. Indeed, heritability estimates of perceived parenting have been shown to increase with children's age (Elkins, McGue, & Iacono, 1997).

Assessment Method

Parental behavior is typically evaluated by self-reports, observations, or child reports (either concurrent reports on current experienced parenting or retrospective reports on the parenting experienced during childhood). In one study that used parental self-reports and observations (Deater-Deckard, 2000), heritability estimates were 55% for individual differences in negative affect as measured by self-reports and 6% for the same parental behavior when evaluated by observations. Moreover, as reports reflect the point of view of the reporter, it is highly likely that parent and child reports on parental behavior will be different. Parents may have a motive to describe themselves in a favorable way, whereas children's perception of the parenting they experience may be affected by their own personality. Indeed, the correlation between DZ twins tends to be higher when it is based on parental reports than when it is based on twins' reports (Wade & Kendler, 2000). Accordingly, the present meta-analysis examines assessment method as a moderator by comparing observations, parent reports, concurrent child reports, and retrospective child reports.

Parent's Sex

Parental behavior has been shown to differ according to the sex of the parent. Mothers tend to be more permissive and authoritative and fathers tend to be more authoritarian and controlling (Cohen et al., 1977; McKinney & Renk, 2008). Importantly, a children-as-twins study (Elkins et al., 1997) found distinctive genetic influences on variation in perceived paternal behavior when compared with the genetic influences on variation in perceived maternal behavior, suggesting that children differentially affect the behavior of each parent.

Parenting Dimension

The magnitude of the genetic and environmental variance components of parental positivity (e.g., warmth, structuring, sensitivity) and parental negativity (e.g., harsh discipline, negative affect, coercion) appears to differ substantially in some studies (Klahr, Thomas, Hopwood, Klump, & Burt, 2013; Knafo, 2011). For example, in one study, Knafo (2011) observed that maternal warmth did not show any genetic influence, while genetic effects on intrusiveness explained 52% of the variance. In another study (Deater-Deckard, 2000), children's genotype was not found to be related to observed positive affect or to negative control, but it was shown to affect responsiveness (49%). This suggests the possibility that certain parental behaviors are more susceptible to the influence of child behavior.

The current investigation focuses primarily on two parenting dimensions, chosen on the basis of their inclusion in all studies and to achieve maximum power: *parental positivity* and *parental negativity*. Parental positivity included positive affect, which consists of warm, accepting, and responsive behavior shown toward the child, and positive control, which consists of parental behavior meant to guide the child through praise and explanations, and includes, for instance, autonomy support, setting limits, teaching, and providing structure (Deater-Deckard, 2000; Karreman, van Tuijl, van Aken, & Deković, 2008). Parental negativity included negative affect, which consists of hostile, angry, and rejecting behavior, and negative control, which consists of power-assertive techniques to control the child's behavior, such as verbal and physical punishment, intrusiveness (Deater-Deckard, 2000; Karreman et al., 2008), and psychological control (Barber, 1996). We compared results for parental positivity and negativity.

A previous review of the genetic effects of children on their parents (Kendler & Baker, 2007) divided parental behavior differently and created three categories: (a) warmth, (b) protectiveness, and (c) control and negativity. In their review, the authors concluded that the emotional quality of the parent–child relationship is more affected by the genetic characteristics of the child than parenting behavior related to disciplinary styles (e.g., control or protectiveness). Warmth/emotional quality and control are considered to be the two main dimensions of parenting (Darling & Steinberg, 1993; Maccoby & Martin, 1983), and comparing them in a formal meta-analysis is of interest. Consequently, as an additional analysis, we also divided the previous literature according to *affect* and *control* (this was not chosen as the primary categorization, because it did not enable the inclusion of all studies). Affect was defined as expressions of emotion toward the child (e.g., warmth, anger, frustration), and control was defined as disciplinary actions (e.g., harsh discipline, assertiveness, respect for autonomy).

The Current Meta-Analysis

Investigating the role of the child in the family environment is crucial for the understanding of family dynamics and child development. As mentioned, we believe that incorporating genetics may be the best way to unequivocally establish the impact of the child's behavior on the parents. Two previous reviews reported on the genetic effects of children on their parents (Kendler & Baker, 2007; Plomin & Bergeman, 1991). The latest one was based on 19 studies (Kendler & Baker, 2007). Five years later, we were able to locate 32 studies for analysis. Most important, a formal meta-analysis has not yet been conducted. Even though the two reviews concluded that the child's genetically influenced behavior shapes parenting, an empirical analysis was still needed. The purpose of the current meta-analysis is to combine the inconsistent data from past research on evocative rGE, and examine possible moderators of the genetic and environmental influences on parental behavior while relying on variability (wide age range, different assessment methods) that can only be obtained by aggregating a diverse set of studies.

Method

To find all children-as-twins papers published by May 2012 that reported on the genetic and environmental effects on parental behavior, we conducted a computerized search in PsycNET and PubMed using the following keywords in various combinations: parenting, twins, heritability, parental behavior, rGE, child effects, parent, warmth, shared environment, adoption, and genetic. The results yielded 27 relevant papers. These papers included 32 studies, which are listed in Tables 1 and 2.

Twelve additional papers (list available from authors) that used the same twin registry but did not add new data (e.g., did not use a different age group or did not report on a different parent) were excluded. Other papers were excluded for four main reasons: (a) Heritability or correlations of parenting were not reported; (b) scores were based on a measurement of the family environment (e.g., the Family Environment Scale) and not specifically of parenting; (c) reports on mothers and fathers were averaged together; and (d) the twin registry consisted of adults who reported on their own parenting style (parents-as-twins design), thus examining direct genetic influences on parenting, which is not the focus of the current article. Because the search yielded only two adoption studies (Deater-Deckard, Fulker, & Plomin, 1999; Rende, Slomkowski, Stocker, Fulker, & Plomin, 1992), both on the same cohort, they were excluded from the analyses. In addition, there were only two cohorts that also included siblings—the nonshared-environment and adolescent-development cohort (O'Connor, Hetherington, Reiss, & Plomin, 1995), and the GENESiS 1219 (G1219) and G1219Twins projects (Pike & Eley, 2009). Sibling correlations were therefore excluded to avoid adding additional assumptions and variance to the analyses. DZ opposite-sex (DZO) twins were included in the analyses, because 8 papers (which correspond with 10 studies) did not provide separate results for DZO and DZ same-sex twins (DZS; 6 of the 10 corrected for sex). In the 2 studies that did report DZO and DZS correlations separately, these correlations were not meaningfully different (Knafo & Plomin, 2006; Moberg et al., 2011).

Statistical Analysis

Analyses were carried out with the Comprehensive Meta-Analysis (CMA) software, Version 2.2.064 (Borenstein, Hedges, Higgins, & Rothstein, 2009), and with the model-fitting program Mx (Neale, Boker, Xie, & Maes, 2003). All correlations were transformed into the normally distributed Fisher's *Z*. The meta-analyses were done separately for DZ and MZ twins. After calculating the summary effects of the correlations, the variance components (i.e., the genetic and environmental contributions to the variance) were estimated. Put differently, the summary effects were calculated in the CMA software separately for DZ and MZ twins (zygosity was defined as a subgroup within studies), based on the pooled correlations from relevant studies, which were calculated as explained below. Summary effect sizes are based on the means of the studies' weighted effect sizes. The random-effects model, which was implemented in the current meta-analysis as described further on, assumes that the true effect size varies between studies due to moderators (i.e., that there is not a fixed-effect size), so the summary effect is the estimate of the mean of the distribution of effect sizes. As a result, under the random-effects model, the weight of each study is based on within- and between-study variances (for an in-depth description of how summary effect sizes are calculated, refer to Borenstein et al., 2009). After the summary effects were calculated (i.e., one correlation for MZ twins and one correlation for DZ twins), and the sample sizes were summed up, they were entered into Mx to estimate the variance components and their 95% confidence intervals (CIs).

Parental Behavior Classification

The various parental behaviors included from each study are listed in Supplementary Table 1. Based on content, all the measured parental behaviors in the reviewed studies were assigned to either parental positivity or parental negativity. In cases where the measured parental behavior did not fit either categorization, it was excluded. When there was more than one measurement of either parenting dimension (e.g., harsh discipline and negative affect represent parental negativity), we calculated a pooled score according to a weighted average based on the sample size of each of the individual correlations. The final pooled effect sizes used in the analyses are shown in Tables 1 and 2. As an additional analysis, we also classified parental behaviors according to affect and control. The final pooled effect sizes of this analysis are shown in Supplementary Table 2.

Moderators

Testing for moderation was done by using the chi-square difference test—comparing a model in which the variance components are constrained to be equal across the levels of the examined moderator (e.g., preschool vs. school-age) to a model in which the variance components are free to vary. Model fit was assessed using the chi-square statistic and the Akaike information criterion (AIC; Akaike, 1987). If the fit of the two models differs significantly, it means that the variance components (i.e., the genetic and environmental estimates) across the different levels of the moderator cannot be

Table 1. Summary Effect Sizes/Correlations for Observations and Parent Report Twin Studies Based on Parental Negativity and Positivity.

Study	Cohort	Assessment method	Age (*M*)	*N*(MZ)	*N*(DZ)	rMZ PN	rDZ PN	rMZ PP	rDZ PP
Deater-Deckard (2000)	TRACKS	Report	3.58	62	58	.76	.58	.73	.66
Forget-Dubois et al. (2007)	QNTS	Report	1.5	110	172	.70	.73		
	QNTS	Report	2.5	111	172	.72	.69		
Boivin et al. (2005)	QNTS	Report	0.42	185	290	.84	.76		
O'Connor, Hetherington, Reiss, and Plomin (1995)	NEAD	Observation	13.7[a]	92	94	.30	.25	.35	.21
Moberg, Lichtenstein, Forsman, and Larsson (2011)	TCHAD	Report	16.5	496	356	.76	.51		
Roisman and Fraley (2012)	ECLS–B	Observation	3[a]	109	324			.72[b]	.72[b]
Knafo and Plomin (2006)	TEDS	Report	3	1,841	2,700	.76	.50	.78	.67
	TEDS	Report	4	2,458	3,536	.75	.51	.77	.65
	TEDS	Report	7	2,052	3,013	.72	.47	.68	.59
Hoffman (2011)	SITSS	Report	7.14	8	28	.84	.81	.80	.87
Knafo (2011)	LIST	Observation	3.5	25	75	.45	.32	.50	.47
Klahr, Thomas, Hopwood, Klump, and Burt (2013)	TBED-C	Observation	8	185	226	.58	.40	.36	.36
Cohen, Dibble, and Grawe (1977)	NOMTC	Report	3	92	43	.91	.84	.93	.90
Leve, Winebarger, Fagot, Reid, and Goldsmith (1998)	Not specified[c]	Observation	8	77	77	.76	.80		
Fearon et al. (2006)	NTR	Observation	0.79	27	49			.69	.66
	LTS	Observation	0.79	30	32			.64	0.68
Riemann, Kandler, and Bleidorn (2012)	JeTSSA	Report	34	226	168			.90	.54

Note. The correlations shown are either the reported correlations in studies that had only one measurement of PN or PP, or weighted averages of all the measured parental behaviors relevant to NP or PP in the study (e.g., negative affect and harsh discipline were averaged in Deater-Deckard, 2000). MZ = monozygotic twins; DZ = dizygotic twins; rMZ = MZ correlations; rDZ = DZ correlations; PN = parental negativity; PP = parental positivity.
[a]These studies did not report the average age of their sample. Therefore, the age specified here was calculated according to the reported details.
[b]The correlations were not given and had to be calculated based on the reported ACE model.
[c]Twin families were identified during 1993 to 1994 in the Williamette Valley of Oregon.

equated without decreasing model fit. This indicates the significance of the moderator and shows that the variance components are indeed significantly different between the groups examined.

Parenting dimension. A comparison was made between parental positivity and parental negativity and between affect and control in mothers and fathers.

Children's age. When the mean age of the sample was not reported, it was estimated according to the age range. Two age groups were created: infancy-preschool (0-5) and school-age (6-17), resulting in a relatively even number of studies in each group. Because the age range used in Knafo and Plomin's (2006) longitudinal study spans the two groups, only age 7 from this study was included. We excluded Riemann, Kandler, and Bleidorn's (2012) study, because the mean age of their sample was 34, which is substantially different from the other samples (ages 6-17). Studies based on child reports were excluded from this analysis of age effects, because they would have presented a bias by only being included in the school-age group. Six studies were used for the meta-analysis of maternal positivity during ages 0 to 5 (four observations and two parent reports), four studies of maternal positivity during ages 6 to 17 (two observations and two parent reports), six studies of maternal negativity during ages 0 to 5 (one observation and five parent reports), and six studies of maternal negativity during ages 6 to 17 (three observations and three parent reports).

Assessment method. Comparisons were made between observations, parent reports, concurrent child reports, and retrospective child reports. When more than one assessment method was used in the same paper, only one was chosen taking into consideration the relative representation of each method.

Table 2. Summary Effect Sizes/Correlations for Child Report Twin Studies Based on Parental Negativity and Positivity.

Study	Cohort	Report type	Age (*M*)	*N*(MZ)	*N*(DZ)	rMZ PN (M)	rDZ PN (M)	rMZ PP (M)	rDZ PP (M)	rMZ PN (F)	rDZ PN (F)	rMZ PP (F)	rDZ PP (F)
Wade and Kendler (2000)	VTR	R	30	555	383	.72	.62			.74	.60		
Kendler (1996)	VTR	R	30	546	390			.83	.76			.86	.84
Rowe (1981)	OCTSAP	C	17	46	43	.44	.47	.54	.31	.43	.46	.58	.33
Elkins, McGue, and Iacono (1997)	MTFS—1978-1982	C	11	172	67	.50	.33	.425	.29	.44	.36	.40	.315
	MTFS—1972-1976	C	17	92	43	.47	.17	.42	.32	.62	.01	.58	.26
Harlaar et al. (2008)[a]	GSAS	R	37	297	635	.54	.26	.57	.35	.62	.50	.61	.45
Plomin, Reiss, Hetherington, and Howe (1994)	NEAD	C	14	93	98	.50	.43	.41	.35	.39	.43	.46	.42
Neiderhiser, Reiss, Lichtenstein, Spotts, and Ganiban (2007)	NEAD	C	16	63	75					.49	.42	.32	.36
Neiderhiser et al. (2004)	NEAD	C	16	63	75	.50	.35	.49	.36				
Lichtenstein et al. (2003)	TMS	R	45	148	174	.57	.48	.72	.46	.48	.49	.68	.56
Beaver (2011)	Add Health	C	15	289	248			.41	.23			.54	.30
Pike and Eley (2009)	G1219Twins project	C	15	328	774	.49	.31	.46	.33	.53	.33	.49	.33
Mackinnon, Henderson, and Andrews (1991)	ATR	R	40	140	196	.58	.18	.63	.26	.61	.26	.73	.47
Jang, Dick, Wolf, and Livesley (2005)	UBC Twin Project	R	31	141	129	.37	.45	.59	.53	.37	.39	.59	.56

Note. The correlations shown are either the reported correlations in studies that had only one measurement of PN or PP, or weighted averages of all the measured parental behaviors relevant to NP or PP in the study (e.g., negative affect and harsh discipline were averaged in Deater-Deckard, 2000). MZ = monozygotic twins; DZ = dizygotic twins; rMZ = MZ correlations; rDZ = DZ correlations; PN = parental negativity; PP = parental positivity; M = mothers; F = fathers; R = retrospective child report; C = concurrent child report.

[a]In this study, it was reported that for more than half of the sample, only one twin was available; therefore, new sample size estimates were calculated according to the reported data.

Parent's sex. Mothers' and fathers' parenting styles were compared. Only four studies used observations or parent reports on paternal behavior. Therefore, mother–father comparisons were only performed on data from child reports.

Publication Bias

Publication bias—the file drawer effect—relates to the selective publication of significant results. As mentioned in previous twin-based meta-analyses (McCartney, Harris, & Bernieri, 1990; Taylor, 2011), this bias is less relevant to meta-analyses of twin studies, because such studies estimate the genetic and environmental variance components of a selected characteristic and are not based on significance tests. Furthermore, twin samples are relatively rare, which makes them valuable. Nonetheless, publication bias was examined by calculating Rosenthal's Fail-safe *N* (Rosenthal, 1979), which is the number of unpublished studies that would need to be added to the analysis to yield a statistically nonsignificant effect. The variance components and their corresponding CIs were calculated in Mx for each of the 32 studies and used as described by Taylor (2011) to calculate Fail-safe *N*. Each study was represented by the summary effects of one parental behavior, which was chosen randomly (applied to studies that measured positivity and negativity and/or paternal and maternal behaviors).

Results

The *Q* statistic indicated significant heterogeneity across studies—MZ twins: $n = 32$, $Q(31) = 548$, $p < .001$; DZ twins: $n = 32$, $Q(31) = 462$, $p < .001$. Further analysis showed that 93% of the total variation between studies were attributable to heterogeneity rather than chance (i.e., a high

I^2 statistic; Higgins, Thompson, Deeks, & Altman, 2003). Thus, the high proportion of between-study differences was due to true variation stemming from potential moderators. For this reason, we chose the random-effects model for the meta-analysis rather than the fixed-effects model, which would have assumed that the variance between studies resulted from a sampling error, and that there was only one true effect size (as opposed to effect sizes that change according to moderators).

A meta-analysis based on 32 children-as-twins studies of parental behavior (each study being represented once by either paternal negativity, paternal positivity, maternal negativity, or maternal positivity, chosen at random) yielded a heritability estimate of 23% (95% CI = [20, 25]), indicating a child evocative effect of a genetic origin (i.e., evocative rGE). The shared- and nonshared-environmental components accounted for 43% (95% CI = [41, 45]) and 34% (95% CI = [33, 35]) of the variance, respectively, indicating not only substantial consistency in parental behavior but also differential treatment within the family (the nonshared-environment effect also includes measurement error). Sensitivity analyses conducted separately for MZ and DZ twin correlations indicated that the omission of any single study from the meta-analysis did not significantly affect the summary effect sizes. The results were robust and not significantly influenced by any one study (outliers). The multiple time points from overlapping samples (Boivin et al., 2005; Forget-Dubois et al., 2007; Knafo & Plomin, 2006; Neiderhiser, Reiss, Lichtenstein, Spotts, & Ganiban, 2007; Neiderhiser et al., 2004; Plomin, Reiss, Hetherington, & Howe, 1994) were also omitted during the sensitivity analyses without significantly affecting effect sizes, and therefore all available time points were included in the subsequent analyses.

Fail-safe *N* for the heritability component was 2,187 indicating that 2,187 studies averaging a heritability or evocative rGE component of zero would have been needed to make the summary evocative rGE estimate nonsignificant. Similarly, the Fail-safe *N* for the shared-environmental component was 3,034 (Fail-safe *N* was not calculated for E because it includes measurement error and is thus always larger than zero). These numbers suggest that the results presented are unlikely to have been affected by publication bias.

Moderators

Parenting dimension. The comparison of parental positivity with parental negativity was done within the subcategories of assessment method to decrease the variability between studies and receive a clearer picture of the nature of the differences. In most cases, the variance components were not significantly affected by whether the positivity or negativity of parental behavior was measured (Table 3).

Parenting dimension, classified as parental positivity/negativity, was a significant moderator of the variance components only in mothers' self-reported parenting and in retrospective child reports of paternal behavior. As shown in Table 4 (for the corresponding correlation table, see Supplementary Table 3), the difference between maternal positivity and maternal negativity, as assessed by the mothers, is very modest and was perhaps only detected due to the exceptionally large sample size. When the study with the significantly larger sample size (Knafo & Plomin, 2006) was excluded from the analysis, the chi-square difference test was still significant, $\Delta\chi^2(3, N = 2{,}303)$, = 15.68, $p < .005$, AIC = 9.68, with the difference being mostly attributable to a difference in the nonshared-environmental component—17% for maternal negativity versus 13% for maternal positivity. In addition, a comparison between paternal negativity and paternal positivity as assessed by retrospective child reports revealed higher genetic influences and lower nonshared-environmental influences on paternal positivity (Table 4). Overall, these two parenting dimensions do not seem to be differently affected by environmental or genetic influences.

The comparison of the second categorization scheme, affect and control, was also made within the subcategories of assessment method. When assessed by parent reports and retrospective child reports, affect was significantly different from control (Supplementary Table 4). Affect was more likely to be influenced by the genetic characteristics of the child (Table 5; for the corresponding correlation table, see Supplementary Table 5), as expected according to previous reviews (Kendler & Baker, 2007; Plomin & Bergeman, 1991), and less influenced by the nonshared environment. However, when assessed by observations, although the difference was not significant (Supplementary Table 4), control appeared to be more genetically influenced than affect, whereas affect was more influenced by the environment. When assessed by concurrent child reports, the difference between affect and control was nonsignificant (Supplementary Table 4) and inconsistent in mothers and fathers (Table 5). Notably, fewer studies were eligible for inclusion in this categorization compared with negativity/positivity. The variance estimates for affect as assessed by observations and parent reports were based on three studies each, which should be taken into consideration when reviewing the results.

Children's age. The chi-square difference test for comparing infancy-preschool with school-age showed that age is a significant moderator of the magnitude of the genetic and environmental influences on parental behavior. As shown in Table 6 (for the corresponding correlation table, see Supplementary Table 6), for parental positivity and negativity, the main differences lay in the shared- and nonshared-environmental components, which decreased and increased with age, respectively. We decided to further examine the results by excluding Hoffman's (2011) dissertation, which relied on an extremely small sample of 8 MZ twins and 28 DZ twins, and found high correlations ranging between .80 and .87. Without Hoffman, positive and negative parenting showed an increase in the heritability and nonshared

Table 3. Chi-square Difference Tests of Possible Moderators of Parental Behavior.

Moderator	$\Delta\chi^2$	*df*	*N*	*p*	AIC
Age					
Negativity (mothers)	113	3	8,193	<.001	106
Positivity (mothers)	174.24	3	6,588	<.001	168.24
Assessment method					
Observations/parent reports					
Negativity (mothers)	134.63	3	18,634	<.001	128.63
Positivity (mothers)	256.35	3	17,553	<.001	250.35
Parent/retrospective reports					
Negativity (mothers)	582.53	3	19,081	<.001	576.53
Positivity (mothers)	407.19	3	20,560	<.001	401.19
Parent/concurrent reports					
Negativity (mothers)	495.89	3	19,677	<.001	489.89
Positivity (mothers)	1,098.74	3	18,716	<.001	1,092.74
Observations/retrospective reports					
Negativity (mothers)	3.54	3	3,628	*ns*	−2.45
Positivity (mothers)	25.07	3	4,064	<.001	19.07
Observations/concurrent reports					
Negativity (mothers)	14.30	3	2,745	<.01	8.30
Positivity (mothers)	49.53	3	3,699	<.001	43.53
Retrospective/concurrent reports					
Negativity (mothers)	15	3	4,671	<.005	9
Positivity (mothers)	55.03	3	5,227	<.001	49.03
Negativity (fathers)	23.26	3	4,671	<.001	17.26
Positivity (fathers)	90.02	3	5,227	<.001	84.02
Parenting dimension					
Observations (mothers)	2	3	2,119	*ns*	−4
Parent reports (mothers)	107.5	3	34,068	<.001	101.5
Retrospective reports (mothers)	5.46	3	5,573	*ns*	−0.54
Retrospective reports (fathers)	19.74	3	5,573	<.001	13.74
Concurrent reports (mothers)	2.18	3	4,325	*ns*	−3.82
Concurrent reports (fathers)	0.22	3	4,325	*ns*	−5.78
Sex of parent					
Negativity (retrospective reports)	3.34	3	5,554	*ns*	−2.66
Positivity (retrospective reports)	19.29	3	5,592	<.001	13.29
Negativity (concurrent reports)	0.26	3	3,788	*ns*	−5.74
Positivity (concurrent reports)	2.76	3	4,862	*ns*	−3.24

Note. The results for age do not include Hoffman (2011). AIC = Akaike information criterion; *ns* = not significant.

environment, and a decrease in the shared environment (Table 6). This suggests that differential parenting increases as the twins grow older, most likely as a result of an increase in the twins' independence and individual experiences. Notably, after the exclusion of Hoffman, the meta-analysis of parental positivity during ages 6 to 17 was based on only three studies. Further research is needed to reach more conclusive conclusions.

Assessment method. The effect of the assessment method was also considered. Almost all comparisons made between the different assessment methods were significant (Table 3):

1. *Observations versus parent reports*: Observational measures yielded lower estimates of heritability and higher estimates of nonshared environment for maternal negativity and maternal positivity (Table 4), indicating that child genetic influences on parental behavior are less evident in observational studies, while differential parenting is less evident in parent reports.
2. *Observations versus child reports*: The difference between the two assessment methods was mostly attributable to the higher heritability and lower shared-environment estimates in child report studies. The difference between maternal negativity as reported by

Table 4. Variance Components and 95% Confidence Intervals According to Parent's Sex, Parenting Dimension (Negativity/Positivity), and Assessment Method.

	A	C	E
Observations			
Maternal negativity	.14 [.00, .32]	.41 [.26, .54]	.45 [.39, .52]
Maternal positivity	.03 [.00, .17]	.52 [.41, .58]	.45 [.39, .50]
Parent reports			
Maternal negativity	.30 [.27, .32]	.48 [.45, .50]	.22 [.22, .23]
Maternal positivity	.30 [.28, .32]	.52 [.49, .54]	.18 [.18, .19]
Retrospective			
Maternal negativity	.32 [.22, .42]	.25 [.16, .33]	.43 [.40, .46]
Maternal positivity	.46 [.36, .56]	.16 [.07, .24]	.38 [.35, .41]
Paternal negativity	.25 [.15, .35]	.33 [.25, .41]	.42 [.38, .45]
Paternal positivity	.37 [.28, .45]	.30 [.22, .37]	.33 [.30, .36]
Concurrent			
Maternal negativity	.33 [.19, .46]	.16 [.04, .27]	.51 [.46, .56]
Maternal positivity	.26 [.13, .39]	.18 [.07, .28]	.56 [.52, .61]
Paternal negativity	.32 [.18, .45]	.18 [.07, .29]	.50 [.45, .55]
Paternal positivity	.32 [.19, .44]	.17 [.07, .27]	.51 [.47, .56]

Note. Confidence intervals are specified in parenthesis. A = variance explained by additive genetic factors; C = variance explained by common environmental factors; E = variance explained by nonshared-environmental factors and measurement error.

Table 5. Variance Components and 95% Confidence Intervals According to Parent's Sex, Parenting Dimension (Affect/Control), and Assessment Method.

	A	C	E
Observations			
Maternal affect	.04 [.00, .28]	.34 [.14, .43]	.62 [.53, .70]
Maternal control	.26 [.06, .46]	.25 [.09, .40]	.49 [.42, .57]
Parent reports			
Maternal affect	.25 [.18, .34]	.64 [.55, .71]	.11 [.09, .12]
Maternal control	.11 [.04, .18]	.69 [.63, .74]	.20 [.18, .23]
Retrospective			
Maternal affect	.47 [.37, .57]	.14 [.06, .23]	.39 [.35, .42]
Maternal control	.35 [.23, .46]	.15 [.05, .25]	.50 [.46, .54]
Paternal affect	.38 [.29, .47]	.29 [.21, .36]	.33 [.30, .36]
Paternal control	.23 [.12, .33]	.30 [.22, .39]	.47 [.43, .51]
Concurrent			
Maternal affect	.23 [.06, .40]	.24 [.08, .38]	.53 [.48, .59]
Maternal control	.27 [.10, .43]	.19 [.06, .31]	.54 [.48, .61]
Paternal affect	.37 [.17, .59]	.18 [.0, .35]	.45 [.39, .51]
Paternal control	.25 [.08, .40]	.23 [.11, .35]	.52 [.46, .59]

Note. Confidence intervals are specified in parenthesis. A = variance explained by additive genetic factors; C = variance explained by common environmental factors; E = variance explained by nonshared-environmental factors and measurement error.

observations and retrospective child reports was not significant. These imply that children are more likely to report differential parenting, and that evocative child effects of a genetic origin are more evident in child reports than in observations.

3. *Parent versus child reports*: As expected, the nonshared-environment effects were higher and the shared-environment effects were lower when based on child reports than when based on parent reports (Table 4). These confirm that parents tend to stress the equality in their parenting style, and that children are more prone to noticing differences in parental treatment.
4. *Concurrent versus retrospective child reports*: The difference between concurrent and retrospective reports of maternal positivity was due to (a) a difference in the nonshared-environmental component, which was higher when based on concurrent reports

Table 6. Variance Components and 95% Confidence Intervals According to Parenting Style and Age (Studies Based on Observations or Parent Reports).

	A	C	E
Ages 0-5			
Maternal negativity	.19 [.11, .27]	.58 [.51, .65]	.23 [.20, .25]
Maternal positivity	.07 [.00, .17]	.67 [.58, .74]	.26 [.22, .30]
Ages 6-17			
With Hoffman (2011)			
Maternal negativity	.23 [.18, .28]	.43 [.38, .47]	.34 [.32, .36]
Maternal positivity	.00 [.00, .06]	.54 [.49, .55]	.46 [.44, .48]
Without Hoffman (2011)			
Maternal negativity	.32 [.26, .37]	.34 [.29, .39]	.34 [.32, .36]
Maternal positivity	.16 [.08, .24]	.33 [.27, .39]	.51 [.48, .54]

Note. Confidence intervals are specified in parenthesis. A = variance explained by additive genetic factors; C = variance explained by common environmental factors; E = variance explained by nonshared-environmental factors and measurement error.

(56% compared with 38%; Table 4) and (b) a difference in the heritability component, which was higher for retrospective reports (46% compared with 26%; Table 4). The differences in paternal behavior and in maternal negativity, as estimated by retrospective and concurrent child reports, stemmed primarily from the shared- and nonshared-environmental components (Table 4). Compared with concurrent reports, the influence of the shared environment was higher when based on retrospective reports, whereas that of the nonshared environment was lower. This suggests that retrospective accounts consider a more global view of parenting, one that is less susceptible to daily fluctuations that may affect reports on parenting as it is experienced at the time of the report.

Parent's sex. The effect of the parent's sex was examined separately in retrospective and concurrent child reports. Table 3 shows that the only significant difference was between maternal and paternal positivity as assessed by retrospective child reports. Retrospective child reports of maternal positivity were more affected by the nonshared environment (38% compared with 33%) and heritability (46% compared with 37%), whereas those of paternal positivity were more affected by the shared environment (30% compared with 16%).

Discussion

The principal purpose of the current article was to determine whether children meaningfully affect parenting, by meta-analyzing studies which used quantitative genetic methods that provide leverage in understanding causality. The aggregation of children-as-twins studies of parental behavior enabled us to determine whether there is a child effect on parenting and, if such an effect exists, determine its extent and how it is moderated. A meta-analysis based on the 32 studies that were found yielded a heritability estimate of 23%, indicating that genetically influenced behaviors of the child evoke certain behaviors in parents and attesting to the role the child plays in shaping parenting. The shared- and nonshared-environmental influences accounted for 43% and 34% of the variance in individual differences in parenting, respectively, indicating not only substantial consistency in parental behavior but also differential treatment within the family. Notably, children's age, assessment method, and parenting dimension significantly moderated these influences as discussed below.

Genetic Child Evocative Effects

Evidence for evocative rGE was obtained with parent and child reports. While the genetic effects discovered by child reports may represent genetic influences on the way children perceive their parents and not on parenting per se, the genetic effects discovered by parent reports cannot be similarly dismissed. The heritability component that is based on a children-as-twins design only estimates the influence of children's genotypes. Therefore, any influence of the parents' genotypes, including the influence on the subjective processes that may affect parents' self-reports, does not affect the heritability estimate (Plomin & Bergeman, 1991). Consequently, the finding that children's genotypes also affect parent reports lends further support to the evocative child effects hypothesis.

Because the heritability estimate is based on the difference between MZ and DZ twins' correlations, it may be inflated if parents base their ratings on the perception of their twins' zygosity and not on true zygosity—that is, parents may be influenced by thinking that their twins are MZ or DZ and not by actual genetic similarities or differences. However, MZ and DZ twins' correlations that are based on the reports of parents who are misinformed regarding their children's zygosity do not differ from correlations that are based on the reports of correctly informed parents (Cohen et al., 1977; Lytton, 1980; Scarr, 1968). Thus, perceived zygosity cannot explain the differences between the parents of MZ and DZ twins. Instead, true zygosity and genetically influenced behaviors of the twins seem to account for this effect.

While the child's genotype was found to have an important effect on parenting whenever the latter was measured by either parent or child reports, it did not show a consistent effect on observed parental behavior. When parental positivity and negativity were assessed by observers, the effect of children's genotype on parents was not significant. This finding corroborates the results of a previous systematic review (Kendler & Baker, 2007). It is possible that, during observed mother–child interactions, the mother and the child show only a limited range of behaviors, causing the mother to be more affected by the situation and her own characteristics than by the inherited characteristics of her child. The findings based on the affect/control categorization shed more light on this issue. When assessed by observers, maternal control was affected by children's genotype, but maternal affect was not, and showed higher environmental influences (Table 5). It is possible that maternal control and use of disciplinary strategies are more susceptible in the short term to the child's behavior. Indeed, at least one parents-as-twins study found that mothers' genotypes affect their positivity as rated by observers but do not affect their levels of control (Neiderhiser et al., 2004). Taken together, these findings suggest that during observed interactions, mothers' positivity is more affected by their inherited personality attributes than control, which is more affected by the child.

Shared-Environmental Effects

Above and beyond the examined moderators, shared-environmental influences were substantial, indicating that there are similarities in the ways parents treat their children. These similarities are most likely accounted for by parents' values and genetically influenced personality characteristics. Indeed, parenting has been shown to be affected by the parents' genotype (Avinun, Ebstein, & Knafo, 2012; Bakermans-Kranenburg & Van IJzendoorn, 2008; Mileva Seitz et al., 2011). Consequently, part of the shared-environment estimate may represent passive rGE, if the genotype that affects parental behavior is inherited by the child (Neiderhiser et al., 2004). The shared-environment estimate may also include cultural and socioeconomic influences.

When examined according to assessment method, the shared-environmental component appeared to vary (Table 4). Shared-environment estimations were considerably higher in observations and parent reports when compared with child reports, especially of the concurrent type. When parents report on their own behavior, it is likely that they will describe themselves as treating their children equally, and similarities in observer reports are probably influenced by the limiting experimental conditions. However, child reports represent each twin's idiosyncratic perception of the parent–child relationship and are therefore likely to provide higher nonshared-environment estimates. Notably, however, the difference between observations/parent reports and child reports should be treated with caution because child report studies are characterized by older samples, and shared-environmental influences tend to decrease with age (Table 6), which corresponds with an increase in independence and in actively choosing one's surroundings (Scarr & McCartney, 1983).

Nonshared-Environmental Effects

The nonshared-environmental influences were relatively high in observations and in child reports (33%-56%). However, parent reports showed somewhat lower (18%-22%; Table 4) nonshared-environmental effects. In addition to the explanation provided above regarding the desire of parents to view themselves as impartial, parent reports are the only measurements where the same individual evaluates parental behavior toward both twins. Because the estimation of the nonshared-environmental component also includes measurement error, it is difficult to know to what extent the individual differences in parental behavior can be attributed to nonshared-environmental influences. Nevertheless, because the high nonshared-environmental component is consistent across various assessment methods, it likely represents true differences in parental behavior within the family.

Indeed, through a design that focuses solely on MZ twins, it has been shown that even when genetic influences are accounted for, differential parenting still exists (Caspi et al., 2004). Differential parenting may be a response to variation in child behavior that arises due to differentiating environmental events (social or biological), such as those occurring during intrauterine development (Kaminsky et al., 2009). Caspi and colleagues (2004) investigated the origin of discordant MZ twin differences by interviewing seven of their mothers. Several possible explanations arose, two of which were illness of only one twin and folk beliefs about twins (e.g., that one twin in a pair must be dominant, or one must be feminine and one masculine), which lead the parent to treat the twins accordingly. More research is needed to uncover the reasons behind differential parenting. In any case, it is probable that nonshared-environmental influences include nongenetic child evocative effects.

It is interesting that nonshared-environmental influences are significantly higher when parental behavior is assessed by concurrent child reports, compared with retrospective child reports, especially because the studies that use retrospective reports are characterized by older samples (*M* age 37 compared with 17). It is possible that concurrent reports are more sensitive to daily fluctuations and may evaluate a more time-dependent parental behavior. Retrospective reports, which assess parental behavior until the age of 16 and are usually given to children aged 30+, provide a global measure of parental behavior that spans across a longer time period. Another difference between the two methods is the reference used for comparison. Because reporters in retrospective studies are usually older, they are more likely to be influenced by their own experience of being a parent, which may also contribute to a more global view of parenting.

Effects of Possible Moderators

Children's age was a significant moderator of the environmental and genetic influences on parental behavior. Environmental influences that make parents treat their children differently increased with age, implying that the unique experiences children accumulate lead to an increase in differential parenting. Genetic evocative effects also increased, indicating that as children grow older, parental behavior is more likely to be influenced by their inherited characteristics. Taken together, the increase in environmentally influenced differential parenting and in the genetic effect of children on parenting may be accounted for by children's increased agency and ability to operate independently as they grow up. Whether environmentally or genetically influenced, children's behavior may be a more powerful predictor of parenting in adolescence.

The examination of parenting dimension did not reveal a consistent difference between positivity and negativity across the various assessment methods. However, the comparison of affect and control did reveal interesting phenomena. When parenting was based on parent reports and on retrospective child reports, affect showed higher evocative rGE influences than control. Higher evocative rGE influences on affect suggest that discipline strategies are more influenced by social norms than expressions of emotion, which are more influenced by child characteristics. However, when parenting was based on observations, control showed higher evocative rGE influences. We suggest that during the time frame of observations, control behaviors may be more responsive to child characteristics than affect, especially because mothers are probably more self-aware of expressing the latter. Notably, concurrent child reports did not show a consistent difference between affect and control, and further research is needed. In addition, the examination of parent's sex as a moderator suggested that based on child reports (where the assessments of paternal behavior were available), paternal and maternal behaviors are affected to the same degree by environmental and genetic influences. A direction for future research is to compare genetic and environmental effects on mothers' and fathers' parental behavior based on observations or parent reports.

Child Evocative Effects

The results of the current meta-analysis are in line with previous research that did not rely on twin samples to find evocative rGE effects. For example, an adoption study (Ge et al., 1996) showed that adopted children who had biological parents with a history of substance abuse or antisocial behavior were more hostile and antisocial and their adoptive parents used more harsh discipline than adoptees whose biological parents had no such histories. Other studies have also examined specific child behaviors that may affect parenting. For instance, among 5-month-old infants, maternal hostile-reactive behaviors were moderately accounted for by genetic factors in the child, an association mediated by infant difficultness (Boivin et al., 2005).

In addition, the presence of evocative rGE is supported by recent molecular genetic studies. To our knowledge, two molecular genetic studies examined the link between children's genotype and the parenting they experience, while also taking into account the parent's genotype to rule out passive rGE (Mills-Koonce et al., 2007; Pener-Tessler et al., 2013). Mills-Koonce et al. (2007) showed that children carrying the *Taq*1 A1 allele of the dopamine receptor D2, which has been associated with decreased reward sensitivity and addiction (Munafò, Clark, Johnstone, Murphy, & Walton, 2004; Munafò, Matheson, & Flint, 2007), exhibit greater negative mood during parent–child interactions and have mothers who are less sensitive. Pener-Tessler et al. (2013) showed that boys' serotonin transporter polymorphism genotype affects maternal positivity through their self-control.

Notably, the origin of child evocative effects may vary. While the heritability component in children-as-twins designs gives a clear indication of evocative effects, other nongenetic evocative effects may be included in the nonshared-environmental influences. For example, association with different peers or a life event occurring only to one twin may change one child's behavior, leading to differential parental treatment. While a genetic potential or tendency is heritable, the environmental trigger may not always be genetically affected. Different environmental experiences may interact differently with the same allele and lead to variability in behavior (Belsky & Pluess, 2009), and thus also to variability in reactive behaviors (e.g., parenting). Because the heritability estimate is based on similarities between the twins, such effects, albeit genetically influenced, will only be included in the nonshared-environmental component.

Several studies have shown how peer relationships can interact with certain alleles and direct the course of development to one path instead of the other (Brendgen, 2012). For instance, Lee (2011) found that affiliation with deviant peers is more strongly associated with overt antisocial behavior for carriers of the high activity allele of the monoamine oxidase-A (MAOA) gene, than for carriers of the low activity allele. Importantly, MAOA was not found to be associated with deviant peer affiliation, suggesting the absence of rGE with respect to this particular gene. As a result, only carriers of the high activity allele that had deviant friends were more likely to show overt antisocial behavior. Therefore, at least in some cases, it depends on the genotype whether discordant experiences lead to twin differences that may later lead to differential parenting.

Future Directions

Focusing on rGE research is crucial for the understanding of family interactions and child development, because some of the findings regarding parental influences may actually partially reflect child influences. For example, it was shown that children's experience of corporal punishment is mediated by the same genetic factors that predispose them to antisocial behavior (Jaffee et al., 2004)—that is, aggressive and oppositional behavior of children is associated with more frequent physical punishment. Maltreatment, however, was not found to be influenced by evocative rGE (Jaffee et al., 2004). Thus, links between harsh discipline and children's antisocial behavior can be accounted for in part by evocative rGE, and possibly also by passive rGE. Directing the spotlight to rGE research may untie some of the links between parental behavior and child outcome, and lead to reevaluation of various psychosocial theories. The importance of considering genetic influences in child-development research was acutely demonstrated when the "refrigerator mother" theory was refuted by genetic findings. If genetic influences are not accounted for, a piece of the puzzle is missing. Environmental and genetic influences should be considered together to further our understanding of development.

Our results lay the foundation for a deeper understanding of the child's influence in the family environment. Demonstrating that children's genetically influenced characteristics affect parenting opens the door for discovering which characteristics and genes are involved. As briefly reviewed above, there are recent studies that follow this path. In addition to molecular genetic and basic twin and adoption studies, there are other innovative designs that can help further the understanding of rGE, such as genetically informative longitudinal models and the extended children-of-twins model (B. N. Horwitz & Neiderhiser, 2011). The current meta-analysis also provides some valuable information regarding which parental behavior should be examined with which assessment method when exploring evocative rGE processes. For instance, it seems that evocative rGE may be difficult to detect with observational methods.

Acknowledging and understanding the bidirectionality in the parent–child relationship (and the lack thereof in cases such as maltreatment; Jaffee et al., 2004) may help improve treatments and interventions by comprehending when it is important to focus on the parent, when it is important to focus on the child, and when the focus should be turned to both. Indeed, with antisocial behavior for example, treating parents and children appears to improve the outcome of the intervention (Beauchaine, Webster-Stratton, & Reid, 2005; Webster-Stratton, Jamila Reid, & Hammond, 2004). In the same vein, understanding the underpinnings of parental behavior is of interest. Here we show that around 23% of the variance in individual differences in parental behavior can be explained in terms of evocative rGE. Parents-as-twins studies (e.g., Neiderhiser et al., 2004) have shown that the genotype of the parents explains on average a similar proportion of the variance (also depending on assessment method and the examined parental behavior). In addition to demonstrating that the genetic effects of children on parenting can be as important as the genetic effects of parents themselves, these explained variances show that there is still a large part of the variance that remains unexplained. Therefore, these lines of investigation also demonstrate the importance of the environment in the determination of parental behavior.

It is worth adding that evocative rGE is not restricted to the family environment. More research should be directed to uncover rGE processes with peers and the social environment. It will be of interest to investigate how evocative and selection processes may create cycles of influence that further underscore the individual's characteristics. For instance, the antisocial child may evoke negative responses from parents and may also choose antisocial peers; both might lead to an escalation of the antisocial behavior.

Limitations

The reviewed studies did not enable an examination of children's sex or parents' age as moderators. Because both have been shown to affect parental behavior (Krpan, Coombs, Zinga, Steiner, & Fleming, 2005; McKinney & Renk, 2008), their influence beyond any particular sample remains a question for future research. Furthermore, because we restricted our analyses to twin designs, it may be beneficial in the future to also include adoption, sibling, and twins reared-apart designs, once more of them become available. It is also worth noting that all of the studies in our meta-analysis used Western samples, and therefore further research is needed in other cultures.

Inferences that are based on the twin design are made possible by the design's "equal environments assumption" (EEA), which posits that environmental influences with an etiological importance to the trait under study are shared to the same extent by MZ and DZ twins, so that they do not depend on the perception of the twins' zygosity (i.e., thinking the twins are DZ or MZ)—that is, for the twin design to be valid, it is required that the mere perception of the twins' zygosity will not affect the way they are being treated by their environment. The EEA has been criticized over the years (A. V. Horwitz, Videon, Schmitz, & Davis, 2003; Joseph, 2002) by scholars who argued that there is a higher environmental similarity between MZ twins that leads to inflated heritability estimates. However, it is important to bear in mind that higher environmental similarities may in fact represent rGE, because identical twins are more likely to affect their environment similarly either by actively choosing similar things or by evoking similar responses from their surroundings.

One way to test the EEA is to exploit cases in which the reporters of the characteristic or environment under question hold a mistaken belief regarding the twins' zygosity. When MZ twins are mistakenly thought to be DZ twins, they can be compared with true MZ and DZ twins. If the correlation of the MZ-thought-to-be-DZ-twins is more similar to true MZ twins, then the EEA is supported, because this shows that genetic influences are at play (the same is true for DZ twins who are thought to be MZ twins). Indeed, the EEA has been tested and found to hold true in many cases (Plomin, DeFries, McClearn, & McGuffin, 2001) and specifically in parenting (Cohen et al., 1977; Lytton, 1980; Scarr, 1968).

Conclusion

The results of the current meta-analysis provide empirical evidence for the role of children's genotype in affecting parenting and show unequivocally that evocative child effects should not be overlooked in developmental research. In addition, It is worth noting that the evocative effects found here do not account for all the influences children have on their parents. Because the environment also shapes children's behavior, not all evocative effects are entirely of a genetic origin. Further study is needed to shed light on the extent and limits of child influences, to elucidate the differences between the various assessment methods, and to find the specific genes and child behaviors that affect parenting. Our findings stress the importance of viewing children as active agents in the family environment and call for a deeper understanding of family socialization as not only a social but also a biological system.

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