CHILD DEVELOPMENT

How Heritable are Parental Sensitivity and Limit-Setting? A Longitudinal Child-Based Twin Study on Observed Parenting

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We examined the relative contribution of genetic, shared environmental and non-shared environmental factors to the covariance between parental sensitivity and limit-setting observed twice in a longitudinal study using a child-based twin design. Parental sensitivity and parental limit-setting were observed in 236 parents with each of their same-sex toddler twin children (M_{age} = 3.8 years; 58% monozygotic). Bivariate behavioral genetic models indicated substantial effects of similar shared environmental factors on parental sensitivity and limit-setting and on the overlap within sensitivity and limit-setting across 1 year. Moderate child-driven genetic effects were found for parental limit-setting in year 1 and across 1 year. Genetic child factors contributing to explaining the variance in limit-setting over time were the same, whereas shared environmental factors showed some overlap.

Parenting has a tremendous influence on children's lives. This is perhaps most obvious from examples of children growing up without parents, as in "An experiment in zero parenting," describing the long-term harm in Romanian orphans who grew up in institutionalized care (Nelson, Fox, & Zeanah, 2014). But it is not only these extreme caregiving environments that leave their marks on children's development. The normal variation in parental sensitivity has also been shown to be related to child behavior problems (Eisenberg et al., 2005; Windhorst et al., 2015), academic achievement (Bindman, Pomerantz, & Roisman, 2015; Razza & Raymond, 2013) and even child brain morphology and functionality (Kok et al., 2015; Thijssen et al., 2017). Such findings raise the question of how parenting is shaped. A number of studies have addressed the question whether differences in (self-reported, other-reported, or observed) parenting behavior are accounted for by genetic or environmental factors (Klahr & Burt, 2014), but behavioral genetic studies of *observed* parenting are relatively few, cross-sectional, and have not covered the age range between 4 and 6 years old. The current paper uses a longitudinal child-based twin design, in which *received* parenting in monozygotic (MZ) and dizygotic (DZ) twins is compared, to estimate the relative weight of genetic and environmental factors in explaining the variance in two basic dimensions of parenting, sensitive responsiveness and limit-setting. The longitudinal nature of the design and the

inclusion of two parenting dimensions creates the opportunity to examine *common* genetic and environmental factors that are relevant for multiple concurrent parenting behaviors and for single parenting behaviors occurring at multiple time points.

Sensitive responsiveness, or sensitivity, refers to the extent to which parents notice child signals, interpret these signals correctly, and respond to these signals promptly and appropriately (Ainsworth, Bell, & Stayton, 1974). Sensitivity is a well-documented predictor of secure infant-parent attachment, as shown in meta-analyses of both correlational and experimental studies (Bakermans-Kranenburg, Van IJzendoorn, & Juffer, 2003; De Wolff & Van IJzendoorn, 1997). In addition, sensitive parenting has been related to a broad range of positive child developmental outcomes in different domains, including delay of gratification, and social and academic skills (e.g., Razza & Raymond, 2013; Sroufe, Egeland, Carlson, & Collins, 2011; Windhorst et al., 2015).

Another well-studied dimension of parenting is parental discipline or limit setting, which becomes increasingly important when sweet babies turn into terrible twos and threes. With developmental advances in cognitive and motor skills, accompanied by a growing need for autonomy and independence, toddlers and preschoolers confront their parents with challenging and disruptive behaviors. In most children externalizing behaviors decline over time, but not all children overcome their difficult behavior (Broidy et al., 2003). Ineffective parental limit setting plays a role in the persistence of difficult child behavior. Negative control and harsh parenting—as well as laxness and a lack of monitoring are associated with more child behavior problems than gentle but firm limit-setting and an authoritative parenting style (Kochanska, Aksan, Prisco, & Adams, 2008). Coercive cycles (Patterson, 1982) and other transactional processes (Akcinar & Baydar, 2016; Sameroff, 1975) may mediate these associations, underscoring that adequate parental limit setting strategies for managing child behavior during the toddler years are highly important.

Parenting behavior has been shown to be transmitted over generations. Prospective studies show that mothers with a history of childhood maltreatment more often maltreat their own children (e.g. Berlin, Appleyard, & Dodge, 2011; Plant, Jones, Pariante, & Pawlby, 2017; Stith et al., 2009; Van IJzendoorn, Bakermans-Kranenburg, Coughlan, & Reijman, 2020). Negative and punitive limit setting is transmitted across generations from grandmothers to mothers (Seay, Jahromi, Umana-Taylor, & Updegraff, 2016). But not only negative, also positive early experiences predict later parenting behavior (Belsky, Jaffee, Sligo, Woodward, & Silva, 2005; Chen, Liu, & Kaplan, 2008). As an example, Kovan, Chung, and Sroufe (2009) videotaped interactions of parents and their 2-year-old offspring, and revisited the 2-year-olds decades later when they had children of their own. They found substantial similarity in parenting behaviors (a correlation of $r = .43$, $N = 61$), even when various confounders were taken into account. Many factors may play a role in such intergenerational transmission of parenting (Lomanowska, Boivin, Hertzman, & Fleming, 2017), including genetic transmission of parent and child characteristics, as well as contextual stressors and supports (Belsky, 1984) which tend to show stability over generations.

Genetic factors may indeed be related to parenting and might thus explain intergenerational transmission of parenting (Mileva-Seitz, Bakermans-Kranenburg, & Van IJzendoorn, 2016), as has been shown in animals (e.g., Champagne et al., 2004, see also Knop, Joels, & van der Veen, 2017). Most studies of human parenting cannot disentangle the effects of shared genes from those of the environment. Genetically informative twin or adoption studies are needed to examine the etiology of parenting behaviors. Such behavioral genetic studies provide estimates for the influence of genetic, shared environmental, and non-shared environmental factors on parenting.

Two types of behavioral genetic studies of parenting can be distinguished (see Bakermans-Kranenburg & Van IJzendoorn, 2016). *Parent-based twin designs* involve adult twin siblings parenting their offspring, and heritability estimates are computed based on a comparison of the similarity between adult MZ twins' and DZ twins' parenting. When MZ adult twin siblings are more similar in their parenting behavior than DZ twin siblings, it can be concluded that their genetics influence this parenting behavior. Such studies are however scarce, because it is more difficult and costly to recruit two families (adult twins and their children) than one family (with twin children). Moreover, as we experienced in our own lab, twin siblings—notwithstanding anecdotal similarities in life course—usually do not have children at the same point in time. The comparability of parenting behaviors in case of divergent timing, numbers, gender, and ages of children is thus problematic.

Child-based twin designs compare parenting behaviors in parents of MZ twins and DZ twins.

The extent to which parenting behavior toward MZ twin siblings is more similar than parenting behavior toward DZ twin siblings indicates child-based genetic influences on parenting because genetically influenced characteristics of the children elicit these parenting behaviors: the child's genetic make-up evokes certain parenting behaviors, and these are child-driven genetic influences on parenting. For example, children's heritable lack of inhibitory control may elicit more firm parental limit-setting. Shared environmental influences on parenting are due to sibling similarities resulting from siblings' shared experiences, regardless of their degree of genetic relatedness, or due to parents' own characteristics (personality or parenting attitudes potentially influenced by their genetic make-up). Thus, somewhat counterintuitively, the implication is that in child-based twin studies, effects of parents' genetic makeup are represented in estimates of the shared environment. In addition, shared environmental influences include factors such as family socio-economic status and the broader sociocultural environment increasing the similarity in parenting behaviors toward twin siblings. Non-shared environmental effects emerge when parents treat siblings differently for reasons unrelated to the children's genetic uniqueness, and they include measurement errors.

A meta-analysis of 27 studies with child-based twin designs yielded estimates for genetic, shared environmental, and non-shared environmental influences on parental warmth, control, and negativity (Klahr & Burt, 2014). Estimates were largely similar across these three parenting dimensions, with genetic influences ranging from 23% to 40%, shared environmental influences between 27% to 39%, and non-shared environmental influences between 32% and 44%. However, most of the child-based twin studies used questionnaire measures of parenting that may reflect parental ideas about (ideal) parenting rather than their actual parenting behavior. The ten studies that were based on observer-rated parenting yielded lower estimates of heritability than either child-reported or parent-reported parenting; genetic influences on warmth and negativity were not significant in this set of observational studies. Parental sensitivity and limit-setting do not exactly match the dimensions of warmth and control. Parents can be very warm yet intrusive, which would not be considered sensitive, and control can be exerted in both gentle and harsh ways, pointing to the significance of assessing both parental sensitivity and parental limit-setting. Moreover, the ten studies that were based on observer-rated parenting did not cover the age group between 4 and 6 years. In studies closely matching our age group, Roisman and Fraley (2008) found that parental sensitivity of 24-month-old children was mostly explained by shared (79%) and non-shared (21%) environmental influences. In line with these results, Fearon et al. (2006) found no genetic factor (residing in the infants) explaining differences in maternal sensitivity in the first year of the infants' lives. The variance in maternal sensitivity was explained by shared environmental (66%) and non-shared environmental (34%) factors. Observing twins aged 43 months, Deater-Deckard (2000) found that estimates for shared and non-shared environmental influences on both positive and negative control were similar in size (around 50%), and no role for genetic factors emerged. However, genetic influences may increase with the children's age, when genetic child characteristics may become more evident (Plomin, 2018, but see Polderman et al., 2015, for contrasting evidence). In two studies with twins that were somewhat older (6–11 years) than the twins in our sample, genetic influences (19%–25%) were found for control. Note however that control in one of these studies (Klahr, Thomas, Hopwood, Klump, & Burt, 2013) was measured during a structured play task (Etch-a-Sketch), while in the other study control was operationalized as the total number of parental directives during free play, a structured play task, and a clean-up session (Leve, Winebarger, Fagot, Reid, & Goldsmith, 1998). Except for the clean-up session, control in these contexts may be related to intrusiveness or lack of support for autonomy of the child, and not be very informative on parental sensitive limit-setting.

The current study aims to extend this body of knowledge by examining the relative influence of genetic, shared environmental and non-shared environmental factors on the variance in observed parental sensitivity and parental limit-setting using a longitudinal child-based twin design. By using a bivariate approach, we are able to model the relative influence of genetic and environmental factors on the covariance between parental sensitivity and parental limit-setting. With this approach we can reveal whether the two types of parenting behavior have similar underlying genetic or environmental factors. If these dimensions indeed have the same underlying genetic or environmental influences, similar factors may impede or promote parental sensitivity and limit-setting; if not, then differential interventions are needed to support each of the parenting dimensions. In addition, using the

longitudinal design, we examined whether similar genetic or environmental factors contribute to the stability over time of both observed parental sensitivity and observed parental limit-setting. It has been suggested that stable personality traits are mostly influenced by genetic factors, whereas personality change is largely determined by environmental factors (McGue, Bacon, & Lykken, 1993). This highlights the potentially relevant insights that can be derived from behavioral genetic analyses of parenting behavior in longitudinal studies.

In sum, we examined four bivariate or common factor models testing the covariance between observed parental sensitivity and limit-setting in parents of toddlers (3–4 years old) twice, with an intervening period of a year, and examined the pattern of covariance underlying the development of parental sensitivity and limit-setting across 1 year.

Method

Participants

Families with twins were recruited in the context of the experimental cohort-sequential twin study of the Leiden Consortium on Individual Development (L-CID) project, testing the effects of a parenting intervention in families with young twins (Euser et al., 2016). They were recruited through the birth records of municipalities in the western region of the Netherlands. Families with same-sex, 31- to 51-month-old twins received an invitation letter and information brochure. Interested families were contacted by phone to check their eligibility. Families were eligible if the children were physically and mentally able to perform the tasks and both the children and their parents were fluent in Dutch. 88% of the children were Caucasian. The sample of the current study includes all families participating in Wave 1 (September 2015–July 2016) and Wave 2 (September 2016–July 2017) of the study, resulting in a total sample size of 236 families, that is, 472 children. No data were available for twins of 23 families who did not participate in Wave 2, as they were considered "lost to follow-up." This resulted in a somewhat smaller sample size for the analyses including only Wave 2 data that is 213 families, that is, 426 children.

Descriptive statistics about the sample can be found in Table 1. The parent who was spending most time with the children was selected as the primary parent, which in most cases (92.3%) was the mother. The mean age of the primary parent was 36.7 years (SD = 4.6). The mean age of the children (52% girls) at Wave 1 was 3.8 years (SD = 0.6), and at Wave 2 4.8 years (SD = 0.6). About half of the twins were MZ, 137 MZ pairs (58%), versus 99 DZ pairs (42%) at Wave 1; and 128 MZ pairs (60%) versus 85 DZ pairs (40%) at Wave 2.

Procedure

The L-CID project has annual assessments from age 3 and onwards. For the current study, data from the two pre-intervention baseline measures were used. The first assessment was a home visit, and for the second assessment families were invited to the laboratory. Home and laboratory visits both consisted of a series of parent-child interaction tasks and individual child tasks. In addition, parents filled out several questionnaires before the home or laboratory visit and carried out ambulatory assessments after the visit. Parental sensitivity and parental limit-setting of the primary parent were measured at both time points using the Etch-a-Sketch task and a don't touch task. Before the first assessment, both parents/legal guardians gave written informed consent. The research protocol was approved by the Central Committee on Research Involving Human Subjects in the Netherlands (CCMO; NL49069.000.14).

Table 1
Descriptive Statistics of the Study Population

	Total	MZ	DZ
N twin pairs$_{wave1}$	236	137	99
N twin pairs$_{wave2}$	213	128	85
M_{age} (SD)	3.8 (0.6)	3.8 (0.6)	3.7 (0.5)
Gender twins (% girls)	52.1	52.6	51.5
Gender primary parent (% mothers)	92.3	93.4	90.8
Mean age primary parent (SD)	36.7 (4.6)	36.4 (4.6)	37.2 (4.4)
Relation primary parent with twins			
Biological parent (%)	98.7	98.5	99.0
Adoptive parent (%)	1.3	1.5	1.0
Parents' marital status[a]			
Married (%)	64.4	58.4	72.7
Registered partners (%)	5.1	5.8	4.0
Unmarried cohabiting (%)	26.7	32.1	19.2
Single parent (%)	3.8	3.6	4.0
Family socioeconomic status[a]			
Low (%)	6.8	8.8	4.1
Middle (%)	36.8	40.4	31.6
High (%)	56.4	50.7	64.3

Note. All descriptive statistics are for year 1 unless specified otherwise.
[a]Indicates the difference between the monozygotic (MZ) and dizygotic (DZ) twins is statistically significant (p < .05).

Measures

Sensitivity

Parental sensitivity was observed for both co-twins separately during a structured play situation. The primary parent performed the task twice, once with each of the twin siblings. The order (oldest child first, youngest child first) was random across families. In a computerized version of the Etch-A-Sketch task (Cents et al., 2014), the parent-child dyads were instructed to make three drawings on a computer screen, following printed examples. They could use four buttons, two for lines going up and down, and two for lines going left and right. Both the child and the parent had to control two buttons, but they could decide for themselves who controlled which pair of buttons. Diagonal lines request high levels of co-operation, as two buttons need to be pressed at the same time. The three drawings were increasingly difficult to reproduce. The duration of the task was 10 min in Wave 1 and 8 min in Wave 2. Four minutes after the start of the game (3 min in Wave 2), an audio sign was given to instruct the participants to start with the second drawing if they had not done so already. Parent-child interaction was filmed and the drawing on the screen was recorded. A single video was created with both recordings side by side.

Parental sensitivity was coded using the revised Erickson 7-point rating scales for supportive presence (1 = *parent completely fails to be supportive to the child*, 7 = *parent skillfully provides support throughout the session*) and intrusiveness (1 = *parent allows the child sufficient time to explore and to attempt to solve tools on her/his own*, 7 = *parent is highly intrusive; her/his agenda clearly has precedence over the child's wishes*; Egeland, Erickson, Clemenhagen-Moon, Hiester, & Korfmacher, 1990). The videos were coded by eight coders, trained by an expert coder (SE). Intercoder reliability (intraclass correlation coefficient; ICC) with the expert coder and among coders was adequate. For Wave 1 (n = 47 tapes), the mean ICC for supportive presence with the expert coder was .82 (range = .79–.85) and among coders .77 on average; for intrusiveness the mean ICC with the expert coder was .80 (range = .74–.85) and among coders .74 on average. For Wave 2 (n = 40 tapes), the mean ICC for supportive presence with the expert coder was .83 (range = .76–.89) and among coders .83 on average; for intrusiveness the mean ICC with the expert coder was .77 (range = .72–.81) and among coders .79 on average. Videos of co-twins or from the same family in two different waves were never coded by the same coder.

Intrusiveness scores were recoded so that higher scores on both scales indicated higher parental sensitivity. The correlation between the two scales ranged from .54 to .65 across twins and study waves. The scores for supportive presence and intrusiveness were standardized, and combined into a single measure of sensitivity by taking the mean of the two standardized scores. Sensitivity scores were missing for 14 children in Wave 1 and for 76 children in Wave 2.

Limit-Setting

Parental discipline or limit-setting was observed in a don't touch task (Van der Mark, Bakermans-Kranenburg, & Van IJzendoorn, 2002). Parents performed this task twice, once with each child. The order of the children was random between families. Parents received written instruction explaining the task before they were handed a bag of attractive toys. They were requested to take all the toys out of the bag, and to tell the child not to touch any of the toys. After 2 min, the child was allowed to play with the least attractive toy only. This episode also lasted 2 min. They were then allowed to play with all of the toys for a few minutes (this episode was not coded). The task was filmed and parental limit-setting was coded by five coders, including an expert coder (CV) who trained the other coders.

Two scales were coded: positive limit-setting, rated on an adapted version of the revised Erickson 7-point rating scales for supportive presence (1 = *parent completely fails to provide positive limit-setting*, 7 = *parent skillfully provides positive limit-setting throughout the session*; Egeland et al., 1990), and physical interference, rated on a 5-point scale (1 = *parent does not interfere physically*, 5 = *parent often interferes physically*; Yagmur, Mesman, Malda, Bakermans-Kranenburg, & Ekmekci, 2014). Intercoder reliability (ICC) with the expert coder and among coders was adequate. For Wave 1 (n = 50 tapes), the mean ICC for positive limit-setting with the expert coder was .77 (range = .71–.80) and among all coders .76 on average. For physical interference the mean ICC with the expert coder was .86 (range = .79–.93) and among coders .83 on average. For Wave 2 (n = 48 tapes), the mean ICC for positive limit-setting with the expert coder was .74 (range = .71–.79) and among all coders .76 on average. For physical interference the mean ICC with the expert coder was .88 (range = .85–.90) and among coders .89 on average. Videos of co-twins or from the same family in two different waves were never coded by the same coder.

The physical interference scores were recoded such that a higher score indicated less physical interference. This way, higher scores on both scales indicated more positive limit-setting. The correlation between the two scales ranged from .26 to .35 across twins and study waves. The scores on both subscales were standardized and then aggregated by taking the mean. Limit-setting scores were missing for 4 children in Wave 1 and for 51 children in Wave 2.

Zygosity

During the laboratory visit in the second year, DNA samples of the twins were obtained using buccal swabs for an identity by descent analysis to determine the zygosity of the twins. For 10% of twins (mostly those who did not participate in the second year), no DNA data was available and zygosity was determined on the basis of an eight-item questionnaire completed by the primary parent. This questionnaire contains items about the physical resemblance of the twins, and how often people struggle to distinguish between the two (Rietveld et al., 2000). Based on the answers, twins were classified as MZ or DZ. In comparison to our own DNA analyses, the questionnaire was reliable in predicting the zygosity in 93% of cases. Three sets of twins were excluded from the analyses as neither DNA nor questionnaire data was available.

Analyses

To estimate the influence of genetic factors (A), shared environmental factors (C), and unique environmental factors/measurement error (E) on individual differences in parenting behaviors elicited by children, we used the OpenMx (version 2.10.0; Neale et al., 2016) software package in R (version 3.5.1). OpenMx uses an extension of structural equation modeling, to discern relative contributions of A, C, and E to the variance in an outcome, and to the covariance between two outcomes, based on twin data. A bivariate Cholesky decomposition was used to compare saturated, ACE, AE, CE, and E models (Verweij, Mosing, Zietsch, & Medland, 2012). The goodness of fit for the models was compared by the difference in minus two log likelihood between two models, which is distributed as chi-square. If the difference is significant, the more parsimonious model significantly reduces the fit, and therefore the more complex model is retained. If the difference is not significant, the more parsimonious model will be retained. If two equally parsimonious, non-nested models both have no significantly worse fit than the more complex model, the one with the lower Aikaike's information criterion is retained. Four separate bivariate models were estimated: (a) covariance between sensitivity and limit-setting in Wave 1; (b) covariance between sensitivity and limit-setting in Wave 2; (c) covariance between sensitivity in Waves 1 and 2; and (d) covariance between limit-setting in Waves 1 and 2.

To obtain the relative contributions of A, C, and E to the individual level variance in parenting, the obtained path loadings from the best fitting model were standardized and squared. This results in figures that can be interpreted as proportions. In addition, the bivariate models provide information on the contribution of A, C, and E to the covariance between two parenting variables, by multiplying the standardized loadings of the A, C, and E factors for one variable by the respective loadings for the other variable, and then dividing the outcome by the cross-trait within-twin correlation (Treur, Boomsma, Ligthart, Willmsen, & Vink, 2016). These contributions of A, C, and E to the covariance between two variables can be interpreted as similar or common factors that can explain variance in both variables. In addition, we obtained the genetic, shared environmental, and non-shared environmental correlation from the correlation matrices of the best fitting models. These correlations indicate the proportion of genetic or environmental factors that are similar for two variables. OpenMx uses full information maximum likelihood estimation, which takes all available data into account to estimate the model, which reduces the need to exclude cases with single missing data points (Enders, 2001). Lastly, in bivariate analyses, the choice of model is driven by both variables, and therefore might not be the best fitting compared to modeling of each variable separately. Therefore, as a sensitivity analysis, univariate models for each of the parenting variables were also estimated.

Results

Correlations

The correlations between parental sensitivity and positive limit-setting are presented in Table 2, per wave, and per twin type. The overall within twin correlations for sensitivity were similar for Wave 1 ($r = .60$) and Wave 2 ($r = .57$), and the same was found for limit-setting in Wave 1 ($r = .55$) and Wave 2 ($r = .54$). In most cases, MZ correlations were higher than DZ correlations, but these

differences varied across outcomes. The cross-construct correlations (between limit-setting and sensitivity) within child were mostly similar for Wave 1

Table 2
Correlations Between the Combined Measures for Limit-Setting and Sensitivity

		Total	MZ	DZ
Within twin correlations per construct				
Sensitivity T1		.60**	.58**	.64**
Sensitivity T2		.57**	.60**	.51**
Limit-setting T1		.55**	.64**	.40**
Limit-setting T2		.54**	.55**	.52**
Within child correlations over construct				
Sensitivity T1	Limit-setting T1	.33**	.37**	.28**
Sensitivity T2	Limit-setting T2	.31**	.31**	.30**
Within child correlations over time				
Sensitivity T1	Sensitivity T2	.48**	.51**	.43**
Limit-setting T1	Limit-setting T2	.39**	.43**	.32**

Note. T1 = Wave 1; T2 = Wave 2. MZ = monozygotic; DZ = dizygotic.
**$p < .01$.

($r = .33$) and Wave 2 ($r = .31$). The MZ correlation at Wave 1 was higher than the DZ correlation, but at Wave 2 they were similar. The correlation between Waves 1 and 2 within child was slightly higher for sensitivity ($r = .48$) than for limit-setting ($r = .39$), and the MZ correlations were higher than the DZ correlations. Overall, correlations in Wave 1 were similar to Wave 2.

Bivariate Behavioral Genetic Analyses

The model fit statistics for each of the four bivariate models can be found in Table 3, and the resulting relative contributions of the A, C, and E factors to the variance and covariance in parental sensitivity and limit-setting are shown in Figure 1. Below, the combined results are presented for each model separately.

For *Model 1, sensitivity at Wave 1 and limit-setting at Wave 1*, the fit of the AE, CE, and E models were significantly worse compared to the ACE model. Therefore, the ACE model was retained as the best fitting model, indicating that

Table 3
*Fit Statistics for the Bivariate Behavioral Genetic Models*

	Model	Estimated variables	−2LL	df	AIC	Compared with	χ^2	p
(1) Sensitivity Wave 1 and limit-setting Wave 1								
1	Saturated model	28	2,083.89	898	287.89	—		—
2	**ACE model**	**11**	**2,097.02**	**915**	**267.02**	**1**	**13.13**	**.73**
3	AE model	8	2,114.71	918	278.71	2	17.68	< .01
4	**CE model**	**8**	**2,104.09**	**918**	**268.09**	**2**	**7.07**	**.07**
5	E model	5	2,274.84	921	432.84	3	177.81	< .01
(2) Sensitivity Wave 2 & limit-setting Wave 2								
6	Saturated model	28	1,887.02	801	285.02	—	—	—
7	ACE model	11	1,897.61	818	261.61	6	10.59	.88
8	AE model	8	1,912.66	821	270.66	7	15.05	< .01
9	**CE model**	**8**	**1,899.22**	**821**	**257.22**	**7**	**1.61**	**.66**
10	E model	5	2,038.22	824	389.22	8	139.61	< .01
(3) Sensitivity Wave 1 & sensitivity Wave 2								
11	Saturated model	28	1,973.24	832	309.24	—	—	—
12	ACE model	11	1,983.00	849	285.00	11	9.76	.91
13	AE model	8	2,009.21	852	305.21	12	26.21	< .01
14	**CE model**	**8**	**1,984.06**	**852**	**280.06**	**12**	**1.06**	**.79**
15	E model	5	2,147.48	855	437.48	13	164.48	< .01
(4) Limit-setting Wave 1 & limit-setting Wave 2								
16	Saturated model	28	1,903.91	867	169.91	—	—	—
17	**ACE model**	**11**	**1,931.08**	**884**	**163.08**	**17**	**27.17**	**.06**
18	AE model	8	1,940.99	887	166.99	18	9.91	.02
19	CE model	8	1,941.08	887	167.08	18	10.00	.02
20	E model	5	2,087.26	890	307.26	19	156.18	< .01

Note. The best fitting model per bivariate combination is shown in boldface. AIC = Aikaike's information criterion.
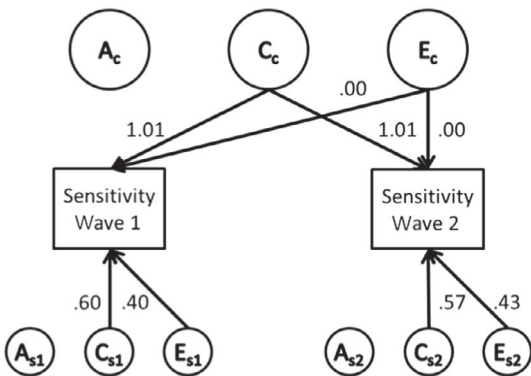
1) Sensitivity Wave 1 & Limit-setting Wave 1
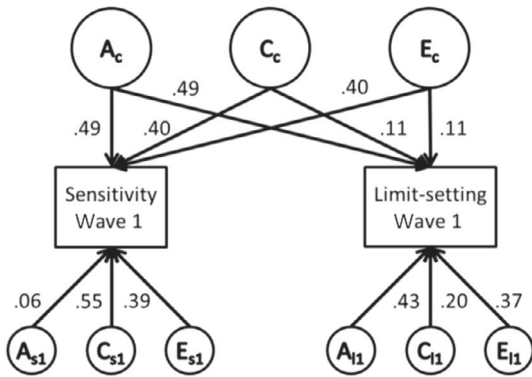
2) Sensitivity Wave 2 & Limit-setting Wave 2
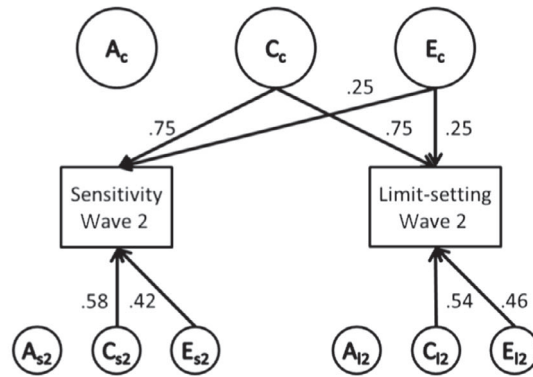
3) Sensitivity Wave 1 & Sensitivity Wave 2

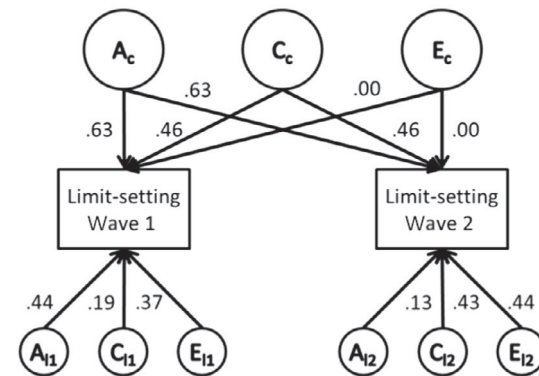## 4) Limit-setting Wave 1 & Limit-setting Wave 2

Figure 1. Parameter estimates of the bivariate models.

Note. Ac, Cc, and Ec = genetic, shared environmental, and non-shared environmental factors respectively, that explain the covariance between the two variables in the model. As1, Cs1, Es1 = genetic, shared environmental, and non-shared environmental factors respectively, that explain the variance in sensitivity at Wave 1 etc. for Wave 2 and limit-setting.

a combination of all three factors—genetic, shared and non-shared environmental factors—contribute to the variance and covariance in these parenting measures. Genetic factors explained only a small proportion of the variance in sensitivity (0.06), but nearly half of the variance in limit setting (0.43) at Wave 1. Shared environmental factors explained just over half of the variance in sensitivity (0.55) and only 20% of the variance in limit-setting. This also leaves a sizeable portion of non-shared environmental factors, at 0.39 and 0.37 for sensitivity and limit-setting, respectively. The explained covariance between sensitivity and limit-setting was attributable to A (0.49), C (0.40), and E (0.11). The model shows a genetic correlation of 1.00, indicating that the genetic factors contributing to sensitivity and limit-setting at Wave 1 are similar. The shared environmental correlation was 0.39, indicating that there is some

overlap in the environmental factors that contribute to sensitivity and limit-setting. The overlap in E is lower, as the non-shared environmental correlation was only 0.09.

For *Model 2, sensitivity at Wave 2 and limit-setting at Wave 2,* the fit of the CE model was not significantly worse compared to the ACE model, and the fit of the E model was worse than the CE model. The fit of the AE model was significantly worse compared to the ACE model. Therefore, the CE model was selected as the best fitting model, indicating that a combination of shared and non-shared environmental factors best explained the variance and covariance in the parenting measures at Wave 2. The contributions of C and E were very similar for sensitivity and limit setting, with a C of 0.58 for sensitivity, and 0.54 for limit-setting, and an E of 0.42 for sensitivity, and 0.46 for limit-setting. In addition, the explained covariance was attributable

to C (0.75), and E (0.25). Lastly, the shared environmental correlation (0.41), and non-shared environmental correlation (0.17) were quite similar to Model 1.

For *Model 3, parental sensitivity across Wave 1 and Wave 2*, the CE model also had the best fit. The contributions of C and E were virtually the same across Wave 1 and 2, at 0.60 and 0.57 for C, and 0.40 and 0.43 for E, respectively. The shared environmental factors explained all of the covariance for sensitivity across waves (1.01), leaving nothing for E. The shared environmental correlation (0.81) also showed considerable overlap between the two waves, whereas the non-shared environmental correlation did not (0.01).

For *Model 4, sensitive limit-setting across Wave 1 and Wave 2*, the ACE model had the best fit, indicating that all three factors contribute to the variance and covariance in limit-setting over time. The contribution of genetic factors seems to decrease from Wave 1 to Wave 2 (0.44–0.13), whereas the contributions of both shared and non-shared environmental factors seem to increase (0.19–0.43, and 0.37–0.44, respectively). The covariance between limit-setting in Wave 1 and limit-setting in Wave 2 is mostly explained by A (0.63), and to a lesser extent by C (0.46), and E (0.00). The model shows that the genetic factors contributing to limit-setting in Waves 1 and 2 were the same, with a genetic correlation of 1.00, whereas the shared environmental showed some overlap, but less at 0.61, and the non-shared environmental correlation was smaller and negative at −0.09.

Sensitivity Analysis

As sensitivity analyses, univariate models for each of the parenting variables were estimated. The estimates obtained with the univariate analyses corresponded exactly to those from the bivariate models in the case of sensitivity. There were only minor discrepancies for limit-setting: in Wave 1, an AE model was the best fit and in Wave 2 a CE model, in contrast to the ACE model in bivariate analyses. Results of the univariate models are presented in Appendix S1 (Table A1).

Discussion

This study for the first time estimated the genetic and environmental factors underlying observed parental sensitivity and limit-setting in families with twins aged 3.5–5 years, using a longitudinal design with two observations and an intervening period of 1 year. Our findings extend the existing literature by using bivariate or common factors models with these two different dimensions of parenting observed in a longitudinal design, in an age group that was under-represented in the literature. We have shed light on the common underlying genetic and environmental factors for sensitivity and limit-setting, and we demonstrated substantial stability in the genetic and shared environmental factors that contribute to parental sensitivity and limit-setting over time.

Our bivariate analyses demonstrated that a large proportion of the factors that influence both parental sensitivity and limit-setting are shared environmental factors. In addition, as shown by the shared environmental correlation, about half of the shared environmental factors are common for these two parenting behaviors. In other words, these are "common" shared environmental factors that influence both dimensions of parenting, apart from "unique" shared environmental factors that are only relevant for either sensitivity or limit-setting. Our findings on the importance of shared environmental factors support and extend results of previous studies indicating that the majority of the variance in various observed parenting behaviors can be explained by shared environmental factors, with only low or non-significant child genetic effects (Deater-Deckard, 2000; Fearon et al., 2006; Klahr & Burt, 2014; Roisman & Fraley, 2008), also in an early childhood age group that was not examined before. In child-based twin designs, shared environmental influences include sibling similarities resulting from siblings' shared experiences (such as the family's socio-economic status or neighborhood characteristics), but also parent characteristics, including those influenced by parental genes. It should be noted that in a recent study a genome-wide polygenic score for educational attainment was related to observed parental sensitivity, also when taking into account parents' own childhood experiences of parenting (Wertz et al., 2019). Furthermore, a meta-analysis including data from 5,853 parent–child dyads indicated small but significant associations between Big Five personality traits and both maternal and paternal parenting (Prinzie, Stams, Dekovic, Reijntjes, & Belsky, 2009). Parents who scored high on extraversion, agreeableness, and conscientiousness generally showed more warm or positive parenting, whereas parents high on neuroticism showed less warm parenting. Other shared environmental factors are related to the context of the parent-child relationship (Belsky, 1984),

like social support or marital quality. According to the spillover hypothesis, parents who are satisfied with their romantic relationship are more likely to be warm and sensitive toward their children. Indeed, two meta-analyses found positive associations between marital quality and parenting (Erel & Burman, 1995; Krishnakumar & Buehler, 2000).

In addition, the effect of "common" shared environmental factors even increased over time. The large role of "common" shared environmental factors in the covariance between parental sensitivity and limit-setting leaves ample room for intervention. In fact, our findings indicate that interventions targeting such "common" shared environmental factors may influence both dimensions of parenting. Such shared environmental factors can also include parent's genetically driven characteristics (e.g., temperamental emotional reactivity or ADHD symptoms), but future studies are needed to identify these "common" factors in order to design interventions that can effectively target both dimensions of parenting at the same time. Of course, it should be kept in mind that genetically driven child characteristics are also open to intervention, whereas not all shared environmental factors can be changed.

Parental limit-setting, in contrast to parental sensitivity, seems to some extent subject to the influence of children's genetic make-up. In fact, all child-driven genetic influences on observed parental limit-setting at two different time points were common, indicating that similar child genetic factors contribute to the parent's limit-setting behavior, irrespective of the child's age. Some common genetic effects were also found in the covariance between sensitivity and limit setting at Wave 1 (but not at Wave 2), which were mainly driven by the influence of children's genetics on limit setting. However, the size of genetic effects decreased over time, which is in contrast to the suggestion that genetic influences increase over time due to more effective shaping of the experienced environment by the developing individual (Jaffee & Price, 2007).

Interestingly, the evidence on the issue of increasing heritability estimates with increasing child age is not unequivocal. Plomin (2018) considers such an increase a "big finding" in behavior genetic research. However, Polderman et al. (2015), who included a very broad range of phenotypes, conclude that heritability estimates decrease over the years. In fact, such divergent conclusions highlight the importance for behavior research to cover the whole age range for as many adequately measured variables as possible, and our study fills a developmental gap, in that no twin studies of

observed parental sensitivity and limit setting in the age period covered by our sample are available in the literature. In the meta-analysis of twin studies of parenting, genetic influences on negativity were found in both parent-based and child-based studies (Klahr & Burt, 2014). This may indicate a process in which, in addition to a potential passive gene–environment correlation (rGE; parents transmit their genes as well as the cargiving environment to their children), children inherit the genetic tendency toward negative behavior from their parents, then through evocative rGE elicit negative parental behavior in their parents (Bakermans-Kranenburg et al., 2016; Klahr & Burt, 2014).

The differences between parental sensitivity and limit-setting might explain why, in the current study, genetic effects were mainly found for parental limit-setting, and not for parental sensitivity. Parental sensitivity and limit-setting were only modestly correlated ($r \approx .30$), indicating that we measured two distinctly different parenting behaviors. Indeed, in a frustrating situation in which parents have to refrain their children from touching attractive toys, both children and parents may be more prone to showing negative behavior as compared to a structured play setting in which the parent can follow the child's lead. The higher level of stress for the parent in combination with the increase in challenging child behavior in the discipline situation might explain the child-driven genetic effects on parental limit-setting behavior. Similarly, a child-based twin study (Oliver, Trzaskowski, & Plomin, 2014) found that the child-driven genetic influence on negative parenting was stronger than on positive parenting. However, a weakness of that study was that self-reports were used, and that the same parent completed the questionnaire twice, once for each twin sibling, creating non-independent scores and a risk for response biases.

Limit-setting may not be qualitatively different from harsh discipline and maltreatment. We believe that they can be seen as a continuum, in line with the current perspective on psychopathology that assumes the dimensionality rather than the categorization of phenotypes (Insel et al., 2010; see also Neumann et al., 2016). Therefore it is important to note that although children can elicit negative parenting behavior, negative child behavior is not responsible for eliciting child maltreatment. According to Jaffee et al. (2004) disruptive child behavior at age 5 could elicit harsh parenting, but risk factors for physical maltreatment would be more likely to reside in parental and environmental factors.

However, when the same sample was measured during adolescence, abuse and neglect were explained by child-driven genetic factors, with estimates of 71% and 47%, respectively (Fisher et al., 2015). Pittner et al. (2017) used an extended family design to demonstrate that there are child-driven genetic effects on experienced child maltreatment, but environmental factors also explained a considerable proportion of variance. Moreover, as they argued, although children may elicit negative parenting behavior, parents are responsible for reacting appropriately to their children, even in the case of challenging child behavior.

All models that were tested in the current study showed substantial non-shared effects in univariate outcomes, but very little or practically zero "common" non-shared effects in bivariate outcomes. This indicates that the non-shared factors are most likely measurement error or factors related to the specifics of the situation in which parenting was observed. For example, in the model with sensitivity at two waves, none of the common factors could be attributed to the non-shared environment, and none of the non-shared environmental factors were common for both waves. Therefore, it can be assumed that unique child characteristics (e.g., different peer experiences) have little impact on the overall parenting style. Depending on the moment and the situation in which the interaction takes place, the parent's skills can or cannot come to their full potential.

In contrast to the true non-systematic measurement error (for example related to intercoder reliability), these situation specific factors might be considered as more systematic measurement error, because they occur in specific situations. For example, if a parent just had an argument with a neighbor or the environment was noisy during the observation, that parent may be distracted and less responsive toward the child's signals. Indeed, in a study with the same Etch-a-sketch task as the current study, non-shared environmental influences on interpersonal warmth and control did not persist over time lags as short as minutes, indicating the non-systematic nature of such non-shared environmental factors (Burt, Klahr, & Klump, 2015). Although the common influence of non-shared environmental factors was very small in our results, we did find a substantial contribution of non-shared environmental factors in the univariate outcomes. Despite the fact that previous studies have proven it very difficult to explain specific non-shared environmental factors after removing error variances (e.g., Mullineaux, Deater-Deckard, Petrill,

& Thompson, 2009), non-shared environmental factors may point toward differential parenting (Dunn & Plomin, 1991). Intentionally or unintentionally, parents may act more responsive toward one of their children. Further studies are necessary to address the question of which factors in the twin siblings' non-shared environment may lead to differential parenting.

When interpreting the findings of the current study, the following should be taken into account. First, it is important to note that child-based twin designs provide information on the effects of children's genetic make-up, but cannot be informative regarding the impact of the parents' own genes or rather their early experiences on their parenting. Only parent-based twin designs can be used to estimate these genetic and (shared and non-shared) environmental effects. An ideal complementary study would use the same observational measures as reported in the current study in a longitudinal parent-based twin design, to quantify the effects of parents' genes and environmental factors on the stability and covariance of their sensitivity and limit setting. In addition, because children share 50% of their genes with each parent, it is difficult to ascertain whether biological parents are more reactive to children's difficult behavior, due to shared genetically influenced behaviors (i.e., moderated rGE). Adoption studies, being natural experiments, provide complementary opportunities to examine child evocative effects on parenting behavior of genetically unrelated parents (e.g., Klahr et al., 2017). Second, the current study has a relatively small sample size for bivariate behavioral genetic analyses. Larger samples are needed to reliably determine confidence intervals around the estimates for the contribution of genetic, shared and non-shared environmental factors, and the results should therefore be interpreted with caution. Furthermore, the first assessment was a home visit, whereas for the second assessment, families were invited to the laboratory, implying a somewhat different measurement context. This was the same for all families and even if it would lead to mean level differences (which was actually not the case in our study), such differences may not affect covariance patterns. However, measurement context cannot be disentangled from child age, leading to the possibility that the decrease in heritability of limit-setting over time could also be related to measurement context.

The most important strength of the current study is the use of observational measures with high intercoder reliability for parenting behavior. Many previous child-based twin studies have used self-

report measures of parenting. Reporting on your own parenting behavior does not only increase response-bias, it can also overestimate the shared environmental component because parents may judge themselves as being more similar toward their twin siblings than they actually are. Thus, although very time consuming, observational measures are needed for reliable estimates of genetic and environmental effects, and especially to give insight in differential parenting. Finally, the inclusion of different dimensions of parenting is another strength of the study. This enabled a description of the contribution of both the unique and common genetic, shared and non-shared environmental factors to both sensitivity and positive limit-setting. Future studies unraveling what (genetic and environmental) factors predict parenting behavior should mirror the complexity of parenting as a multidimensional construct to provide insight in the common predictors that can be targeted in interventions.

The present study showed that the parenting children receive is to a large extent influenced by their shared environment, including parental characteristics. Child-driven genetic effects were only found for parental limit-setting, indicating that children may elicit negative parenting behavior through the process of evocative rGE. Bivariate or common factor models further showed that about half of the shared environmental factors are common for parental sensitivity and limit-setting, which leaves room for intervention effects. However, current interventions often have only modest effects (Bakermans-Kranenburg & Van IJzendoorn, 2015; Euser, Alink, Stoltenborgh, Bakermans-Kranenburg, & Van IJzendoorn, 2015), which points to the need for future studies to identify common environmental processes that are relevant for multiple dimensions of parenting. Only with such knowledge can we develop targeted interventions addressing a broad range of parenting behaviors, thereby optimally promoting child developmental outcomes.

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Supporting Information

Additional supporting information may be found in the online version of this article at the publisher's website:

Appendix S1. Univariate models for the parenting variables