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Do Non-shared Environmental Influences Persist over Time? An Examination of Days and Minutes

S. Alexandra Burt, Ashlea M. Klahr, and Kelly L. Klump

Department of Psychology, Michigan State University

Abstract

Non-shared environmental influences show only minimal stability over time prior to adulthood. The long assessment lags (typically 3-5 years) that characterize most longitudinal twin studies, however, make it difficult to interpret these results. To more rigorously evaluate non-shared environmental stability prior to adulthood, we fitted biometric correlated factors models to 1) seven consecutive days of self-reported negative and positive affect in 239 twin pairs aged 16-25 years and 2) seven consecutive minutes of observer rated warmth and control in 687 twin pairs aged 6-10 years. We then empirically examined patterns of etiologic stability over time using a mixed effects analog to the one-way ANOVA. Genetic and shared environmental correlations were found to be highly stable over both days and minutes. By contrast, non-shared environmental correlations decreased monotonically with increasing lag length, and moreover, were small-to-moderate in magnitude when examining intervals longer than a few minutes. Such findings imply that the non-shared environment may be comprised primarily of transient and idiosyncratic effects prior to adulthood.

Keywords

non-shared environment; stability; genetic

Behavioral genetic research has historically concluded that the more important environmental influences on psychological and behavioral outcomes result in differences between siblings (referred to as non-shared or person-specific environmental influences) (Plomin & Daniels, 1987). This argument was based primarily on findings from twin and adoption studies, which have converged in suggesting that non-shared environmental influences account for a moderate-to-large proportion of the variance in virtually all psychological and behavioral outcomes examined to date, including personality, cognitive abilities, and psychopathology.

Given the sheer magnitude of these non-shared environmental effects, as well as their consistency across phenotypes, it is rather surprising to note that they do not appear to persist over time in any substantive way prior to adulthood (Burt, McGue, Iacono, & Krueger, 2006; Rutter, Silberg, O'Connor, & Simonoff, 1999; Turkheimer & Waldron,

2000). As an example, in one of the largest longitudinal twin studies to date (i.e., data at four waves was available for over 1,000 twin pairs), Bartels and colleagues (2004) examined etiological stability and change in internalizing and externalizing spectrum problems across ages 3, 7, 10, and 12 (Bartels et al., 2004). In contrast to genetic and shared environmental influences, non-shared environmental influences were observed to be largely (and, in some cases, exclusively) age-specific, accounting for at most 6-10% of the stability in internalizing and externalizing problems over time. These findings of temporal instability in non-shared environmental influences in particular do not appear to be specific to twin studies, as a recent longitudinal adoption study of antisocial behavior across adolescence and into emerging adulthood found highly similar patterns of instability (Burt, McGue, & Iacono, 2010). Nor are these findings specific to youth psychopathology. Low levels of non-shared environmental stability over time have also been reported for outcomes as disparate as adolescent perceptions of the parent-child relationship (McGue, Elkins, Walden, & Iacono, 2005) and self-reported personality during emerging adulthood (Hopwood et al., 2011).

These findings of temporal instability for the non-shared environment have been as consistent as they are surprising, and have led to recent speculations that non-shared influences may be largely idiosyncratic and unsystematic in nature prior to adulthood (Burt, 2009; Burt, McGue, & Iacono, 2009; Burt et al., 2006; Rutter et al., 1999; Turkheimer & Waldron, 2000). There are two critical caveats to this conclusion, however. First, although there is little evidence of non-shared environmental stability prior to adulthood, recent work has reported moderate levels of non-shared environmental stability during adulthood. For example, Hopwood et al. (2011) examined personality development in 626 twin pairs at ages 17, 24, and 29 years. Results revealed that only 13-15% of the non-shared environmental influences on negative and positive emotionality at age 17 persisted to age 24, whereas 31-36% persisted from age 24 to 29 (Hopwood et al., 2011).

Second, the above results do not rule out non-shared environmental continuity over shorter assessment intervals (i.e., temporally-limited stability), particularly since the vast majority of child and adolescent twin studies have examined etiologic influences across lags of a year or more (with 3-5 years being the most typical). Although these long assessment intervals certainly underscore the high levels of genetic and shared environmental stability observed over time, they also render prior findings for the non-shared environment difficult to interpret. It is entirely possible, for example, that the non-shared environment evidences higher levels of stability across shorter (but still meaningful) intervals of weeks or days. If true, such findings would suggest that, rather than reflecting unsystematic and idiosyncratic variance, the non-shared environment is comprised of environmental influences that shape behavior over a given set of weeks or days, but that these effects shift over time.

The current study sought to more rigorously evaluate non-shared environmentally-mediated stability in interpersonal/emotional behaviors prior to adulthood by examining far shorter assessment intervals. We first examined self-reported negative and positive affect as collected daily for seven days in a sample of 239 twin pairs in late adolescence/emerging adulthood. We then examined child interpersonal warmth and control as assessed via a seven-minute interaction in a sample of 687 twin pairs in middle childhood (Klahr, Thomas,

Hopwood, Klump, & Burt, 2013). Subjecting these daily and minute-by-minute slices of behavior to standard longitudinal twin modeling at multiple developmental stages (i.e., childhood and late adolescence/emerging adulthood) should allow us to more definitively answer questions regarding the stability of non-shared environmental influences prior to adulthood.

METHODS

Participants

Specific sample sizes for each study are detailed below. Both samples were recruited as part of the Michigan State University Twin Registry (MSUTR; $N \sim 25,000$ twins). Recruitment procedures for the MSUTR are detailed in prior publications (Burt & Klump, 2013; Klump & Burt, 2006). Current response rates range from 54-75%, which are as good or better than those of other twin registries that use similar types of anonymous recruitment mailings (Baker, Barton, & Raine, 2002; Hay, McStephen, Levy, & Pearsall-Jones, 2002). Moreover, participating families have been shown to be representative of both families residing in the recruitment area and recruited families more specifically (Burt & Klump, 2013).

Sample 1—Participants in the daily study were 239 same-sex female twin pairs in late adolescence/emerging adulthood (aged 16-25 years) who participated in the Twin Study of Hormones and Behavior across the Menstrual Cycle project from the MSUTR. This longitudinal study examines associations between changes in ovarian hormones and changes in disordered eating across the menstrual cycle. Because this study focuses on ovarian hormone functioning, several inclusion and exclusion criteria were needed, including: 1) menstruation every 22-32 days for the past 6 months; 2) no hormonal, psychotropic or steroid medications within the past 8 weeks; 3) no pregnancy or lactation within the past 6 months; and 4) no history of genetic or medical conditions known to influence hormone functioning or appetite/weight. Zygosity was established using physical similarity questionnaires administered to the twins (Peeters, Van Gestel, Vlietinck, Derom, & Derom, 1998). There were 133 MZ pairs and 106 DZ pairs. On average, the physical similarity questionnaires used by the MSUTR have accuracy rates of 95% or better.

Sample 2—Participants in the minute-by-minute study were 687 child twin families (comprising 1,374 mother-child dyad pairs) assessed as part of the on-going Twin Study of Behavioral and Emotional Development in Children (TBED-C), an independent study within the MSUTR. The TBED-C includes two independent samples: 1) a population-based sample of child twins and their parent(s) (current $N=500$ families); 2) an “at-risk” sample of child twins and their parent(s), for which families were also required to reside in moderately to severely disadvantaged neighborhoods to be eligible for participation (observer-ratings are available for 187 families as of now). The twins were 53.4% male and ranged in age from 6 to 10 years (mean = 8.16, $SD = 1.45$; although a few twins had turned 11 by the time they participated). Zygosity was again established using physical similarity questionnaires administered to the twins’ primary caregiver (Peeters et al., 1998). There were 311 MZ pairs and 376 DZ pairs in the minute-by-minute study.

Procedures

Sample 1—All study measures and procedures were approved by the Michigan State University Institutional Review Board (IRB). Participants provided daily behavioral data for 45 consecutive days. Because negative and positive affect may change across the menstrual cycle, the current study focused exclusively on women in the follicular phase of their cycle (days 2-8). Questionnaires were completed each evening (after 5:00 PM) using an online data system or pre-printed scantrons. The Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988) was included in the questionnaire packet and was used in the present study to measure negative (i.e., depression and anxiety) and positive (i.e., alert, excited) affect via the Negative and Positive Affect scales. These scales exhibit good convergent and discriminant validity (Watson et al., 1988) and excellent internal consistency (average $\alpha = .85$ in our sample). To adjust for positive skew, NA was log-transformed prior to analysis. Means and standard deviations for NA and PA are presented in the Appendix I. Phenotypic correlations for mood across days are presented in the Appendix III.

Sample 2—All study measures and procedures were approved by the Michigan State University IRB. Children gave informed assent, while parents gave informed consent for themselves and their children. In the current task, each mother-child dyad was asked to use an Etch-a Sketch to draw specific pictures, but each participant could only use one dial, thereby requiring their cooperation (Deater-Deckard, Pylas, & Petrill, 1997). This mildly to moderately frustrating task was originally designed for use in child twin families, and has been found to be a reliable and valid tool for assessing interpersonal behaviors and the parent-child relationship in school-age children.

Trained raters coded interpersonal behaviors using a computer joystick apparatus (the Microsoft Sidewinder Force Feedback 2) and related software (Klahr et al., 2013; Sadler, Ethier, Gunn, Duong, & Woody, 2009). The joystick-monitoring software program displays a Cartesian plane on the computer screen and depicts the axes of interpersonal warmth and interpersonal control. A dot in the Cartesian plane moved in accordance with the current position of the joystick. The software program recorded the joystick position within the Cartesian plane (i.e., the x and y coordinates) twice per second. The scale on both axes ranged from -1,000 to 1,000. Coding was completed separately for each participant (i.e., each mother-child dyad was rated twice, once for the mother and once for the child). Warm behaviors included social smiling or leaning towards the other person, verbal praise, eye contact, and warm physical contact (such as a hug or pat on the back) whereas cold behaviors included looking away, unresponsiveness, and rude or sarcastic comments. Controlling behaviors included giving instructions and grabbing the etch-a-sketch toy whereas submissive behaviors included following the other person's lead and asking for permission.

To minimize idiosyncratic interpretations/random error by individual coders, coders were divided into teams of three to four raters, all of whom coded the interactions assigned to that team. Ratings were averaged across coders to obtain a composite rating for each moment of the interaction, as suggested by Sadler et al. (2009). The composite ratings of each moment were then averaged across all moments, separately for each minute of the interaction,

creating mean scores of warmth and control for each member of the dyad for each minute of the interaction (i.e., children each received an overall score for both control and warmth during minutes 0-1, 1-2, 2-3, 3-4, 4-5, 5-6, 6-7).¹ Importantly, different teams coded the two dyads within a given family (e.g., mother and twin 1; mother and twin 2), thereby eliminating shared “informant” effects on parameter estimates. For the current study, we focused solely on child behavior in order to facilitate straightforward interpretation of genetic and environmental effects (Klahr & Burt, 2014). In particular, because the twin children are the genetically-informative piece of this design, genetic influences on child interpersonal behavior index the influence of the child’s genes on his or her behavior (Klahr & Burt, 2014). Genetic effects on maternal behavior in this design, by contrast, index evocative rGE effects, which are not of interest for the current study. To ensure that our results were not influenced by twin age, sex, or ethnicity, these variables were regressed out of control and warmth prior to analysis (McGue & Bouchard, 1984).

Inter-rater reliability was calculated for the time-series and the minute averages. Moment-by-moment reliability (i.e., the correlation between raters over a time-series) was good for control and acceptable for warmth ($r = .86$ and $.68$, respectively). Inter-rater reliabilities for the minute averages were also calculated for each of the coding teams using Cronbach’s alpha (average alphas across coding teams were $.90$ for control and $.64$ for warmth). Means and standard deviations for warmth and control are presented in the Appendix II. Phenotypic correlations for interpersonal behavior across the 7 minute interaction are presented in the Appendix IV.

Statistical Analyses

The same set of analyses was conducted separately for each of the four phenotypes. Initial analyses centered on the correlated factors model (see Figure 1), which decomposes both the variance within assessments and the covariance across assessments into their genetic and environmental components (e.g., average negative affect on days 0-1, 1-2, 2-3, 3-4, 4-5, 5-6, and 6-7). These genetic (A), shared (C), and non-shared (E) environmental covariances can then be standardized on their respective variances to produce genetic, shared environmental, and non-shared environmental correlations. These statistics reveal the extent to which a specific effect (e.g., the genetic effect) during a given assessment is correlated with the same effect during a subsequent assessment. A genetic correlation of 0, for example, would indicate that none of the estimated genetic influences overlapped across assessments. A genetic correlation of 1.0, by contrast, would indicate that 100% of the genetic influences overlap across assessments.

Because there was a small amount of missing data (e.g., 2.3% of videos were not codeable, daily measures were occasionally skipped, etc.), we made use of Full-Information Maximum-Likelihood raw data techniques (FIML), which produce less biased and more efficient and consistent estimates than techniques like pairwise or listwise deletion in the face of missing data (Little & Rubin, 1987). Mx (Neale, Boker, Xie, & Maes, 2003) was used to fit models to the raw data. When fitting models to raw data, variances, covariances,

¹As in Klahr et al. (2013), the final minute of the 8-minute interaction was not analyzed since many dyads discontinued the task prior to the full eight minutes.

and means of those data are first freely estimated by minimizing minus twice the log-likelihood ($-2\ln L$). The minimized value of $-2\ln L$ in the baseline model was then compared with the $-2\ln L$ obtained in the biometric models to yield a likelihood-ratio chi-square test. The chi-square was then converted to the Akaike's Information Criterion (AIC; $AIC = X^2 - (2 \cdot df)$; Akaike, 1987), so as to measure model fit relative to parsimony. Lower AIC values reflect a better fit to the data, and were used to determine the best-fitting model from a series of fitted models.

We then made use of the Q-test for homogeneity (which is distributed as a chi-square) to statistically evaluate whether the genetic and environmental correlations were homogenous with respect to lag length (e.g., do the correlations change significantly with increasing lag length?). Analyses were conducted separately for each of the four phenotypes using a mixed effects analog to the one-way ANOVA (Lipsey & Wilson, 2001; Wilson, 2005), estimated via iterative maximum likelihood. Consistent with recommendations (Lipsey & Wilson, 2001; Wilson, 2005), individual correlations were converted to z-statistics using Fisher's r to z transformation prior to analysis. The resulting effect sizes were then weighted using a combination of inverse variance weights and the random effects variance component (Lipsey & Wilson, 2001; Wilson, 2005). Following analyses, these estimates were converted back into correlations using an inverse z to r transformation to facilitate interpretation.

RESULTS

The best-fitting correlated factors models varied across the four phenotypes. For the daily measures of negative and positive affect in sample 1, the AE model provided an improved fit to the data (NA: $X^2 = 223.20$ on 175 df, $p = .008$, $AIC = -126.80$; PA: $X^2 = 177.92$ on 175 df, $p = .42$, $AIC = -172.08$) relative to the ACE model (NA: $X^2 = 220.41$ on 147 df, $p < .001$, $AIC = -73.59$; PA: $X^2 = 177.20$ on 147 df, $p = .045$, $AIC = -116.80$). For child interpersonal warmth (assessed as part of sample 2), the E model provided the best fit to the data (ACE: $X^2 = 666.26$ on 147 df, $p < .001$, $AIC = 372.26$; AE: $X^2 = 670.50$ on 175 df, $p < .001$, $AIC = 320.50$; CE: $X^2 = 669.68$ on 175 df, $p < .001$, $AIC = 319.68$; E: $X^2 = 699.72$ on 203 df, $p < .001$, $AIC = 293.72$). For child interpersonal control (also assessed as part of sample 2), the full ACE model provided the best fit to the data ($X^2 = 585.27$ on 147 df, $p < .001$, $AIC = 291.27$). As genetic and environmental correlations between non-significant variance components may or may not be meaningful, we focused our heterogeneity analyses on the correlations generated from the best-fitting models. Univariate ACE estimates from those models are presented in Table I.

The genetic and environmental correlations for negative and positive affect are presented in Table II. As seen there, genetic correlations approached unity for both positive and negative affect, and did so regardless of the number of days between assessments. In sharp contrast, non-shared environmental correlations appeared to decrease more or less monotonically with increasing lag length. Highly similar results were obtained for child interpersonal control (see Table III), such that genetic and shared environmental correlations approached unity whereas non-shared environmental correlations again decreased monotonically with increasing lag length. Non-shared environmental correlations for child interpersonal warmth similarly decreased with increasing lag lengths.

Homogeneity analyses were conducted to statistically evaluate these early impressions. We began with the daily results obtained from sample 1 (see Table IV). As seen there, there was little-to-no evidence of heterogeneity in genetic correlations with lag length. They were consistently estimated to be at or near unity, indicating that the genetic influences present on a given day overlap entirely with those during subsequent days over the week. By contrast, there was evidence of considerable and consistent heterogeneity in non-shared environmental correlations with lag length for both positive and negative affect, such that the average non-shared environmental correlation decreased monotonically with increasing lag length, and did so rather dramatically. For example, while a small portion of the non-shared environmental variance in NA persisted across sequential days ($.38^2 = 14.4\%$), only a very small amount (2.0%) persisted for up to 5 days. Similar results were obtained for PA: 24.0% of the non-shared environmental variance persisted across sequential days while only 9.6% persisted up to 5 days.

The same pattern of results could also be seen when examining continuity across minutes (see Table V). The genetic and shared environmental correlations on child interpersonal control were consistently estimated to be at or near unity, although there was evidence of some decrease in genetic stability with increasing lag length (r_A dropped from 1.00 to .81; such results may reflect the increasing heritability of interpersonal control over the course of the interaction, as seen in Table I). By contrast, although more than half (57.8%) of the non-shared environmental influences on child interpersonal control in a given minute persisted to the subsequent minute, the introduction of even a one minute lag between assessments reduced this non-shared environmental stability by nearly half (to 33.6%). This decay in non-shared environmental stability was further accentuated by longer lags, such that only 7.3% of the non-shared influences on child interpersonal control present during the first minute of the interaction were still present during the final minute of the interaction. A notably similar pattern was observed when examining child warmth, even though E accounts for 100% of the variance in this phenotype.

DISCUSSION

The primary aim of the present study was to examine the persistence of genetic, shared, and non-shared environmental influences for several emotional/interpersonal behaviors over the span of two different, but very short, assessment intervals. Results revealed that, as predicted, genetic and shared environmental influences persisted more or less in full across these short assessment intervals, such that 64-100% of the genetic and shared environmental influences on the various phenotypes continued to be important up to 5 days later. By contrast, non-shared environmental correlations decreased monotonically with increasing lag length and did so even across assessment intervals as short as 5 minutes. Such results provide an important extension of previous twin and adoption studies (Burt, et al., 2010; Hopwood et al., 2011; McGue et al., 2005; Rutter et al., 1999; Turkheimer & Waldron, 2000), and imply that non-shared influences on many emotional/interpersonal behaviors may be idiosyncratic and unsystematic in nature prior to adulthood.

There are several limitations to bear in mind when interpreting the results of this study. First, the current results apply only to childhood and emerging adulthood, as there is evidence that

the non-shared environment becomes less idiosyncratic during adulthood (Dickens, Bean, & Turkheimer, 2009; Hopwood et al., 2011). It thus remains unclear whether these findings would generalize to later developmental stages. The lack of genetic and shared environmental influences on child interpersonal warmth is important to mention. Observer-ratings of child temperament generally show small to moderate genetic effects (Matheny, 1983; Saudino, 2005), although no published studies have examined the heritability of child interpersonal warmth. The reliability of our observer-ratings of interpersonal warmth was lower than interpersonal control, suggesting that increased measurement error may have contributed to inflated estimates of E, at the expense of A and/or C (particularly as we were unable to isolate measurement error from our estimates of E). However, the presence of moderate to large phenotypic correlations across minutes argues against this interpretation as a sole explanation for these findings (see Appendix IV), since unsystematic measurement error should not persist over time. To further probe the stability of E, future research might use a design that allows for estimates of E that are free of measurement error (e.g., common factor models). It is also important to note that our sample for daily ratings of affect only included females, as these data were pulled from a study involving menstrual cycles. Men and women differ somewhat in mean levels of positive and negative affect (Crawford & Henry, 2004), although it is unclear whether there are etiological differences by sex. We did attempt to circumvent the potential impact of menstrual cycle variability on E instability by only including affect ratings from the follicular phase of the menstrual cycle. However, further research should seek to confirm our findings across sexes. Finally, although our results were consistent across several domains of observed and reported behavior, additional research is needed to more firmly establish the stability of non-shared environmental influences on other phenotypes during childhood, adolescence, and emerging adulthood.

Despite these limitations, the current study has several important and interrelated implications. First, genetic influences on interpersonal control, positive affect, and negative affect performed precisely as most behavioral geneticists would have predicted given the very short assessment intervals: they persisted almost in their entirety to all other days/minutes in the study. This stability in genetic effects is fully consistent with that observed in other longitudinal work (Bartels et al., 2004; Burt, Carter, McGue, & Iacono, 2007; Haberstick, Schmitz, Young, & Hewitt, 2006; Hopwood et al., 2011; McGue et al., 2005), and serves to reinforce extant notions regarding the critically important role of genetics in shaping human behavior.

The current study also serves to enhance our understanding of the role of the environment on behavior. Behavioral genetic research has historically concluded that the more important environmental influences on psychological and behavioral outcomes result in differences between siblings (Plomin & Daniels, 1987), a conclusion that continues to influence theory and interpretation up to the present day. More recent research conducted during childhood and adolescence, however, has argued against this proposition. This work has instead indicated that the shared environment is moderate in magnitude for many childhood phenotypes (Burt, 2009), evidences high levels of stability across intervals of even several years (Burt et al., 2010), and is readily identifiable (Burt, Barnes, McGue, & Iacono, 2008; Burt, Krueger, McGue, & Iacono, 2003; Burt, McGue, Krueger, & Iacono, 2007; Klahr, McGue, Iacono, & Burt, 2011). The current findings do little to undercut these conclusions,

in that shared environmental influences were observed to be significant and moderate in magnitude for one of the two child phenotypes examined here (child control). Moreover, when present, they persisted in their entirety across all seven minutes of the interaction.

By contrast, the current study found that non-shared environmental influences did not persist to any noteworthy extent across days or even minutes for any of the four emotional/interpersonal phenotypes examined herein. Indeed, less than 20% of the non-shared influences on child interpersonal control and warmth during the first minute of the interaction were still present during the final minute of the interaction. Such findings imply that, at least prior to adulthood, non-shared influences on emotional/interpersonal behaviors may be largely idiosyncratic and unsystematic in nature, a conclusion bolstered by the fact that efforts to identify non-shared environmental influences have largely failed. Indeed, specific non-shared environmental factors typically account for no more than 2% of the variance in the outcome (Turkheimer & Waldron, 2000). When viewed in conjunction with these findings, the current results suggest that the non-shared environment is comprised primarily of idiosyncratic and unsystematic effects (including measurement error) with relatively little predictive utility prior to adulthood, at least at the between-persons level. That said, recent innovations in modeling (Molenaar, Smit, Boomsma, & Nesselrode, 2012) now allow researchers to estimate genetic and environmental influences at the subject-specific (or idiographic) level rather than solely at the between-persons (or nomothetic) level. This novel approach to examining the origins of human behavior may well revolutionize the field, not least of which because it may allow us to identify meaningful non-shared environmental effects at the subject-specific level. Future work should seek to do just this.

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Appendix

Appendix I

Means and Standard Deviations for Negative and Positive Affect Measured Daily for Seven Days.

	Negative Affect		Positive Affect	
	M	SD	M	SD
Day 2	14.92	5.04	23.86	7.61
Day 3	15.11	5.16	23.63	7.70
Day 4	14.76*	5.31	23.52	8.02
Day 5	15.41*	5.64	23.19	7.85
Day 6	14.79	5.16	22.82	7.99
Day 7	14.72	5.14	23.07	7.93

	Negative Affect		Positive Affect	
	M	SD	M	SD
Day 8	14.78	5.42	22.31	7.87

Note.

* mean is significantly different from the mean of the previous day, $p < .05$.

Appendix II

Means and Standard Deviations for Child Interpersonal Control and Warmth Measured Across Seven Consecutive Minutes.

	Control		Warmth	
	M	SD	M	SD
Minute 1	-18.66	219.35	61.03	101.22
Minute 2	-88.19**	342.59	102.78**	154.84
Minute 3	-94.21	386.75	139.28**	182.82
Minute 4	-122.36**	426.02	164.74**	207.56
Minute 5	-136.61*	436.65	192.87**	221.94
Minute 6	-144.64	444.96	214.24**	235.56
Minute 7	-150.58	458.88	229.26**	251.70

Note. Mean is significantly different from the mean of the previous minute

* $p < .05$

** $p < .01$.

Appendix III

Phenotypic Correlations for Negative and Positive Affect Measured Daily for Seven Days.

Positive Affect							
	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8
Day 2	-						
Day 3	.58**	-					
Day 4	.61**	.60**	-				
Day 5	.51**	.54**	.56**	-			
Day 6	.51**	.58**	.57**	.60**	-		
Day 7	.47**	.52**	.52**	.57**	.65**	-	
Day 8	.49**	.52**	.54**	.55**	.62**	.67**	-
Negative Affect							
Day 2	-						
Day 3	.54**	-					
Day 4	.53**	.57**	-				
Day 5	.44**	.46**	.58**	-			
Day 6	.52**	.48**	.49**	.56**	-		
Day 7	.41**	.40**	.37**	.47**	.53**	-	
Day 8	.41**	.43**	.42**	.44**	.49**	.57**	-

Note.

**
 $p < .01$.

Appendix IV

Phenotypic Correlations for Child Interpersonal Control and Warmth Measured Across Seven Consecutive Minutes.

Child Control							
	Minute 1	Minute 2	Minute 3	Minute 4	Minute 5	Minute 6	Minute 7
Minute 1	-						
Minute 2	.83**	-					
Minute 3	.71**	.86**	-				
Minute 4	.66**	.78**	.89**	-			
Minute 5	.62**	.74**	.79**	.89**	-		
Minute 6	.57**	.69**	.75**	.81**	.90**	-	
Minute 7	.54**	.64**	.72**	.76**	.82**	.90**	-
Child Warmth							
Minute 1	-						
Minute 2	.76**	-					
Minute 3	.63**	.83**	-				
Minute 4	.57**	.73**	.86**	-			
Minute 5	.50**	.67**	.73**	.86**	-		
Minute 6	.47**	.61**	.70**	.77**	.87**	-	
Minute 7	.45**	.57**	.68**	.74**	.78**	.89**	-

Note.

**
 $p < .01$.

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Note. The variance in each phenotype (as measured daily or minute-by-minute) is parsed into additive genetic effects (A1 through A7, not pictured), shared environmental effects (C1 through C7, not pictured), and non-shared environmental effects (E1 through E7). The genetic, shared environmental, and non-shared environmental correlations (those for E are represented above by double headed

Figure I.
A 7-variable Correlated Factors Model.

Table I

Univariate estimates of Genetic (A), Shared (C), and Non-shared (E) Environmental Influences on Daily and Minute-by-Minute Measures.

Phenotype	Model	A	C	E	Total unstandardized variance
Daily measures of negative affect (sample 1)	Day 2	.39 *	--	.61 *	.089
	Day 3	.31 *	--	.69 *	.090
	Day 4	.47 *	--	.54 *	.094
	Day 5	.27 *	--	.73 *	.101
	Day 6	.45 *	--	.56 *	.091
	Day 7	.31 *	--	.69 *	.090
	Day 8	.31 *	--	.69 *	.095
Daily measures of positive affect (sample 1)	Day 2	.25 *	--	.74 *	58.86
	Day 3	.34 *	--	.66 *	59.61
	Day 4	.25 *	--	.75 *	64.36
	Day 5	.34 *	--	.66 *	61.96
	Day 6	.28 *	--	.72 *	64.32
	Day 7	.32 *	--	.68 *	62.17
	Day 8	.28 *	--	.72 *	62.94
Minute-by-minute observations of child interpersonal control (sample 2)	Minute 1	.16 *	.18 *	.66 *	45,589
	Minute 2	.19 *	.25 *	.57 *	106,810
	Minute 3	.22 *	.24 *	.54 *	135,880
	Minute 4	.25 *	.24 *	.52 *	167,220
	Minute 5	.32 *	.20 *	.47 *	174,960
	Minute 6	.40 *	.14 *	.46 *	179,190
	Minute 7	.40 *	.13 *	.47 *	193,670
Minute-by-minute observations of child interpersonal warmth (sample 2)	Minute 1	--	--	1.00 *	10,106
	Minute 2	--	--	1.00 *	23,176
	Minute 3	--	--	1.00 *	32,470
	Minute 4	--	--	1.00 *	40,880
	Minute 5	--	--	1.00 *	47,244
	Minute 6	--	--	1.00 *	53,467
	Minute 7	--	--	1.00 *	61,976

Note.

* indicates that the estimate is significantly larger than zero at $p < .05$. To adjust for positive skew, NA was log-transformed prior to analysis. Interpersonal warmth and control scores ranged from -1000 to $+1000$.

Table II
Genetic (rA) and Non-shared Environmental (rE) Correlations for Negative and Positive Affect Measured Daily for Seven Days.

	rA for Negative Affect					rE for Negative Affect					rA for Positive Affect					rE for Positive Affect								
No lag/sequential (e.g., T1-T2)	.97	.94	.81	.90	.97	.96	.37	.33	.45	.39	.30	.42	.87	.88	.87	.86	.92	.98	.45	.48	.42	.49	.53	.54
1 day lag (e.g., T1-T3)	.94	.94	.94	.94	.94		.23	.27	.12	.31	.27		.96	.97	.90	.79	.98		.49	.32	.46	.45	.47	
2 day lag (e.g., T1-T4)	.91	.99	.90	.99			.26	.20	.07	.23			.80	.95	.98	.81			.38	.40	.33	.43		
3 day lag (e.g., T1-T5)	.99	.99	.85				.19	.19	.13				.96	.84	.94				.33	.35	.40			
4 day lag (e.g., T1-T6)	.94	.97					.18	.17					.99	.89					.25	.36				
5 day lag (e.g., T1-T7)	.95						.14						1.0						.31					

Note. The correlations were obtained via a biometric seven-variable correlated factors model on a sample of 239 twin pairs. The best-fitting model was the AE model; hence, only rA and rE are presented here.

Genetic (rA), Shared Environmental (rC), and Non-shared Environmental (rE) Correlations for Child Interpersonal Control and Warmth Measured Across Seven Consecutive Minutes.

Table III

	rA for Control					rC for Control					rE for Control					rE for Warmth								
No lag/sequential (e.g., T1-T2)	.99	.95	1.0	1.0	.98	1.0	.96	.99	1.0	1.0	1.0	.97	.72	.75	.78	.76	.79	.77	.76	.82	.86	.86	.87	.88
1 minute lag (e.g., T1-T3)	.89	.97	1.0	.98	.98		.97	1.0	1.0	.99	.96		.54	.58	.56	.61	.62		.63	.72	.72	.77	.78	
2 minute lag (e.g., T1-T4)	.92	.97	.99	.98			.97	1.0	1.0	.96			.44	.50	.50	.54			.56	.65	.69	.73		
3 minute lag (e.g., T1-T5)	.91	.91	.98				.95	.99	.98				.37	.44	.45				.49	.59	.67			
4 minute lag (e.g., T1-T6)	.83	.90					.95	.94				.34	.38						.46	.55				
5 minute lag (e.g., T1-T7)	.81						.97					.27							.43					

Note. The correlations were obtained via the best-fitting seven-variable correlated factors model for child interpersonal control and warmth, respectively, on a sample of 687 twin pairs.

Table IV

Mean Genetic (rA), Shared Environmental (rC), and Non-shared Environmental (rE) Correlations for Negative Affect (NA) and Positive Affect (PA), measured daily for seven days.

Lags	Mean rA NA	Mean rE NA	Mean rA PA	Mean rE PA
No lag/ sequential days (e.g., T1-T2)	.94 (.89, .97)	.38 (.33, .42)	.91 (.83, .95)	.49 (.45, .53)
1 day lag (e.g., T1-T3)	.94 (.89, .97)	.24 (.19, .29)	.94 (.89, .97)	.44 (.39, .48)
2 day lag (e.g., T1-T4)	.97 (.93, .99)	.19 (.13, .25)	.92 (.82, .96)	.39 (.33, .43)
3 day lag (e.g., T1-T5)	.97 (.93, .99)	.17 (.10, .24)	.93 (.83, .97)	.36 (.30, .43)
4 day lag (e.g., T1-T6)	.95 (.86, .98)	.18 (.09, .26)	.97 (.90, .99)	.31 (.22, .39)
5 day lag (i.e., T1-T7)	.95 (.78, .99)	.14 (.01, .26)	1.00 (.98, 1.00)	.31 (.19, .42)
Between-Group Q (on 5 df)	3.65	43.37 *	16.13 *	27.54 *
p-value	.60	<.0001	.007	<.0001

Note. Weighted averages of the genetic, shared, and non-shared environmental correlations, reported separately by the length of lag between assessments. 95% confidence intervals are presented below the estimate in brackets. All correlations with confidence intervals that do not overlap with zero are significantly greater than zero.

* indicates that the correlations varied significantly across lag length (as evidenced by a statistically significant between-groups Q).

Table V

Mean Genetic (rA), Shared Environmental (rC), and Non-shared Environmental (rE) Correlations for Child Interpersonal Control and Warmth by Minute Intervals.

Lags	Mean rA CONTROL	Mean rC CONTROL	Mean rE CONTROL	Mean rE WARMTH
No lag/ sequential (e.g., T1-T2)	1.00 (.99, 1.00)	.99 (.99, 1.00)	.76 (.75, .78)	.85 (.82, .87)
1 minute lag (e.g., T1-T3)	.98 (.93, .99)	.99 (.98, 1.00)	.58 (.56, .61)	.72 (.68, .77)
2 minute lag (e.g., T1-T4)	.97 (.89, .99)	.99 (.97, 1.00)	.49 (.46, .53)	.66 (.60, .72)
3 minute lag (e.g., T1-T5)	.95 (.77, .99)	.98 (.93, .99)	.42 (.38, .46)	.59 (.50, .66)
4 minute lag (e.g., T1-T6)	.87 (.35, .98)	.95 (.80, .99)	.36 (.30, .41)	.51 (.39, .61)
5 minute lag (i.e., T1-T7)	.81 (.21, .99)	.97 (.81, 1.00)	.27 (.19, .35)	.43 (.25, .59)
Between-Group Q (on 5 df)	19.74 *	9.77	603.86 *	111.22 *
p-value	.0014	.082	<.0001	<.0001

Note. Weighted averages of the genetic, shared, and non-shared environmental correlations, reported separately by the length of lag between assessments. 95% confidence intervals are presented below the estimate in brackets. All correlations with confidence intervals that do not overlap with zero are significantly greater than zero.

* indicates that the correlations varied significantly across lag length (as evidenced by a statistically significant between-groups Q).