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More than two decades after adoption: Associations between infant attachment, early maternal sensitivity and the diurnal cortisol curve of adopted young adults

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Abstract

The focus of this study was on the longitudinal relation between infant attachment, early maternal sensitivity and the diurnal cortisol curve of adopted young adults. 86 adoptees (mean age at adoption 11 weeks) were followed from infancy to young adulthood. Attachment quality and maternal sensitivity were observed in infancy. When the adoptees were 23 years of age saliva samples were collected at six time points across the day, on two different days. To assess compliance to the instruction the Medication Event Monitoring System (MEMS) was used. The data were analyzed with growth models using multilevel analyses (Day 1) and structural equation modeling (as a more confirmatory approach, Day 2). Results revealed no associations between early attachment security, attachment disorganization, maternal sensitivity and the diurnal cortisol curve more than two decades later. Attachment experiences (in the normative range) may not induce changes in the later diurnal cortisol curve in the same way as severe chronic stressors do. Alternatively, adoption related experiences may dampen associations between attachment experiences and the diurnal cortisol curve in later life.

Keywords: adoption; sensitive parenting; attachment; cortisol
1. Introduction

Early caregiving experiences affect the Hypothalamic-Pituitary-Adrenal axis (HPA axis) that is involved in the secretion of cortisol (Hostinar & Gunnar, 2013). Adoptees have experienced a major separation from at least one primary caregiver early in their lives, and have often lived in adverse circumstances before adoption (Van IJzendoorn & Juffer, 2006). However, in their adoptive families adoptees get chances for new positive experiences. Although the effects of social relationships on the stress-system in early childhood have been widely documented, less is known about the effects of observed attachment related experiences in early life on the functioning of the HPA-axis in later life (see for reviews Hostinar & Gunnar, 2013; Hostinar, Sullivan, & Gunnar, 2014). In this study we examine these long-term effects in a sample of young adults who were adopted at an early age.

1.1 Development of Adoptees

Many studies have highlighted the protective factors and the risks associated with adoption. In general, adoption appears to be a successful intervention. Several meta-analyses have shown that adopted children are able to at least partly redress the balance for incurred delays in areas such as physical development, cognitive development, self-esteem, and attachment security and disorganization (Van IJzendoorn & Juffer, 2006). Although these results provide an optimistic picture on the developmental outcomes of adopted children, it seems that these children do not catch up on all accounts. For example, compared to their non-adopted current peers, adopted children are at risk of developing insecure or disorganized attachment relationships (Van den Dries, Juffer, Van IJzendoorn, & Bakermans-Kranenburg, 2009) and problem behavior (Juffer & Van IJzendoorn, 2005), especially when they have experienced higher levels of deprivation. It might be the case that the (re)programming of biological processes through early experiences is associated with these developmental risks (Nelson, Fox, & Zeanah, 2014). It is clear that more knowledge of the long-term interplay between early negative experiences, possible corrective experiences and neurobiological processes that play a role in the development of adoptees is needed (Palacios & Brodzinsky, 2010).

1.2 The Attachment Relationship and Stress Regulation

The quality of the attachment relationship with the (adoptive) parent is of crucial importance for early and later child outcomes. Securely attached children experience their parent as a safe haven from which they can explore the world (Bowlby, 1969). Secure attachment relationships have proven to be predictive of beneficial developmental outcomes such as better social development (see for a meta-analysis Groh et al., 2014), and fewer externalizing and internalizing behavior problems (see for two meta-analyses Fearon, Bakermans-Kranenburg, Van IJzendoorn, Lapsley, &
In general, securely attached children are better able to cope with stress. They usually have a history of sensitive caregiving and learn to rely on the availability of their parent (Ainsworth, Blehar, Waters, & Wall, 1978). Several studies have documented the buffering effect of sensitive parenting and secure attachment relationships on the biological reactions to stressors (e.g., Luijk et al; 2010; Oosterman, De Schipper, Fisher, Dozier, & Schuengel, 2010; Spangler & Schieche, 1998).

1.3 Stress Regulation Through HPA-axis Functioning
The effects of early attachment experiences on the stress-system can be examined by measuring cortisol levels in saliva or blood. The HPA-axis is the biological system that regulates the secretion of cortisol. In general, the diurnal cortisol curve shows relatively high levels of cortisol in the morning that rapidly increase even more in the first half hour after awakening. This increase is known as the Cortisol Awakening Response (CAR: e.g., Fries, Dettenborn, & Kirschbaum, 2009). During the rest of the day cortisol levels decrease (Hostinar & Gunnar, 2013). Apart from this diurnal pattern of cortisol secretion, cortisol levels rise in reaction to stressful situations in order to mobilize energy (Hostinar & Gunnar, 2013). Abnormal patterns of cortisol secretion during the day (e.g., Fries et al., 2009) as well as in response to stress are associated with physical problems (Miller, Chen, & Zhou, 2007) and negative behavioral and psychological outcomes such as problem behavior (e.g. Alink et al., 2008; McBurnett, Lahey, Rathouz, & Loeber, 2000) and psychopathology (Buitelaar, 2013).

Deviant patterns of cortisol secretion may be induced by early experiences of stress (see for a meta-analysis Miller et al., 2007) such as the deprivation and separations that many adoptees have gone through. Several explanations for the effects of chronic stress on HPA-axis functioning have been put forward. It has been proposed that chronic stress contributes to increased levels of cortisol and that this increase results in illnesses and psychological problems.

However, in the last decades several studies found that experiences of stress were related to lower levels of cortisol, or so-called ‘hypocortisolism’ (Heim, Ehlert, & Hellhammer, 2000). Early (over-)stimulation of the HPA-axis due to negative experiences may lead to a down-regulation over time, with lower basal cortisol levels and a less steep decline of cortisol levels during the day mostly resulting from lower morning levels (see for a review Gunnar & Vazquez, 2001). Hypercortisolism may be relevant when examining short-term effects of stress, while hypocortisolism may be more applicable to long-term effects of stress on the HPA-axis functioning (Miller et al., 2007).
1.4 Early Experiences and HPA-axis Regulation in (Early) Childhood
Adopted children with experiences of severe deprivation often show lower basal levels of cortisol and flatter diurnal slopes (Gunnar & Vasquez, 2001). Kroupina and colleagues (2012) found lower cortisol morning levels (30 min after awakening) for post-institutionalized toddlers (one month after adoption) compared to data from normative children. A follow-up assessment revealed that the morning cortisol values of these children had increased significantly after six months. However, not all studies report results in the same direction. Van den Dries and colleagues investigated the diurnal curves of girls adopted from foster care or institutional care in China when they were between 11 and 16 months old. Hardly any differences in diurnal curves were found between former foster children, previously institutionalized children, and non-adopted children, and cortisol patterns did not change between the assessments 2 and 6 months after arrival (Van den Dries, Juffer, Van IJzendoorn, & Bakermans-Kranenburg, 2010). A Ukrainian sample of 3 to 6-year-olds did not show differences in the cortisol pattern over the day between family-reared and institution-reared children either, but temporarily stunted children showed higher levels of cortisol production than family-reared or chronically stunted children (Dobrova-Krol, Van IJzendoorn, Bakermans-Kranenburg, Cyr, & Juffer, 2008). Gunnar, Morison, Chisholm, and Schuder (2001) followed a sample of school-age children who were raised in Romanian orphanages for at least the first 8 months of their lives. Their cortisol levels during the day were elevated compared to children adopted at an earlier age. The more time the children had spent in the institution, the higher the levels of cortisol were. Kertes, Gunnar, Madsen, and Long (2008) also found higher levels of cortisol for children aged 7 - 11 years who had experienced deprivation and showed growth delays at adoption.

In sum, most studies confirm that experiences of early deprivation affect the diurnal cortisol curve of young children. Flatter slopes and lower basal cortisol levels have been found, but also higher basal cortisol levels and non-deviant curves. When interpreting these results, one should keep in mind the developmental changes in basal cortisol levels (Gunnar & Donzella, 2002; Gunnar, Wewerka, Frenn, Long, & Griggs, 2009). Also timing, chronicity, specificity, controllability and severity of the negative experiences may specifically induce hypocortisolism or hypercortisolism and can therefore explain the different results that have been found (Miller et al., 2007).

1.5 Early Experiences and HPA-axis Regulation in Adolescence and Adulthood
It is evident that early negative experiences put adopted children at risk for maladaptive biological stress management in early life. Long-term effects that become evident in adolescence and adulthood may be different due to hormonal changes in the body (Gunnar, Wewerka, Frenn, Long, & Griggs, 2009) and possible adaptation of the stress system over time (Hostinar & Gunnar, 2013). Several studies have confirmed associations between severe adverse circumstances, such as maltreatment, and later
HPA-axis functioning (Hostinar & Gunnar, 2013). One longitudinal adoption study in the Netherlands has demonstrated that experiences of early neglect or abuse affected the cortisol curve of international adoptees in adulthood (Van der Vegt, Van der Ende, Kirschbaum, Verhulst, & Tiemeier, 2009). Severe neglect and abuse were associated with lower morning cortisol levels and severe neglect also with a flatter slope. Moderately severe abuse however was associated with higher morning cortisol levels and steeper slopes. Rearing experiences after adoption (as reported by the adoptee) did not change these results (Van der Vegt et al., 2009), but retrospective self-reports of experienced parenting may not fully reflect the reality of parenting practices.

1.6 The Present Study
To our knowledge, no studies have examined the long-term effects of early attachment relationship quality and maternal sensitivity on the daily cortisol curve of adopted adults. Studying this development from deprivation via adoption into early adulthood might clarify the contribution of childhood experiences on the functioning of the HPA-axis of adoptees. In this longitudinal adoption study we examined the effects of early attachment security and disorganization, and observed maternal sensitivity in early childhood on the daily cortisol curves of young adopted adults aged 23 years. All adoptees were adopted before the age of 6 months which makes it possible to study the effects of early attachment experiences without the confounding of long-term severe deprivation. We hypothesize that attachment security, attachment disorganization, and maternal sensitivity are associated with the height and slope of the diurnal cortisol curve, and the Cortisol Awakening Response (CAR) at age 23. Because of divergent results in the literature and the lack of outcomes on adopted adults we refrain from formulating specific expectations about the direction of these associations.

2. Method

2.1 Participants
In this longitudinal study, 86 internationally adopted young adults, 34 men and 52 women, participated in the collection of saliva when they were 23 years of age (mean age at adoption 11.23 weeks, SD = 5.16). They were born in Sri Lanka (n = 43), South Korea (n = 31), or Colombia (n = 12), and originated from a sample of 160 adopted children who were followed from infancy to young adulthood. All children in this study were adopted before the age of six months and were placed in Caucasian adoptive families with mainly middle-(upper)class backgrounds. The adoptive families were randomly recruited through Dutch adoption organizations. Some families did not have any (biological) children at the time of adoption (n = 46; Juffer, 1993), other families already had one or more biological or adopted children (n = 40; Rosenboom, 1994). In
all cases the mother was the primary caregiver (for more details see Stams, Juffer, & Van IJzendoorn, 2002). When their children were between 6 and 9 months of age, 26 of them were part of a randomly selected group of 50 families that received a short-term intervention aimed at promoting maternal sensitivity (Juffer, Bakermans-Kranenburg, & Van IJzendoorn, 2005).

2.2 Procedure
In infancy home visits were made at several points in time to administer questionnaires and observe mother-child interaction. Also, at 12, 18, and 30 months mother and child participated in lab sessions in which mother-child interaction was observed. At 7 and 14 years of age home and lab sessions were administered. At age 23, the adopted young adults visited the university to complete various assessments. They completed several questionnaires and collected saliva at home on two separate days.

2.3 Attrition
Of the 160 families who participated in infancy, 146 families participated in middle childhood, and a partly overlapping group of 146 families participated in adolescence. At 23 years of age 109 adult adoptees agreed to participate in the study again. Lack of time, death of the adoptive mother, time constraints, lack of interest, and health problems in the family were the main reasons for attrition in the different stages of the study (for details see Jaffari-Bimmel, Juffer, Van IJzendoorn, Bakermans-Kranenburg, & Mooijaart, 2006; Schoenmaker et al., 2013; Stams et al., 2002). Of the 107 participants at 23 years of age, 86 adoptees participated in the collection of saliva. Bonferroni-corrected tests confirmed the absence of selective attrition in the earlier stages of the study (Jaffari-Bimmel et al., 2006; Stams et al., 2002). In the current study, we confirmed the absence of selective attrition with respect to gender, social economic status, experimental condition, maternal sensitivity, and attachment security and organization for the group of 21 participants who did not collect saliva at age 23.

2.4 Measures
2.4.1 Attachment security and disorganization at 12 months. When the children were 12 months of age their attachment security and disorganization were assessed with the Strange Situation Procedure (SSP; Ainsworth et al., 1978). Interrater reliability for the main attachment classifications (Cohen’s kappa) ranged from .80 to 1.0 (n = 155; see Stams et al., 2002). Of all the participants of the current study 76% were classified as secure (n = 65), 23% as avoidant (n = 20), and 1% as resistant (n = 1). Eleven participants were disorganized (13%) and 75 organized (87%). In order to improve the power of our study, we used continuous scores for both security of attachment (see Stams et al., 2002) and disorganized attachment. The scores for attachment security were derived from the sub-classifications from the SSP (Main, Kaplan, and Cassidy, 1985;
Van IJzendoorn, Sagi, and Lambermon, 1992). The most insecure infants (A1 and C2) were assigned the score of 1. The A2 and C1 infants were assigned the score of 2, the B4 infants the score of 3, and the B1 and B2 infants scored a 4. The most secure infants, classified as B3, were assigned the score of 5. Intercoder reliability was satisfactory; intraclass correlations ranged from .81 to .95, using four pairs of raters (see Stams et al., 2002). The scores for attachment disorganization were based on the nine-point-rating scale derived from Main and Solomon (1990) with higher scores pointing to more disorganization. In order to reduce the skewness of the distribution of disorganization we used a root transformation of the scores.

2.4.2 Maternal sensitivity at 12, 18, and 30 months. Maternal sensitivity was based on measures at 12, 18, and 30 months. At all three occasions, mother’s sensitive behavior was assessed during structured tasks (building a tower or solving puzzles) in the laboratory. The Egeland/Erickson scales (Egeland, Erickson, Clemenhagen-Moon, Hiester, & Korfmacher, 1990; Erickson, Sroufe, & Egeland, 1985) were used to rate emotional support, structure and limit setting, respect for autonomy, hostility, and quality of instruction. In addition, cooperation and sensitivity (Ainsworth, Bell, & Stayton, 1974) were coded in the child’s home at 12 months, and in the laboratory at 30 months. On each of the three time points, principal component analyses revealed a one-dimensional solution in which all sensitivity measures were included (explained variance 44%, 59%, and 49%, respectively; see Stams et al., 2002). For the final aggregated score, the three standardized scores for maternal sensitivity were combined into one overall score for infancy with an explained variance of 58% (see Stams et al., 2002).

2.4.3 Daily curve cortisol at age 23. When the adopted adults reached the age of 23 we assessed their daily salivary cortisol curves on two separate days (Kirschbaum & Hellhammer, 1994). The young adults were asked to take a saliva sample at 6 points during the day: immediately after waking up, half an hour after waking up, at noon, at 3 p.m., 5:30 p.m., and in the evening just before going to sleep. The saliva was collected by keeping a cotton ball in the mouth for one minute. We asked the young adults not to eat anything for half an hour prior to the assessment and to rinse their mouth 10 min before assessment. Also, the subjects were asked to choose two regular workdays or schooldays on which no special stress-inducing events, such as an exam or an important interview, occurred. The time at which the assessments were done were reported by the young adults themselves, and also through means of the Medication Event Monitoring System (MEMS). The MEMS is a cap that can be screwed on a bottle with the cotton balls in it. Each time the bottle is opened the exact date and time are registered. The MEMS report made it possible to detect non-compliance that might affect the reliability of the cortisol assessments (Kudielka, Broderick, & Kirschbaum, 2003).
The salivary cortisol concentration (nmol/l) was determined using a time-resolved fluorescence immuno-assay. The intra-assay coefficient of variation was between 4.0% and 6.7%, and the corresponding inter-assay coefficients of variation were between 7.1% and 9.0% (Dressendörfer, Kirschbaum, Rohde, Stahl, Strasburger, 1992). In order to reduce the skewness of the cortisol distributions, we used the natural log-transformation of the cortisol values (after adding a constant of 1 to avoid having negative values). Correlations between transformed cortisol values of the first and second day varied between .20 and .50 at the different points in time. Cortisol values that exceeded 3 standard deviations from the mean at a particular point in time were winsorized (Tabachnick & Fidell, 2001). The correlations between the different time points according to self-report and according to the report of the MEMS on the first day ranged from .66 to .80. On the second day correlations ranged from .49 to .92. There were mean level differences with the MEMs time being later than the time according to self report on three time points at both days with a maximum difference of 24 min. When registration by the Medication Event Monitoring System (MEMS) was available, we used this as time of measurement, as we expected it to be more reliable than the self-reported time. We only used self-report when the MEMS time was missing. This was the case for 15% of the cortisol measurements. 86 participants collected cortisol samples at Day 1 one and 84 participants had cortisol samples at Day 2. Ten participants were excluded from the analyses for Day 1 and seven for Day 2 because of unreliable measurements due to: a) non-compliant time reports b) incompatible reports of time between MEMS and self. The mean transformed cortisol values for the final groups were 1.59 ($SD = 0.70$; Day 1) and 1.58 ($SD = 0.72$; Day 2). In Table 1 the descriptives of the predictor variables for these groups are given.

2.4.4. Weight for age at birth. To obtain an indication of infants’ weight for age, adoption records were searched for the earliest available information about the child’s weight. In order to get comparable scores, z-scores were calculated for these weights with the help of the program WHO Antro 2005 (WHO Anthro, 2005) which relies on weight-for-age calculations on a norm group ($N = 8440$) from diverse cultural backgrounds. Gender and preterm birth were taken into account when comparing the scores of the adoptees to the norm (Schoenmaker et al., 2013). For children who were born prematurely, the number of weeks of prematurity was subtracted from their chronological age. The continuous z-scores were included in the analyses. To reduce skewness of the distribution, outliers were winsorized with preservation of order (Tabachnick & Fidell, 2001).
Table 1. Descriptives of the predictor variables for the final sample of Day 1 (n = 76) and Day 2 (n = 77)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Day 1</th>
<th></th>
<th>Day 2</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean</td>
<td>sd</td>
<td>min</td>
<td>max</td>
</tr>
<tr>
<td>Security continuous(^a)</td>
<td>3.36</td>
<td>1.32</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Disorganized continuous(^ab)</td>
<td>2.56</td>
<td>1.67</td>
<td>1</td>
<td>7.5</td>
</tr>
<tr>
<td>Maternal sensitivity(^a)</td>
<td>0.05</td>
<td>0.71</td>
<td>-2.04</td>
<td>1.38</td>
</tr>
<tr>
<td>Weight for age(^a)</td>
<td>-1.12</td>
<td>1.12</td>
<td>-3.87</td>
<td>0.63</td>
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<tr>
<td>Gender female</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Organized attachment</td>
<td>68</td>
<td>89.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Secure attachment</td>
<td>57</td>
<td>75.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Experimental condition</td>
<td>23</td>
<td>30.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(^a) not centered</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(^b) not transformed</td>
<td></td>
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</tr>
</tbody>
</table>

2.5 Analytical Strategy

We analyzed the data of Day 1 with multilevel modeling that can handle repeated measures data with unbalanced data and different time intervals. In stage one we tested multilevel growth models that focused on the effects of maternal sensitivity together with a) attachment security and b) attachment disorganization on the daily curve of cortisol. We used three different level 1 predictors: Time, time squared and a coding that modeled the Cortisol Awakening Response (CAR). For our first time predictor of Day 1 we used the mean time intervals that were present in the final sample: 0, 0.48, 3.64, 6.81, 9.31, and 14.02 h. For the second time predictor we squared the first time predictor. The third time predictor distinguished the second measurement from the other ones in order to capture the CAR, and was coded as 0,1,0,0,0,0. The main level 2 predictors were attachment security and maternal sensitivity in the first model, and attachment disorganization and maternal sensitivity in the second model. We examined whether the level 2 predictors could explain variation in a) the initial cortisol level at time of awakening, and b) the slope of the cortisol daily curve, and c) the awakening response. All level 2 predictors were centered around the mean. The predictors did not correlate significantly with each other. To control for possible covariates we repeated the analyses including gender, weight for age, first time point (time of awakening) and experimental condition, indicating if the parents of the adoptees received the intervention in infancy. All models were estimated with Full Maximum Likelihood which made it possible to compare nested models by inspecting the deviances (-2 log-likelihood). To compare nested models we used the \(\chi^2\) difference test, where
p-values larger than .05 indicate that there is no significant difference between two models (Tabachnick & Fidell, 2001). As common in the multilevel context we report unstandardized weights β.

In the next stage, we validated the findings from Day 1 by reformulating the growth model as a structural equation model (SEM), using EQS 6.2 (Bentler, 1995), and estimating this model on data from Day 2 (Hox & Stoel, 2005). This reformulation allowed us to take a confirmatory approach. We used several indices to test model fit. NNFI and CFI values that exceed .95 and RMSEA values lower than .05 indicate good model fit (Byrne, 2006; Tabachnick & Fidell, 2001). In order to test the plausibility of absences of relations between variables we compared models with and without predictive paths through means of the χ² difference test.

In order to examine the robustness of our results, analyses of both days were rerun excluding another group of participants who showed irregularities between MEMS and self-report or showed very irregular cortisol curves (Day 1, n = 6; Day 2, n = 5). We also reran our analyses excluding one pregnant participant because pregnancy may influence the diurnal cortisol curve (Kirschbaum & Hellhammer, 1994). If results yielded differences in significance of predictors, these differences are reported. Six participants indicated that they used medication on one or two days. Preliminary analyses showed that there were no differences in cortisol production between the participants using and not using medication. Therefore, these participants were not excluded from the analyses.

2.6 Missing Data
In total, there were 26 (3%) missing cortisol values across 15 participants. Also, 16 participants measured their awakening response more than an hour after waking up. Their cortisol values on the second measurement point were handled as missing data. The data from Day 1 were analyzed with a multilevel approach (see previous section) that is particularly efficient for handling missing data on level 1 (i.e., missing cortisol data on one or more measurement occasions). The data of Day 2 were analyzed using a SEM approach that is not specifically designed to handle unbalanced data. Therefore we decided to impute missing data on the third to sixth measurement point (16 data points) with a curve fitting procedure in SPSS 19. Missing data that concerned the first or second measurement (and therefore had an effect on the CAR) were imputed with the individual CAR of the other day and if not available, the mean CAR of the group on the same day. Of all level 2 predictors in our model, only one had missing values: two participants did not have weight-for-age data. These missing values were imputed with the grand mean.
3. Results

3.1. Day 1
The observed cortisol curve displayed the expected decline across the day and the expected cortisol awakening response. In the first step we specified an unconditional growth model (see Table 2), entering time, time squared and the CAR coding. The time variable predicted the cortisol levels in the expected way. The estimate for the linear slope showed that there was a decreasing pattern of cortisol values over the day, $\beta = -.11, SE = .02, p < .001$. Also, the CAR coding revealed that (on top of this decreasing pattern) on average the cortisol values increased from awakening up to half an hour after awakening ($\beta = .27, SE = .06, p < .001$). The time squared variable did not predict the cortisol curve significantly, but was maintained because of its theoretical function in the model. Figure 1 shows the observed and predicted daily cortisol curve for the final group. The conditional intraclass correlation (ICC) indicated that 19% of the variation in cortisol values stemmed from inter-individual differences, after accounting for the time effect (see also Hruschka, Kohrt, & Worthman, 2005). Deviance statistics revealed that including random slopes of time as well as time-squared improved the model significantly, $\chi^2$ dif (5) = 15.43, $p = .009$. Therefore these slopes were specified as random effects in the models including level 2 predictors.

3.1.1 Attachment security. In the next models we investigated the effects of attachment security and maternal sensitivity on the diurnal cortisol curve. In models 1 to 5 (Table 2) we included the main effects, and the interactions of attachment security and maternal sensitivity with time, time squared, and the CAR coding. Models 1, 2, and 3 did not reveal significant main effects of attachment security or sensitivity, nor interaction effects with time, or time squared. Model 4 revealed a significant cross-level interaction between the CAR and attachment security ($\beta = -.099, SE = .04, p = .022$) with more secure children showing a less steep incline in cortisol levels between awakening and half an hour after awakening in young adulthood. Figure 2 shows the cortisol curves based on a median split on attachment security. The most parsimonious model (5) that only retained the significant predictors improved the unconditional growth model significantly, $\chi^2$ dif (5) = 15.43, $p = .009$. Therefore these slopes were specified as random effects in the models including level 2 predictors.

3.1.2 Attachment disorganization. In models 6 to 10 (Table 3) we entered main effects of attachment disorganization and maternal sensitivity, and interactions between these variables and time, time squared and the CAR coding. Models 6, 7, and 8 did not
Table 2. Main multilevel models of the cortisol curve on Day 1 predicted by attachment security and maternal sensitivity (n = 76)

<table>
<thead>
<tr>
<th>Model</th>
<th>Unconditional growth model</th>
<th>Model 1 Security and sensitivity</th>
<th>Model 2 Security and sensitivity interaction time</th>
<th>Model 3 Security and sensitivity interaction time squared</th>
<th>Model 4 Security and sensitivity interaction CAR</th>
<th>Model 5 Security interaction CAR</th>
</tr>
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<tbody>
<tr>
<td>Fixed effects</td>
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</tr>
<tr>
<td>Intercept</td>
<td>2.087**</td>
<td>2.087**</td>
<td>2.087**</td>
<td>2.087**</td>
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<td>2.087**</td>
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<tr>
<td>Security</td>
<td>0.033</td>
<td>-0.017</td>
<td>0.004</td>
<td>0.045</td>
<td>0.044</td>
<td></td>
</tr>
<tr>
<td>Sensitivity</td>
<td>-0.005</td>
<td>0.027</td>
<td>0.028</td>
<td>-0.006</td>
<td></td>
<td></td>
</tr>
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<td><strong>Rate of change</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time</td>
<td>-0.108**</td>
<td>-0.108**</td>
<td>-0.108**</td>
<td>-0.108**</td>
<td>-0.108**</td>
<td>-0.108**</td>
</tr>
<tr>
<td>Time squared</td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
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<tr>
<td>CAR</td>
<td>0.272**</td>
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<td>0.271**</td>
<td>0.271**</td>
<td>0.269**</td>
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<td>Security * time</td>
<td>0.007</td>
<td></td>
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</tr>
<tr>
<td>Security * time squared</td>
<td></td>
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</tr>
<tr>
<td>Sensitivity * time squared</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Security * CAR</td>
<td>-0.099*</td>
<td></td>
<td></td>
<td></td>
<td>-0.099*</td>
<td></td>
</tr>
<tr>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Within person, residual</td>
<td>0.158**</td>
<td>0.158**</td>
<td>0.158**</td>
<td>0.158**</td>
<td>0.157**</td>
<td>0.157**</td>
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<td><strong>Level 2</strong></td>
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<tr>
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<td>0.130**</td>
<td>0.122**</td>
<td>0.124**</td>
<td>0.123**</td>
<td>0.123**</td>
</tr>
<tr>
<td>Rate of change time</td>
<td>0.009*</td>
<td>0.009*</td>
<td>0.009*</td>
<td>0.009*</td>
<td>0.009*</td>
<td>0.009*</td>
</tr>
<tr>
<td>Rate of change time squared</td>
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<td>0.000*</td>
<td>0.000*</td>
<td>0.000*</td>
<td>0.000*</td>
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<tr>
<td>Goodness-of-fit</td>
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<tr>
<td>Deviance</td>
<td>587.828</td>
<td>586.096</td>
<td>582.332</td>
<td>583.365</td>
<td>580.913</td>
<td>580.933</td>
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<tr>
<td>$\Delta \chi^2$</td>
<td>1.732 (2)</td>
<td>5.496 (4)</td>
<td>4.463 (4)</td>
<td>6.915 (4)</td>
<td>6.895 (2)*</td>
<td></td>
</tr>
</tbody>
</table>

Note. CAR = Cortisol Awakening Response. Unstandardized coefficients are reported.
* $p < .05$; ** $p < .01$
* compared to unconditional growth model
Table 3. Main multilevel models of the cortisol curve on Day 1 predicted by attachment disorganization and maternal sensitivity (n = 76)

<table>
<thead>
<tr>
<th>Model</th>
<th>Unconditional growth model</th>
<th>Model 6 Disorganization and sensitivity</th>
<th>Model 7 Disorganization and sensitivity Interaction time</th>
<th>Model 8 Disorganization and sensitivity Interaction time squared</th>
<th>Model 9 Disorganization and sensitivity Interaction CAR</th>
<th>Model 10 Disorganization Interaction CAR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fixed effects</strong></td>
<td></td>
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<td>Initial status</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Intercept</td>
<td>2.087**</td>
<td>2.087**</td>
<td>2.087**</td>
<td>2.087**</td>
<td>2.087**</td>
<td>2.087**</td>
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<tr>
<td>Disorganization</td>
<td>0.014</td>
<td>-0.052</td>
<td>-0.038</td>
<td>-0.014</td>
<td>-0.013</td>
<td>-0.013</td>
</tr>
<tr>
<td>Sensitivity</td>
<td>0.001</td>
<td>0.019</td>
<td>0.024</td>
<td>0.024</td>
<td>0.003</td>
<td>0.003</td>
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<td>Rate of change</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Time</td>
<td>-0.108**</td>
<td>-0.108**</td>
<td>-0.108**</td>
<td>-0.108**</td>
<td>-0.108**</td>
<td>-0.108**</td>
</tr>
<tr>
<td>Time squared</td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
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<tr>
<td>CAR</td>
<td>0.272**</td>
<td>0.272**</td>
<td>0.272**</td>
<td>0.272**</td>
<td>0.272**</td>
<td>0.272**</td>
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<tr>
<td>Disorganization*time</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Sensitivity*time</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Disorganization*time squared</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sensitivity*time squared</td>
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<tr>
<td>Disorganization*CAR</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sensitivity*CAR</td>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td><strong>Variance components</strong></td>
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<tr>
<td>Level 1</td>
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<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Within person, residual</td>
<td>0.158**</td>
<td>0.158**</td>
<td>0.158**</td>
<td>0.158**</td>
<td>0.154**</td>
<td>0.154**</td>
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<tr>
<td>Level 2</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Initial status</td>
<td>0.124**</td>
<td>0.124**</td>
<td>0.124**</td>
<td>0.124**</td>
<td>0.127**</td>
<td>0.127**</td>
</tr>
<tr>
<td>Rate of change time</td>
<td>0.009*</td>
<td>0.009*</td>
<td>0.009*</td>
<td>0.009*</td>
<td>0.009*</td>
<td>0.010*</td>
</tr>
<tr>
<td>Rate of change time squared</td>
<td></td>
<td>0.000*</td>
<td>0.000*</td>
<td>0.000*</td>
<td>0.000*</td>
<td>0.000*</td>
</tr>
<tr>
<td><strong>Goodness-of-fit</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deviance</td>
<td>587.828</td>
<td>587.782</td>
<td>586.613</td>
<td>586.132</td>
<td>583.867</td>
<td>583.959</td>
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<td>13</td>
<td>15</td>
<td>15</td>
<td>15</td>
<td>13</td>
</tr>
<tr>
<td>Δ χ²</td>
<td>.05 (2)</td>
<td>1.22 (4)</td>
<td>1.70 (4)</td>
<td>3.96 (4)</td>
<td>3.87 (2)</td>
<td></td>
</tr>
</tbody>
</table>

Note. CAR = Cortisol Awakening Response. Unstandardized coefficients are reported.
* p < .05; ** p < .01
*compared to unconditional growth model
reveal any significant main effects or interaction effects. Model 9 revealed a significant interaction between the CAR coding and (the transformed values of) attachment disorganization ($\beta = .24, SE = .12, p = .047$) with more disorganized children showing a steeper incline in cortisol levels between awakening and 0.5 h after awakening in young adulthood. However, this interaction proved unstable, as the most parsimonious model (10) that only retained significant predictors did not improve the unconditional growth model significantly, $\chi^2_{\text{dif}} (2) = 3.87, p = .144$. Also, removal of the suspect cortisol cases led to non-significance. In order to inspect this difference more carefully without relying too much on the p-value in a smaller sample we compared the standardized effects. The standardized weight was .04 in the total group and .03 in the smaller group. To control for possible covariates we reran our analyses with inclusion of gender, time of first assessment, experimental condition and weight for age. Results were similar. No covariate contributed significantly to the model.

3.2 Day 2

3.2.1 Attachment security. In the first latent growth model (Table 4, LGM 1) we tested the cortisol curve predicted by time, time squared and the CAR (see Figure 1 for observed and predicted values). In order to validate the results of Day 1, we explicitly tested the plausibility of the absence of relations by leaving out the predictive paths from attachment security, sensitivity, and gender to the cortisol curve. Fit indices (Table 4, LGM 1) indicated that the first model did not show good fit, $\chi^2 (df = 32, n = 77) = 39.05, p = .182, \chi^2/df = 1.22$, NNFI = .86, CFI = .89, RMSEA = .05, and CI (RMSEA) = .00 – .11. All time variables predicted the cortisol curve significantly. In the second step we added a predictive path from gender to the model, as it was a significant covariate on Day 1, and this improved model fit significantly, $\chi^2_{\text{dif}} (1) = 7.29, p = .006$. Fit indices (Table 4, LGM 2) indicated good model fit, $\chi^2 (df = 31, n = 77) = 31.76, p = .428, \chi^2/df = 1.02$, NNFI = 1.00, CFI = 1.00, RMSEA = .00, and CI (RMSEA) = .00 – .09 (see Table 4). In the next three models (see Table 4, LGMs 3, 4, and 5) we added main effects of attachment security and maternal sensitivity and the interactions of attachment security and maternal sensitivity with time, time squared and the CAR. No significant effects were found, and none of the proposed models improved model fit significantly compared to the model that only included gender (see Table 4; Figure 2 shows the cortisol curves based on a median split on attachment security).

3.2.2 Attachment disorganization. In the first step (LGM 6) we tested the cortisol curve predicted by time, time squared and the CAR. Predictive paths from attachment disorganization, sensitivity, and gender to the cortisol curve were left out. All time-related variables predicted the cortisol curve significantly but the model did not show good fit (see Table 4), $\chi^2 (df = 32, n = 77) = 38.59, p = .196, \chi^2/df = 1.21$, NNFI = .87, CFI = .89, RMSEA = .05, and CI (RMSEA) = .00 – .10. In the second step (LGM 7) we
added a predictive path from gender to the initial cortisol level to the model and this improved model fit significantly $\chi^2_{\text{dif}}(1) = 7.29, p = .007$. Fit indices (Table 4) indicated good model fit, $\chi^2 (df = 31, n = 77) = 31.30, p = .451, \chi^2/df = 1.01$, NNFI = 1.00, CFI = 1.00, RMSEA = .00, and CI (RMSEA) = .00 – .09 (see Table 4). In the next three LGMs (LGMs 8, 9, and 10) we added main effects of attachment disorganization and maternal sensitivity and the interactions of attachment disorganization and maternal sensitivity with time, time squared and the CAR. No significant effects were found, and none of these models improved model fit significantly (see Table 4).

### Table 4. Fit indices for latent growth models Day 2: Security and disorganization as predictors of cortisol

<table>
<thead>
<tr>
<th>Latent Growth Model</th>
<th>$df$</th>
<th>$\chi^2$</th>
<th>$\chi^2/df$</th>
<th>NNFI</th>
<th>CFI</th>
<th>RMSEA</th>
<th>RMSEA 90% CI</th>
<th>$\Delta \chi^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Security</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Independence model</td>
<td>36</td>
<td>90.19</td>
<td>2.51</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 Model without predictions $^a$</td>
<td>32</td>
<td>39.05</td>
<td>1.22</td>
<td>.86</td>
<td>.89</td>
<td>.05</td>
<td>.00–.11</td>
<td>51.14 (4), $p &lt; .01^b$</td>
</tr>
<tr>
<td>2 Model including gender</td>
<td>31</td>
<td>31.76</td>
<td>1.02</td>
<td>1.00</td>
<td>.00</td>
<td>.00–.09</td>
<td></td>
<td>7.29 (1), $p &lt; .01^b$</td>
</tr>
<tr>
<td>3 ABC and sensitivity * time</td>
<td>27</td>
<td>30.32</td>
<td>1.12</td>
<td>.94</td>
<td>.96</td>
<td>.04</td>
<td>.00–.10</td>
<td>1.44 (4), $p = .84^c$</td>
</tr>
<tr>
<td>4 ABC and sensitivity * time squared</td>
<td>27</td>
<td>28.78</td>
<td>1.07</td>
<td>.98</td>
<td>.98</td>
<td>.02</td>
<td>.00–.10</td>
<td>2.98 (4), $p = .56^c$</td>
</tr>
<tr>
<td>5 ABC and sensitivity * CAR</td>
<td>27</td>
<td>29.66</td>
<td>1.10</td>
<td>.95</td>
<td>.97</td>
<td>.03</td>
<td>.00–.10</td>
<td>2.10 (4), $p = .72^c$</td>
</tr>
<tr>
<td><strong>Disorganization</strong></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Independence model</td>
<td>36</td>
<td>89.73</td>
<td>2.45</td>
<td></td>
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<td></td>
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<tr>
<td>6 Model without predictions $^a$</td>
<td>32</td>
<td>38.59</td>
<td>1.21</td>
<td>.87</td>
<td>.89</td>
<td>.05</td>
<td>.00–.10</td>
<td>51.14 (4), $p &lt; .01^b$</td>
</tr>
<tr>
<td>7 Model including gender</td>
<td>31</td>
<td>31.30</td>
<td>1.01</td>
<td>1.00</td>
<td>.00</td>
<td>.00–.09</td>
<td></td>
<td>7.29 (1), $p &lt; .01^b$</td>
</tr>
<tr>
<td>8 DIS and sensitivity * time</td>
<td>27</td>
<td>29.46</td>
<td>1.09</td>
<td>.96</td>
<td>.97</td>
<td>.03</td>
<td>.00–.10</td>
<td>1.84 (4), $p = .77^d$</td>
</tr>
<tr>
<td>9 DIS and sensitivity * time squared</td>
<td>27</td>
<td>29.95</td>
<td>1.11</td>
<td>.95</td>
<td>.96</td>
<td>.03</td>
<td>.00–.10</td>
<td>1.35 (4), $p = .85^d$</td>
</tr>
<tr>
<td>10 DIS and sensitivity * CAR</td>
<td>27</td>
<td>29.75</td>
<td>1.10</td>
<td>.95</td>
<td>.97</td>
<td>.03</td>
<td>.00–.10</td>
<td>1.55 (4), $p = .82^d$</td>
</tr>
</tbody>
</table>

**Note.** NNFI = non-normed fit index; CFI = comparative fit index; RMSEA = root mean square error of approximation; ABC = security; DIS = disorganization; CAR = Cortisol Awakening Response

$a$ Predictors are modeled as unrelated variables in this model; $^b$ Compared to previous model; $^c$ Compared to model 2; $^d$ Compared to model 7
Figure 1. Observed and predicted diurnal cortisol curve (transformed values) for Day 1 ($n = 64-75$) and Day 2 ($n = 77$)

Figure 2. Observed transformed cortisol values of Day 1 and Day 2 for a median split on Security
4. Discussion

In this longitudinal study we investigated the interplay between adopted children’s attachment security and disorganization, sensitivity of the adoptive mother, and the diurnal cortisol curves of the adoptees at age 23 years. With our growth models we were able to describe the observed cortisol curves quite well. We found no consistent evidence for effects of infant attachment and maternal sensitivity in early childhood on the diurnal cortisol curve or the Cortisol Awakening Response (CAR) of adoptees some twenty years later. Day 1 revealed a significant two-level interaction effect with more secure children showing less increase in the CAR, but this result was not replicated on Day 2.

Although several studies have demonstrated that early stressors can lead to changes in the functioning of the HPA-axis such as a down regulation over time (Gunnar & Vazquez, 2001; Miller et al., 2007), we did not find evidence for effects of early attachment experiences on later HPA-axis functioning in this adoption sample. These findings may be of importance, particularly because to our knowledge, the longitudinal associations between early observed attachment experiences and later HPA-functioning in adoptees have not been studied before.

While early attachment quality and sensitive parenting behavior may affect functioning of the HPA-axis in early life (through stress-buffering), it is possible they do not have a direct effect on the diurnal cortisol curve in later life. This apparent absence of the link between early experiences of stress and HPA-axis functioning may be explained by several stressor and person characteristics (Miller et al., 2007). First, in our study, the time elapsed since the onset of the stressor is more than 20 years. It may be that effects simply do not endure across such a long time-frame. In addition, we may wonder about the strength of the stressor. Examples of severe and chronic stressors that affect HPA-axis functioning in later life mentioned in the literature are: the death of important people (Meinlschmidt & Heim, 2005; Nicolson, 2004), divorce of parents (Meinlschmidt & Heim, 2005), and child abuse (e.g., Tricket, Nol, Susman, Shenk, & Putnam, 2010). It is not clear whether insecure and disorganized attachment are actually comparable to these quite severe examples. It may be possible to partly overcome early negative attachment experiences.

Contrary to our findings, Roisman and colleagues (2009) found that maternal insensitivity in childhood predicted lower awakening levels of cortisol in adolescence, albeit with a small effect size. The difference in results may be explained by the fact that in the Roisman et al. (2009) study genetically-related parent-child dyads were investigated. Although until now no evidence has been found for a genetic base for attachment security and attachment disorganization (e.g., Bokhorst et al., 2003; Luijk et al., 2011), we should keep in mind that genetic resemblance between parents and children may explain variation in quality of the attachment relationship and maternal
sensitivity, as well as in the diurnal cortisol curve. It may also be plausible that, in the general population, attachment security and parental sensitivity affect the diurnal cortisol curves in later life, but that the specific adoption experiences of our sample dampen these effects. Although all of our adoptees were adopted at a very young age, it still is possible that prenatal and perinatal problems set the stage for HPA-axis functioning (Gunnar & Fisher, 2006). This is consistent with evidence that the prenatal and perinatal periods are of importance for child development (e.g., Laurent, Ablow, & Measelle, 2011; Talge, Neil, & Glover, 2007). In our sample, the birthmothers of the adoptees may have been at a relatively high risk for depression, stress, malnutrition and other (maternity) problems. This assumption is supported by the fact that at birth, the adopted children were less healthy than normative new-borns in terms of weight for age. Of course, making definite statements regarding this issue without having studied a control group of non-adopted adults is not possible.

Another line of thinking may point to more positive specific adoption experiences. Miller and colleagues (2007) found that the controllability of stressors may have an effect on the associations between these stressors and the HPA-axis functioning. Although certain experiences specific to adoptees have been shown to be developmental risk factors, the majority of adoptees do not show behavior problems (Juffer & Van IJzendoorn, 2005) and their level of self-esteem is comparable to that of non-adoptees (Juffer & Van IJzendoorn, 2007). Relatively many adoptees are referred to special services and receive support that may be helpful in coming to terms with their adoptive status (Juffer & Van IJzendoorn, 2005; Van IJzendoorn, Juffer, & Klein Poelhuis, 2005). A positive appraisal of the adoptive status (see Storsbergen, Juffer, Van Son, & ’t Hart, 2010) and the ability to overcome negative early experiences may counterbalance enduring negative effects on HPA-axis functioning. Gunnar, Frenn, Wewerka, & Ryzin (2009) even explore the possibility that experiencing some degree of early adversity may make children more resilient against later stress exposure. Future longitudinal studies that include both adoptees and non-adoptees and precise measures of early deprivation could further test these hypotheses.

Apart from the lack of a control group, this study has some other limitations. First, some of the variables studied showed limited range. Many adoptees in our sample were securely attached to their adoptive mother and all were adopted at a very young age. This homogeneity of the sample may have been a disadvantage in terms of detecting relations between attachment or early deprivation and the cortisol curve. On the other hand, homogeneity in early adversity made it possible to detect effects of early attachment experiences of the adoptees per se without the confounding with long periods of deprivation. Also, studying a small window of child adversity makes it possible to add to the body of evidence that can detect possible sensitive or critical periods for HPA-axis development (Hostinar & Gunnar, 2013). Second, cortisol measures were only done on two days. Measurements across more days would have
improved the reliability of our findings. In order to improve the reliability of our cortisol measures we used the Medication Event Monitoring System (MEMS) which made it possible to monitor the ability or willingness of participants to follow the instructions. Results indicated that it is difficult for participants to measure their cortisol at the exact requested times and also that self-reports of time may not always be accurate. This may especially be the case for young adults because in general they have not reached optimal stability and structure in their lives yet. Finally, we did not include sensitivity and attachment measurements beyond childhood. We specifically focused on the associations between early attachment experiences and the diurnal cortisol curve in young adulthood.

In conclusion, although positive parenting and attachment experiences in the early lives of adoptees have shown to predict beneficial outcomes in several developmental areas, early maternal sensitivity and attachment security and disorganization do not seem to be associated with the diurnal cortisol curve and Cortisol Awakening Response of adopted young adults. Despite the lack of associations between early experiences and adult cortisol secretion, it is remarkable how the average cortisol curve of adult adoptees appears to show the typical pattern to be expected of healthy, uncompromised individuals. In this sense we may suggest the current study illustrates the positive effect of adoption as social intervention.

5. Highlights

- Adopted young adults appear to show a normative diurnal cortisol curve
- Early maternal sensitivity is not associated with adoptees’ cortisol production
- Infant attachment quality does not predict adopted adults’ diurnal cortisol curve
References


