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Environmental Influences in Family Similarity in Afternoon Cortisol Levels: A Parent-Offspring Design

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Summary

Modest genetic effects on morning, but not late-day, cortisol levels have been established. Environmental demands may influence basal cortisol levels later in the day. Thus, we anticipated that individuals in the same family would have similar afternoon cortisol levels to the extent that they share aspects of their environment. We examined afternoon basal cortisol levels measured across three consecutive days in mothers and fathers and in multiple offspring in two separate large, longitudinal studies. Study I involved 321 families with singletons while study II involved 233 families with twins. Modest family similarity was apparent for afternoon basal cortisol levels in both studies. Spouses' cortisol levels were also correlated. Data from Study II demonstrated that family resemblance in afternoon cortisol was accounted for by underlying shared environmental factors but not underlying genetic factors. Shared environment accounted for 62% of the variation in twin afternoon basal cortisol levels and 14% of the variation in parent afternoon basal cortisol levels. We used pooled data from the two studies to examine whether parental depression, socioeconomic status (SES), and offspring sex and age impacted cortisol levels. Female offspring had higher cortisol levels than males, and cortisol decreased with age until about nine years of age, after which cortisol increased with age. Family similarity persisted after accounting for parental depression, SES, time of day, and offspring sex and age, which suggests that the shared family environment influences parent and offspring stress hormone levels throughout the childhood years.

Keywords

cortisol; context; environment; family; genetics; twins

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1. Introduction

Cortisol is secreted under basal conditions as well as in response to environmental stimuli. Basal activity follows a circadian rhythm with the highest levels typically appearing in the morning and then dropping throughout the day. Afternoon reflects a more quiescent period of the circadian cycle; thus, environmental factors may exert more influence in the afternoon.

Individuals from the same family may have correlated cortisol levels because of shared genetic or environmental influences, which vary throughout the day (Linkowski et al., 1993; Bartels et al., 2003b). Bartels and colleagues (2003a) found significant genetic contribution to cortisol levels after waking in the morning, a moderate genetic contribution around noon, and no genetic contribution during early afternoon. Similarly, other studies have also found genetic influence on cortisol during the awakening period but not on cortisol collected later in the day (Wüst et al., 2000; Kupper et al., 2005). These studies support the hypothesis that environmental factors have more influence on cortisol later in the day. Thus, we expect individuals in the same family, to the extent that they share the same environment, will have similar afternoon cortisol levels. To our knowledge, no previous study has considered cortisol in a parent-offspring design, and the existing evidence from twin studies would also benefit from extension to new samples.

The present paper examined family similarity in afternoon cortisol in two large, longitudinal studies. Study I examined afternoon basal cortisol in families with single-born children. Study II involved families with monozygotic (MZ) and dizygotic (DZ) twins, allowing estimation of genetic and environmental influences on afternoon basal cortisol. Combining Study I and Study II increased the sample size, permitting examination of how parental depression, socioeconomic status (SES), time of saliva collection, and offspring sex and age, variables common to both studies, influenced family similarity in afternoon cortisol. Previous research has demonstrated that SES and parental depression are associated with cortisol (Essex et al., 2002; Lupien et al., 2005).

We predicted that Study I would demonstrate that afternoon basal cortisol is similar among family members. We expected to replicate this finding in Study II, and using twin-twin and twin-parent analyses, to demonstrate that shared environment accounts for most of the variance in afternoon basal cortisol. Finally, in a combined analysis with Study I and Study II, we expected that higher parental depression and lower SES would be associated with higher offspring cortisol (Essex et al., 2002; Lupien et al., 2005); that females would have higher cortisol (Essex et al., 2002); and that children's cortisol would increase with age (Shirtcliff et al., 2005). However, we expected that family similarity would persist after taking these common variables into account.

2. Study I - Wisconsin Study of Family and Work (WSFW)

2.1. Subjects

Study I included 321 families recruited from prenatal clinics with ongoing participation in the WSFW (Essex et al., 2002). The present analyses include 296 mothers, 250 fathers, 292 children (147 female) and 169 siblings (78 female) who provided saliva on at least two of three collection days when the child was age 4.5. Refer to Table 1 for mean ages of all family members. Saliva collection days included a mix of weekdays and weekend days. We excluded 25 families because mothers were pregnant or had given birth within three months of the visit. This study was approved by the University of Wisconsin's Institutional Review Board. All families provided informed consent.

2.2. Saliva collection

Families were instructed to collect saliva (passive drool) on three consecutive days at the same time, prior to dinner, with target collection times ranging between 1500h and 1900h. Families were instructed not to eat or drink for one hour prior to saliva collection and to immediately store samples in their freezer. The research team subsequently collected all samples and stored them at −80°C until assay. Time of collection was correlated among family members (M r=0.74, ps<.001).

2.3. Cortisol Assay

Salivary cortisol was assessed with the Pantex (Santa Monica, CA) RIA modified for saliva. The mean interassay and intraassay variation (CV) was 7.4% and 3.8%, respectively. Cortisol was correlated with time of sample collection (M r=−0.18, ps range from 0.001 to 0.17). To account for variability in time of collection, we regressed each family member's median cortisol level on collection time and computed unstandardized residuals. Analyses used $\log_{10}$-transformed and residualized cortisol values.

2.4. Statistical Analysis

Intraclass correlations indexed similarity in cortisol among family members. Analyses of variance examined how medications affected cortisol. We categorized medications as: (0) none/insignificant, (1) non-narcotic anti-inflammatories, (2) antibiotics/non-steroidal cold, allergy, asthma medications, (3) non-oral steroids, (4) psychotropics, (5) oral contraceptives, and (6) other. Individuals taking oral steroids were excluded.

Medication use in mothers (N=114) affected their cortisol, F(1, 294)=9.25, p=0.002, d=−0.35. Mothers taking oral contraceptives (N=16) had high cortisol, F(1, 294)=14.28, p=0.001, d=−0.49. No other medications influenced cortisol in mothers. Medication use in fathers (N=101) affected their cortisol, F(1, 248)=5.31, p=0.02, d=−0.29, but no single medication drove this finding. Index children on medications (N=93) had higher cortisol than those not taking medications, F(1,290)=7.31, p=0.007, d=−0.33, due to higher cortisol in young children taking antibiotics (N=58), F(1, 290)=9.36, p=0.002, d=−0.45. No other medications were associated with cortisol levels.

2.5. Study I Results

Table 1 presents intraclass correlations among family members for cortisol. In general, correlations were significant and positive. To examine whether medication usage reduced family similarity, stepwise regression analyses first controlled for medications, then included each respective family member's cortisol in the second step. Cortisol was associated with the other family member's cortisol after controlling for medications, ps<0.002.

3. Study II - Wisconsin Twin Project (WTP)

3.1. Subjects

Study II included 233 families participating in the ongoing WTP, a statewide longitudinal study of the development of psychopathology in twins (Van Hulle et al., 2002). Eligible families were identified from state birth records and recruited by mail. Analyses focused on 219 mothers, 184 fathers, 206 twin pairs (88 MZ, 45 female; 59 same sex DZ, 22 female; and 58 opposite sex DZ), and 125 siblings (51 female) who provided cortisol measures on at least two of three collection days when twins were age 8 (see Table 1 for mean ages of family members). Saliva collection days included a mix of weekdays and weekend days. Zygosity was determined using the Zygosity Questionnaire for Young Twins (Goldsmith, 1991), supplemented by in-person and photograph examination and information from birth records. The questionnaire

yields over 95% agreement with zygosity by genotyping (Forget-Dubois et al., 2003). Two pairs (1%) were excluded because zygosity could not be unambiguously determined. This study was approved by the University of Wisconsin's Institutional Review Board. All families provided informed consent.

3.2. Saliva collection

Families were instructed to collect saliva samples with salivettes (Starstedt). All other instructions and procedures were identical to Study I procedures. Time of sample collection was correlated among family members (M r=0.63, p<.001).

3.3. Cortisol Assay

Samples were assayed for cortisol using a salivary enzymeimmunoassay (Salimetrics, PA). This assay was different from Study I due to discontinuation of the Pantex RIA. Two internal controls were included in each assay. For the low control, the average value was 0.082 μg/dl with inter- and intra-assay CVs of 7.2% and 6.1%, respectively. For the high control, the average value was 0.84 μg/dl with inter- and intra-assay CVs of 8.1% and 5.3% respectively. Cortisol levels were correlated with time of sample collection (M r=−0.19, ps range from <0.001 to 0.22). Cortisol was transformed according to the Study I protocol.

3.4. Statistical Analysis

We used intraclass correlations to examine similarity in cortisol among family members. Medication effects were examined as above. Mothers taking oral contraceptives (N=16) had higher cortisol, F(1, 219)=4.03, p=0.03, d=−0.52. No other medications were associated with mothers' cortisol. Fathers taking psychotropic medication (N=7) had higher cortisol, F(1, 185) =6.65, p=0.01, d=−0.98, but no other medications were associated with fathers' cortisol.

3.5 Study 2 Results

3.5.1. Family Cortisol Correlations—The correlations in Table 1 indicate modest positive associations in cortisol among family members; however, not all correlations were significant. In general, sibling correlations (twin-twin and twin-nontwin sibling) were somewhat higher than parent-offspring correlations. MZ cotwins were not more similar than DZ cotwins, a finding that excludes genetic effects. Medication effects were examined using stepwise regression as in Study I. All significant correlations among family members remained significant after controlling for medications, ps<0.05.

3.5.2. Twin–Twin and Twin-Parent Analysis—We fit a series of biometric models to covariance matrices using Mx software to decompose the variance in cortisol into genetic, shared and nonshared environment components (Neale et al, 2002). Total phenotypic variance across all family members was significantly higher among individuals in families with DZ twins than families with MZ twins (ps<0.03). To accommodate this unexpected—perhaps chance—finding (zygosity is not expected to influence individual cortisol variability), we added a non-shared environmental variance component to the model for families of DZ twins, and this component remained in all subsequent models. Next, we fit a standard univariate model (Posthuma & Boomsma, 2005) that only allowed for additive genetic (A), shared or common environmental (C) and nonshared environmental (E) contributions to cortisol. Initial estimation of the ACE model showed that the A (genetic) factor was estimated to be zero and could be dropped from the model, yielding a CE model, without changing the good overall model fit: $\chi^2(2)=2.24$, p=.32. Two additional indices of practical fit suggested a reasonable fit of the CE model (Akaike's information criterion (AIC)= −1.75, Root Mean Square Error of Approximation (RMSEA)=.025).

We then sought to support the twin model with a parent-offspring model using data from mothers, fathers, and twins (Neale & Cardon, 1992). The model assumes that the same genetic, shared and non-shared environmental factors influence cortisol in parents and twins (albeit to varying degrees), and allows for a phenotypic correlation between parents. Parental phenotypes also contribute to the shared environment experienced by twins (i.e., cultural transmission). To maximize power, we fit the models to raw data. The results of the parent-offspring model were similar to the twin model. The genetic parameter (A) was estimated at or near zero, and shared environmental variance (C) accounted for 62% of the phenotypic variation in twin cortisol and 14% of the phenotypic variation in parent cortisol. A series of submodels were then fit to the data (AE, CE, E only, equating parameters across parents, and equating parameters across generations). Both the AE and E only models resulted in a loss in fit ($\Delta\chi^2$ = 16.9, p<.001 and $\Delta\chi^2$ = 77.3, p<.001 respectively). Unstandardized parameter estimates from the full model and best fitting submodel are show in Table 2. There was no differential mother versus father contribution to twins' shared family environment ($\Delta\chi^2$ =.738, p=.99), although parameter estimates varied significantly across generations (($\Delta\chi^2$ =23.7, p=.001). Thus, the CE model showed excellent fit ($\Delta\chi^2$ = 0, p=1.0), supporting the hypothesis that individual differences in afternoon cortisol levels are primarily the product of environmental factors, some of which are shared by family members.

4. Combined Analyses from Study I and Study II

We combined the two samples to increase our sample size to examine whether parental depression, SES, time of collection, offspring sex and age influenced family similarities in cortisol. Principal components analysis was used to compute family SES based on family income, father education, and mother education. The first component accounted for 56% and 63% of the variance in Study I and Study II, respectively. Parental depression was assessed using the Center for Epidemiologic Studies-Depression scale (CES-D; Radloff 1977) and the Beck Depression Inventory (BDI; Beck et al, 1961) in Studies I and II, respectively. Cortisol was z-transformed because studies used different assays. A comparison of 100 samples using both the EIA and RIA assays revealed a very high correlation with a non-parametric Spearman's rho of 0.95 (N=100, $p < 0.001$). Hierarchical Linear Modeling accounted for the nesting of multiple children in families. Level 1 included 997 offspring; Level 2 included 524 families.

Offspring cortisol was not significantly influenced by SES (B=−0.027, p=0.48), mother depression (B==0.042, p=0.34), or father depression (B=0.021, p=0.62). Offspring cortisol was not significantly influenced by mother time of sample collection (B=0.005, p=0.94), father time of sample collection (B=−0.008, p=0.87), or variability in time of collection among family members (B=−0.025, p=0.16). Offspring cortisol was predicted by sex, B=−0.12, p=0.05, such that females had higher cortisol than males. Age of offspring did not affect cortisol, B=−0.02, p=0.25, but there was substantial variability in the effect of age, p=0.002, suggestive of a nonlinear effect. We included age-squared as a fixed effect and found a significant linear, B= −0.16, p=0.001, and quadratic, B=.009, p<0.002, effect of age on cortisol. The quadratic term accounted for heterogeneity in the effect of age on cortisol, SD=.04, p=0.12. Figure 1 shows that cortisol decreases with age in young children, but around age 9, cortisol increases with age. Mothers' and fathers' cortisol continued to predict offspring cortisol, ps<0.001, suggesting that SES, parental depression, time of day, sex and age do not fully account for family similarity in cortisol levels.

5. General Discussion

Research suggests that environmental factors may have a large influence on afternoon cortisol. We found modest associations in afternoon basal cortisol among family members in both Study

I and Study II. Using a twin-parent analysis in Study II, we demonstrated that shared environmental influence accounted for 62% of the variation in twin afternoon basal cortisol and 14% of the variation in parent afternoon basal cortisol. Spousal cortisol was also consistently correlated. Although our data do not speak to the issue, we speculate that the greater influence of shared environment on child cortisol may be due, in part, to parents operating in a wider variety of individual contexts, such as employment situations, whereas young children are primarily influenced by the family environment. Children may also have fewer or different coping skills for dealing with stress.

We then combined the two studies to investigate if parental depression, socioeconomic status (SES), time of collection, and offspring sex and age impacted family similarity in cortisol. Girls had higher cortisol levels than boys. Cortisol decreased with age in young children, until around age 9 when cortisol levels increased (Figure 1). Parental depression and SES were not found to be associated with afternoon basal cortisol. These variables were available in both studies as concurrent measures, and it is possible that only chronic exposure to parental depression beginning in infancy (Essex et al., 2002) or acute stress as measured by a physical or social evaluative threat (Dickerson & Kemeny, 2004) is associated with afternoon cortisol. In our sample, family similarity persisted even after taking these important control and environmental variables into account. Nevertheless, the literature on environmental influence as a function of time of day is quite sparse. Other than the two studies cited above, only a few studies have reported findings related to time of day. One study found that the offspring of parents with bipolar disorder had higher cortisol levels in the afternoon compared with the offspring of parents with no mental disorder, possibly suggesting that the offspring of parents with bipolar parents may have more stressful home environments (Ellenbogen et al., 2004). Another study found that time of day modulates the effects of stress on human declarative memory such that stress during the morning tends to impair declarative memory for emotionally arousing material while stress in the afternoon does not (Maheu et al., 2005).

Results highlight that people are imbedded in social environments, and there may be many factors beyond the individual that can influence stress hormone levels. For instance, parent's role overload stress predicted child cortisol in an earlier analysis (Essex et al., 2002). The importance of understanding the individual within a particular social context is further emphasized by primate research. Animal studies illustrate that cortisol levels change as a function of the individual's ranking as well as the stability of the social hierarchy of the group (Sapolsky, 2005). Our results coupled with the supporting literature suggest that the influence of both genes and environmental factors will differ across the day and future research should consider time of day issues as well as social-contextual factors that might influence an individual's stress hormones.

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References

Bartels M, de Geus EJ, Kirschbaum C, Sluyter F, Boomsma DI. Heritability of daytime cortisol levels in children. Behav Genet 2003a;33:421–433. [PubMed: 14574141]

Bartels M, Van den Berg M, Sluyter F, Boomsma DI, de Geus EJ. Heritability of cortisol levels: review and simultaneous analysis of twin studies. Psychoneuroendocrinology 2003b;28(2):121–137. [PubMed: 12510008]

Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. Arch Gen Psychiatry 1961;4:561–571. [PubMed: 13688369]

Dickerson SS, Kemeny ME. Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. Psychol Bull 2004;130(3):355–391. [PubMed: 15122924]

Ellenbogen MA, Hodgins S, Walker CD. High levels of cortisol among adolescent offspring of parents with bipolar disorder: a pilot study. Psychoneuroendocrinology 2004;29(1):99–106. [PubMed: 14575732]

Essex MJ, Klein MH, Cho E, Kalin NH. Maternal stress beginning in infancy may sensitize children to later stress exposure: effects on cortisol and behavior. Biol Psychiatry 2002;52(8):776–784. [PubMed: 12372649]

Forget-Dubois N, Perusse D, Turecki G, Girard A, Billette JM, Rouleau G, Boivin M, Malo J, Tremblay RE. Diagnosing zygosity in infant twins: physical similarity, genotyping, and chorionicity. Twin Res 2003;6:479–485. [PubMed: 14965457]

Goldsmith HH. A zygosity questionnaire for young twins: A research note. Behav Genet 1991;21:257–269. [PubMed: 1863259]

Linkowski P, Van Onderbergen A, Kerkhofs M, Bosson D, Mendlewicz J, Van Cauter E. Twin study of the 24-h cortisol profile: evidence for genetic control of the human circadian clock. Am J Physiol 1993;264(2 Pt 1):E173–181. [PubMed: 8447383]

Lupien SJ, Fiocco A, Wan N, Maheu F, Lord C, Schramek T, et al. Stress hormones and human memory function across the lifespan. Psychoneuroendocrinology 2005;30(3):225–242. [PubMed: 15511597]

Mahue FS, Collicutt P, Kornik R, Moszkowski R, Lupien SJ. The prefect time to be stressed: a differential modulation of human memory by stress applied in the morning or in the afternoon. Prog Neuropsychopharmacol Biol Psychiatry 2005;29(8):1281–1288. [PubMed: 16229931]

Neale, MC.; Boker, SM.; Xie, G.; Maes, HH. Mx: Statistical modeling. Vol. 6. Richmond, VA: Virginia Commonwealth University, Department of Psychiatry; 2002.

Neale, MC.; Cardon, LR. Methodology for genetic studies of twins and families. Dordrecht: Kluwer Academic Publishers; 1992.

Posthuma D, Boomsma DI. Mx scripts library: structural equation modeling scripts for twin and family data. Behav Genet 2005;35(4):499–505. [PubMed: 15971030]

Radloff LS. The CES-D scale: A self-report depression scale for research in the general population. Appl Psychol Measurement 1977;1:385–401.

Sapolsky RM. The influence of social hierarchy on primate health. Science 2005;308:648–652. [PubMed: 15860617]

Shirtcliff EA, Granger DA, Booth A, Johnson D. Low salivary cortisol levels and externalizing behavior problems in youth. Dev Psychopathol 2005;17(1):167–184. [PubMed: 15971765]

Van Hulle CA, Lemery KS, Goldsmith HH. Wisconsin Twin Panel. Twin Res 2002;5:1–4. [PubMed: 11893274]

Wüst S, Federenko I, Hellhammer DH, Kirschbaum C. Genetic factors, perceived chronic stress, and the free cortisol response to awakening. Psychoneuroendocrinology 2000;25(7):707–720. [PubMed: 10938450]

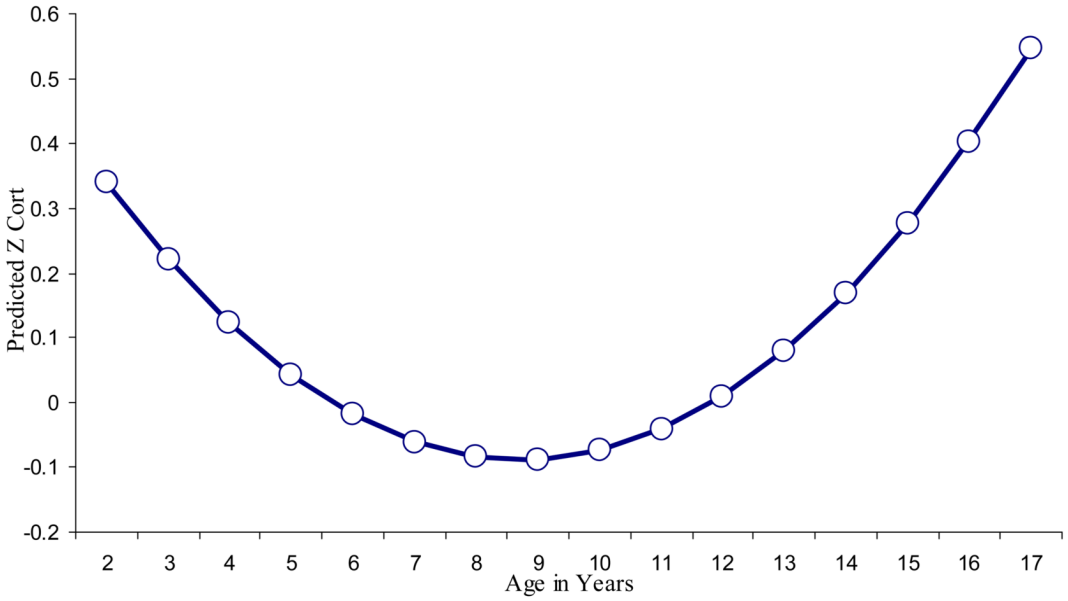

Figure 1.
Effect of Age on Predicted Cortisol in Offspring

Table 1

Mean Age and Cortisol Levels of Individuals and Cortisol Correlations Among Family Members

Wisconsin Study of Family and Work						
		Mom	**Dad**	**Sibling**	**Index Child**	
Mean Cortisol	Day 1	.122 (.086)	.115 (.110)	.106 (.089)	.142 (.185)	
	Day 2	.130 (.097)	.106 (.094)	.115 (.107)	.141 (.158)	
	Day 3	.129 (.108)	.115 (.099)	.117 (.126)	.142 (.148)	
Mean Age		35.0 (3.9)	36.9 (4.1)	6.0 (2.2)	4.6 (.1)	
Age Range		25–49	26–57	1.8–10.4	4.5–5.2	
Correlations		Mom	Dad	Sibling	Index Child	
Mom			.21** N=221	.29** N=154	.26* N=258	
Dad				.30** N=134	.23* N=219	
Sibling					.36* N=157	
Wisconsin Twin Project						
		Mom	**Dad**	**Sibling**	**Twin A**	**Twin B**
Mean Cortisol	Day 1	.102 (.090)	.104 (.077)	.119 (.231)	.123 (.394)	.115 (.352)
	Day 2	.111 (.132)	.108 (.120)	.099 (.078)	.096 (.098)	.091 (.075)
	Day 3	.117 (.136)	.110 (.129)	.090 (.074)	.094 (.121)	.089 (.102)
Mean Age		38.4 (4.47)	40.41 (5.18)	10.82 (2.96)	8.64 (.75)	8.64 (.75)

Wisconsin Study of Family and Work					
	Mom	**Dad**	**Sibling**	**Index Child**	
Age Range	26–50.5	27–58.1	3.33–16.75	5.7–12.1	5.7–12.1
Correlations	Mom	Dad	Sib	Twin A	Twin B
Mom		.19 p = .10 N=74	.17 p = .26 N=48	.32** p<.01 N=84	.23* p = .04 N=84
Dad	.26** p < .01 N=108		.31* p < .05 N=41	.27* p = .02 N=71	.16 p = .19 N=71
Sibling	.10 p = .42 N=74	.02 p = .85 N=65		.41** p < .01 N=44	.42** p < .01 N=46
Twin A	.18* p < .05 N=115	.27** p < .01 N=98	.30 p = .01 N=67		.41** p < .01 N=85
Twin B	.17 p = .07 N=116	.24* p < .02 N=99	.35** p < .01 N=67	.52** p < .01 N=114	

WTP = Wisconsin Twin Project. WSFW = Wisconsin Study of Family and Work. Mean cortisol values are displayed in μg/dl. Mean age is displayed in years. Standard Deviation is shown in ( ). Correlations are presented prior to adjusting for medication usage. In the WTP correlation matrix, the top diagonal presents correlations among monozygotic twins and family members and the bottom diagonal presents correlations among dizygotic twins and family members.

Table 2

Unstandardized Parameter Estimates

	a*	c*	e*
Full Model	−.01 (−.21 .21)	.06 (−.09 .09)	.19 (−.21 .21)
	0.00 (−.11 .11)	−.16 (−.19 −.12)	.13 (.11 .15)
CE model	---	.06 (.03 .09)	.18 (.16 .21)
		−.16 (−.19 −.14)	.13 (.11 .15)

* parents (top line) and offspring (bottom line) Confidence intervals shown in ( ).