Differential Pathways to Preterm Delivery for Sexually Abused and Comparison Women

Jennie G. Noll,[1] PhD, Jay Schulkin,[2] PhD, Penelope K. Trickett,[3] PhD, Elizabeth J. Susman,[4] PhD, Lesley Breech,[1] MD, and Frank W. Putnam,[1] MD

[1]Department of Pediatrics, University of Cincinnati College of Medicine, Cincinnati Children's Hospital Medical Center, 45229-3039, [2]Department of Physiology, Georgetown University, Washington DC, 20057, [3]University of Southern California, School of Social Work, Los Angeles, CA 90089, and [4]Department of Human Development, The Pennsylvania State University, University Park, PA, 16802, USA

Objective Two distinct conditions, Hypothalamic Pituitary Adrenal (HPA) axis disruptions and maternal alcohol use, have been linked to preterm delivery. These conditions have also been cited as potential sequelae of childhood abuse. Studies have linked childhood abuse to increased rates of preterm delivery but mechanisms explaining this association are unclear. **Methods** This prospective study compared preterm birth rates across offspring born to mothers who were sexually abused in childhood (OA; $N = 67$) and offspring born to nonabused comparison mothers (OC; $N = 56$). **Results** Preterm delivery rates were higher for the OA group (Odds $= 2.80 \pm 1.44$, $p < .05$). Maternal prenatal alcohol use mediated this relationship, but HPA axis functioning did not. Heightened maternal cortisol was significantly related to preterm status, but only for the OC group. **Conclusions** Results support the hypothesis that childhood abuse is a risk-factor for preterm delivery, however pathways are likely different for women with and without histories of sexual abuse.

Key words childhood sexual abuse; cortisol; HPA axis; prenatal alcohol use; preterm delivery.

The United States has one of the highest preterm birth prevalence rates for developed nations at just over 12%, with the etiology of 50–60% of cases unknown, and recent prevention programs largely unsuccessful (CDC, 2002). Although the majority of premature newborns survive, numerous short- and long-term developmental outcomes have been associated with preterm delivery and include; cerebral palsy, visual, auditory and intellectual impairments, respiratory, gastrointestinal and renal problems, and other neurological disorders (Slattery & Morrison, 2002). Preterm birth continues to have significant emotional, social, health, and economic impact on infants and families because few risk factors and preventive measures have been identified (Adams, 1995).

There is increasing speculation that high levels of maternal stress can lead to premature delivery. Physiologic response to stress and anxiety may influence gestational age at delivery. For example, the peripheral components of the stress system include the peripheral limbs of the Hypothalamic-Pituitary-Adrenal (HPA) axis, the efferent sympathetic adrenomedullary system, and components of the parasympathetic system (Dole et al., 2003). During elevated stress, various hormones including corticotropin-releasing-hormone (CRH) and glucocorticoids such as cortisol are released into the systemic circulation. Increased maternal cortisol secretion during pregnancy is associated with increased levels of placental CRH which can stimulate fetal hormonal activity in accordance with preparation for parturition (Power & Schulkin, 2006). Abnormally high maternal cortisol secretion may upwardly regulate placental CRH prematurely, thus rendering a woman more vulnerable to preterm labor and delivery (Majzoub et al., 1999) and subsequent fetal morbidity or mortality.

Maternal stress biology is likely determined by a multiplicity of genetic and environmental factors that

All correspondence concerning this article should be addressed to Jennie G. Noll PhD, Cincinnati Children's Hospital Medical Center, Department of Pediatrics, Division of Behavioral Medicine and Clinical Psychology, 3333 Brunet Avenue MLC 3015, Cincinnati, OH 45229-3039, USA. E-mail: jennie.noll@cchmc.org.

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impinge on stress adaptability throughout development. For example, exposure to severe and chronic stress early in life or during periods of high neuronal plasticity can produce lasting alterations of the HPA axis (Meaney et al., 1993). Evidence from rat and nonhuman primate studies demonstrates how maternal separation and/or deprivation can be detrimental to the developing stress physiology (Kuhn, Pauk, & Schanberg, 1990; Levine, 1994; Reite, Kaemingk, & Boccia, 1989), whereas increased maternal attention such as touch and grooming can promote the development of adaptive physiological responses to stress (Caldji et al., 1998; Meaney et al., 1993). In humans severely deprived orphanage-raised children have shown substantial HPA disruption later in development (Carlson & Earls, 1997; Gunnar, Morison, Chisholm, & Schuder, 2001) and adults who retrospectively reported severe early traumatic experiences show altered stress hormone responsivity (Heim et al., 2002). Thus, it is reasonable to hypothesize that pre-pregnancy experiences influence the HPA axis so as to increase the risk for preterm birth.

Few conditions exemplify severe and chronic stress to a greater extent than the experience of childhood abuse. The past 10 years has seen a surge of studies examining the relationship between HPA axis functioning and childhood abuse. Maltreated pre-schoolers (Hart, 1995) and school-aged children (Cicchetti & Rogosch, 2001), as well as adults retrospectively reporting childhood abuse (Heim, Newport, Bonsall, Miller, & Nemeroff, 2001; Heim et al., 2000) have exhibited marked HPA dysregulation as compared to their nonabused counterparts. HPA dysregulation takes several distinct forms with evidence for both cortisol hypersecretion (DeBellis et al., 1994; Heim, Ehlert, & Hellhammer, 2000) and attenuated cortisol secretion or hypocortisolism (Hart, 1995; King, Mandansky, King, Fletcher, & Brewer, 2001). The magnitude differences between cortisol secretions observed in abused versus comparison samples varies by study with reported differences between 20% and 60%. Although the nature, magnitude, and developmental course of HPA axis disruption in childhood abuse victims are somewhat unclear (Susman, 2005), the possibility of an altered HPA activity provides a basis for the hypothesis that females abused in childhood may be at risk for preterm deliveries in accordance with these alterations (Horan, Hill, & Schulkin, 2000). Several retrospective studies have reported that mothers referencing childhood abuse had a greater number of preterm infants than nonabused controls (Stevens-Simon, Kaplan, & McAnarney, 1993; Van Der Leder & Raskin, 1993) even after controlling for potential confounds such as poor prenatal care and substance abuse. However, there are no prospective studies examining rates of preterm delivery for women with childhood abuse experiences. Moreover, no studies to date have tested whether or not there is a direct connection between the biology of stress and preterm births for women who were abused as children.

Alcohol is a potent teratogen and its use during pregnancy has been identified as one of the leading preventable causes of adverse pregnancy outcomes (USDHHS, 2000). Clear associations have been drawn between prenatal alcohol use and perinatal death (Jones, Smith, Streissguth, & Myrianthopoulos, 1974), congenital anomalies (Hanson, Jones, & Smith, 1976), fetal growth (Whitehead & Lipscomb, 2003), birthweight (Mills, Graubard, Harley, Rhoads, & Berendes, 1984), and fetal alcohol syndrome (Ouellette, Rosett, Rosman, & Weiner, 1977). Research specifically focused on the connection between prenatal alcohol use and preterm delivery has yielded conflicting results. Several studies have reported that moderate to high alcohol consumption during the prenatal period is associated with preterm delivery (Albertsen, Andersen, Olsen, & Gronbaek, 2004; Berkowitz, Holford, & Berkowitz, 1982; Kesmodel, Olsen, & Secher, 2000; Lundsberg, Bracken, & Saftlas, 1997), while others have reported relatively small or zero effects (Marbury et al., 1983; Verkerk, van Noord-Zaadstra, Florey, de Jonge, & Verloove-Vanhorick, 1993). The probable confounds of additional high-risk lifestyle factors (such as domestic violence, smoking, poor prenatal care, and dietary deficiencies) have also been cited as possible explanations for this association (Schoeman, Grove, & Odendaal, 2005; Scholl, Miller, Salmon, Cofsky, & Shearer, 1987).

There is considerable evidence for a strong association between early child maltreatment experiences and subsequent alcohol abuses in adulthood (Beitchman et al., 1992; Kilpatrick et al., 2000; Widom, Ireland, & Glynn, 1995; Wyatt & Powell, 1988) even when co-occurring forms of childhood adversity are taken into account (Anda et al., 2002). Inasmuch as prenatal alcohol use constitutes a risk factor for preterm delivery, women with childhood abuse histories may be especially vulnerable due to the high incidence of alcohol usage in this population. Alcohol usage is also associated with additional high-risk pregnancy characteristics (such as unintended and teen pregnancies) which have also been shown to be inordinately high for women with childhood abuse histories (Noll, Trickett, & Putnam, 2003).

It is unclear whether women who have survived childhood abuse are at high risk for preterm delivery

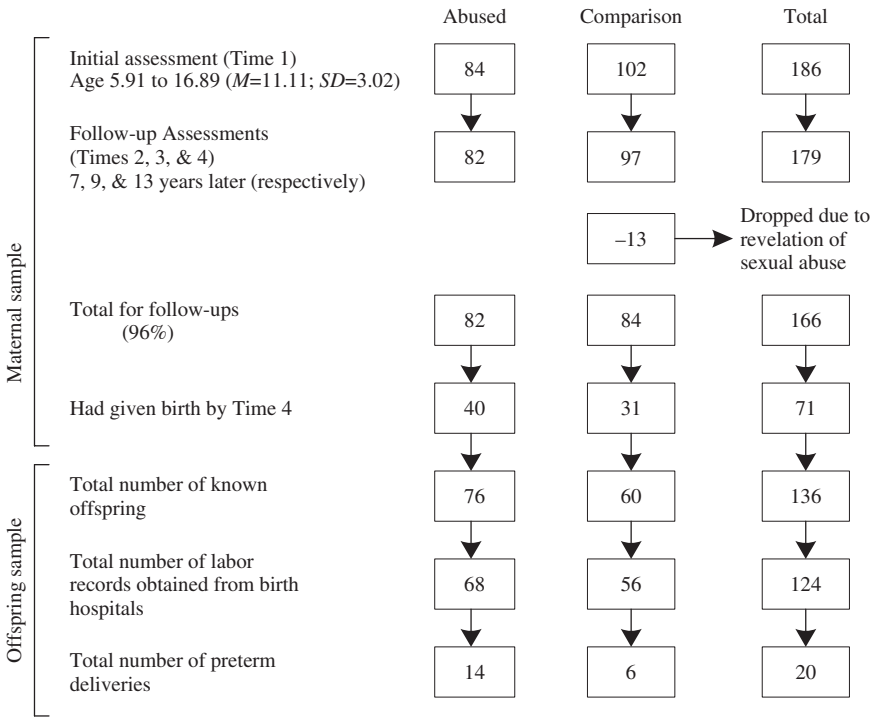


Figure 1. Flow of maternal and offspring samples.

and/or whether maternal HPA axis disruption or prenatal alcohol use in such women may help explain this association. In the present study, salivary cortisol, parity histories, and labor and delivery records were assessed on several occasions throughout late adolescence and early adulthood for females with and without substantiated childhood sexual abuse. Corresponding incidences of preterm delivery, maternal cortisol levels, and maternal prenatal alcohol use were compiled for progeny born to this original sample of women. Hypotheses tested included: (a) the group of offspring born to abused mothers (OA) will have a higher incidence of being born preterm than offspring born to nonabused comparison mothers (OC); and (b) maternal cortisol and maternal prenatal alcohol usage will help explain the association between maternal childhood sexual abuse and subsequent preterm delivery.

Methods

Subjects and Procedures

Sexually abused females ($n = 84$) were referred by child protective service (CPS) agencies in the greater Washington DC, metropolitan area. Eligibility criteria included: (a) age 6–16; (b) participation within 6 months of disclosure; (c) substantiated contact sexual abuse including genital contact and/or penetration; (d) perpetration by a family member (parent, grandparent, older sibling, uncle); and (e) participation of a nonabusing caregiver to be an additional informant and to provide parental permission. CPS records indicated that the median age at onset of abuse was 7.8 years, the median duration was 24 months, 70% experienced vaginal and/or anal penetration, and 60% of perpetrators were the primary father figure which included biological fathers, step fathers, and mother's live-in boyfriends. It is not possible to estimate with precision how similar the sample is to the average caseloads of protective service agencies. However, the information on the perpetrators, the average age of onset, and the average duration is similar to comparable information reported in national surveys of protective services caseloads in years proximal to obtaining the sample via data reported in the National Incidence Study conducted by the National Center on Child Abuse and Neglect (NCCAN, 1988).

Comparison females ($n = 102$) were recruited via community advertisements in neighborhoods where abused families resided, were screened for prior contact with CPS agencies, and were matched to abused females regarding ethnic group, age, pre-disclosure socioeconomic status, family constellation, and residing zip codes.

The design flow is depicted in Fig. 1. In 1987 the study began with recruitment and the initial assessment (Time 1) spanning approximately 18 months. At Time 1

females ranged in age from 5.91 to 16.89 ($M = 11.11$, $SD = 3.02$) and reported low to middle Socioeconomic States (SES) using a previously developed and widely used measure (Hollingshead, 1975). Forty-nine percent were Caucasian, 46% African American, 4% Hispanic, and 1% Asian American. Three follow-up assessments were conducted (Times 2 through 4; approximately 7, 9, and 13 years after Time 1, respectively) with over 96% of the sample reassessed at least once. During the follow-up interviews 13 comparison subjects revealed some form of childhood sexual abuse occurring after Time 1 and were excluded from further analyses. By the conclusion of Time 4, 71 subjects reported having had at least one child. The focus of the current set of analyses is on the offspring of these 71 women (abused = 40; comparison = 31).

All assessments were scheduled to begin at 0830 hr, concluded at 1200 hr, and included a host of bio-psychosocial measurements. Nonabusing caretakers provided consent for those subjects under the age of 18, 18 and over signed for themselves, and those 7–17 also provided assent. Subjects then provided a resting salivary sample approximately 30 min after arrival (between 0900 and 1000 hr) and parity histories. Primiparous and pregnant subjects provided permission for release of labor records. Subjects were awarded monetary compensation at a rate put forth by the National Institutes of Health Normal Volunteer Office. The study received approval from the University Institutional Review Board and a Federal Certificate of Confidentiality.

Offspring Sample

The focus of the current analyses is on the offspring born to 71 abused and comparison women participating in the study. Of the 136 known offspring, *complete* hospital labor and delivery records were obtained for 91%, constituting a sample of 124; 68 offspring of abused mothers (OA), 56 offspring of comparison mothers (OC). At the Time 4 follow-up, offspring ranged in age from 5 months to 10 years (abused $M = 4.60$, $SD = 3.35$, comparison $M = 3.56$, $SD = 2.57$) and were 53.66% minority (mostly African American with 3% Hispanic and 1% Asian). The sample included 65 singlets, 18 sibling pairs, 5 sibling trios, 2 families with four siblings, and 0 multiples. There was no significant difference in sibling number across OA and OC groups.

Measures

Preterm Status

After obtaining hospital labor and delivery records, birth dates, gestational age, and preterm status (PT) were gleaned for all offspring. Two independent raters (blind to group status) coded each medical record, and consultation with the fifth author was obtained until 100% agreement was reached. Preterm delivery was gleaned from hospital records and was defined as gestational age less than 37 weeks from last menstrual period excluding elective inductions. There were 14 infants born preterm in the OA group and six infants born preterm in the OC group. Preterm infants ranged in gestational age from 29.89 to 36.99 weeks with a mean of 33.45 ($SD = 3.44$). For offspring born preterm, there were seven first children, seven second children and six third children, and zero sibling pairs (i.e., there were no preterm offspring nested within a single family). Sibling order was roughly evenly distributed across PT groups.

Maternal Cortisol

Procedures for each maternal cortisol assessment included obtaining stimulant-free salivary basal cortisol samples between 0900 and 1000 hr after a 20 min resting period. All samples were stored at −70°C for 1–6 months and then assayed for salivary cortisol in duplicate using a highly sensitive enzyme immunoassay by Salimetrics Laboratories (State College, PA) with no appreciable sample degradation. The test used 25 µl of saliva per determination, has a lower limit of sensitivity of .003 µg/dl, standard curve range from .007–1.8 µg/dl, and average intra- and inter-assay coefficients of variation 5.10% and 8.20%, respectively. There was no appreciable variation in basal cortisol distributions across time (Time 2 $M = 0.33$, $SD = 0.25$, skewness = 1.24; Time 3 $M = 0.31$, $SD = 0.27$, skewness = 1.371; Time 4 $M = 0.29$, $SD = 0.30$, skewness = 1.41). Maternal cortisol was defined as free basal cortisol assayed at the follow-up time points (Times 2 through 4) when mothers were not pregnant. Pregnancy status was ascertained either by responses to direct queries during assessments or by calculating offspring date-of-birth minus data of assessment being less than 11 months. To increase reliability and control for potential daily and diurnal variation, cortisol levels were (a) obtained at roughly the same time of day across all subjects and across all time points, and (b) averaged over all available nonpregnant samples taken at Time 2, 3, and/or 4. All mothers had at least two nonpregnant cortisol samples available for averaging with roughly 20% having three nonpregnant samples available for averaging. A dichotomous variable defining the number of samples (2 vs. 3) was shown to be unrelated to OA versus OC group status, pregnancy variables, or cortisol levels in

Table I. Characteristics of the Offspring Sample ($N = 124$) by OA vs. OC groups

	OA ($N = 68$) Mean (*SD*)	OC ($N = 56$) Mean (*SD*)
SES	32.11 (15.11)	35.81 (12.21)
Minority status (%)	43.28 (55.03)	66.07 (50.02)*
Maternal age at birth (years)	21.14 (2.54)	21.97 (2.33)
Born preterm (%)	20.58 (40.11)	10.71 (31.63)*,[a]
Maternal basal cortisol (salivary μg/dl)	.25 (.16)	.36 (.23)
Maternal prenatal alcohol use (%)	11.76 (32.11)	2.62 (20.11)*,[a]

Note: *significant group difference at $p < .05$.

[a]Minority status and sibling number covaried.

OC, Offspring born to comparison mothers; OA, Offspring born to abused mothers.

SES via maternal Hollinghead (1975) scores.

bivariate phi coefficients or point biserial correlational analyses. The average span between available samples was 2.34 ($SD = 1.35$) years with no significant difference across OA and OC groups.

Prenatal Alcohol Use

Maternal prenatal alcohol use was gleaned from the prenatal care history sections of labor and delivery records obtained from hospitals (1 = ''yes'' checked next to direct query on medical chart); 0 = ''no'' checked next to direct query on medical chart). Records which did not include indications of direct prenatal alcohol use queries were considered incomplete and were excluded from analyses ($N = 12$; see description of offspring sample above). Level of usage (e.g., moderate vs. heavy) or prenatal timing of usage (e.g., first trimester vs. third trimester) were not consistently reported in all records and, therefore, were not included in the present analyses.

Covariates

Univariate tests were performed to identify potential demographic covariates (Table I). There were no OA versus OC group differences in SES or maternal age at the birth of an index child. There were significantly more minorities in the OC group (66.07% vs. 43.28%; $\chi^2(1) = 6.37$, $p < .01$). Therefore, minority status (1 = minority, 0 = white) was used as a covariate in all subsequent analyses. The sample of offspring contained several siblings, or individuals nested within families, thus necessitating some control for estimation bias due to a lack of independence of observations. Intraclass correlations were performed to ascertain the extent of intrafamilial dependence across; (a) OA versus OC groups, and (b) preterm versus full-term groups. These correlations were very small ($r = .03$, $p = .89$ and $r = .01$, $p = .98$, respectively) suggesting that bias due to intrafamilial dependence would be negligible in analyses. However, because the binary outcome was obtained under the assumption of independence (Hoe, 2005), a weighted continuous variable representing sibling number (0 = singlet; 1 = one sibling; 2 = two siblings; 3 = three siblings) was included as an additional covariate in all analyses to effect control over potential sample dependence and to minimize Type I error and estimation bias.

Results

OA versus OC Group Comparisons

There were 14 (20.58%) children born preterm in the OA group and six (10.71%) born preterm in the OC group (Table I). Because of the relatively low base-rate of preterm delivery in the population, logistic models utilizing PT as the dependent variable would yield an unbalanced design. Therefore, the logistic analysis was performed using LogXact® V6 (Cytel Corp.; Cambridge, MA) to calculate ''exact'' point estimates and control for estimation bias due to unbalanced logistic designs. Results from this logistic regression revealed that maternal childhood abuse status was a significant predictor of preterm delivery status (covariates controlled), with the OA group 2.80 times more likely to be born preterm [point estimate β (df = 1,120) = 1.03; 95% confidence interval ± .37; Odds $e^{\beta} = 2.80 \pm 1.44$, $p < .05$]. Table I also shows that the OA and OC groups did not differ with respect to maternal cortisol levels. The incidence of maternal prenatal alcohol use was significantly higher in the OA group than in the OC group [$F(1,120) = 5.63$, $p < .05$)].

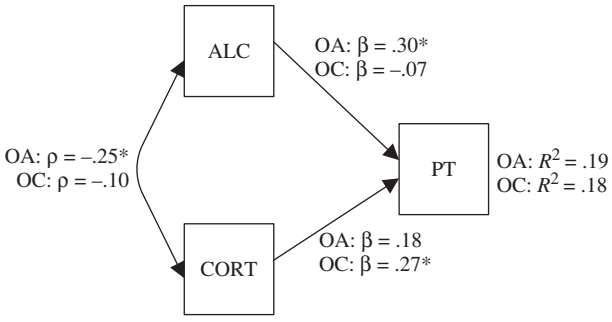
Mediation Analysis

Table II includes a stepwise regression analysis (Baron & Kenny, 1986) of the potential mediating effects of maternal alcohol use (ALC) and maternal cortisol (CORT) in the relationship between OA versus OC group status and PT. Model 1 indicated a significant main effect for group in the prediction of PT. With the addition of ALC in Model 2, the group main effect β changed to a nonsignificant level and the R^2 change from Model 1 was significant—consistent with mediation. In Model 3, the addition of CORT did not result in a significant change to the group main effect β nor did this addition result in a significant R^2 change from Model 1. These results indicated that ALC mediated the relationship between

Table II. Maternal Prenatal Alcohol use (ALC) and Maternal Cortisol (CORT) Mediating the Effect of Maternal Childhood Abuse on Preterm Status (PT) for Offspring Born to Abused Mothers (OA) vs. Offspring Born to Comparison Mothers (OC)

Mode	IVs	Standardized Beta Predicting PT	Model Evaluation:	Mediator?[a]
1	Group	$\beta = .18$ $p = .02$	$R^2 = .07$	–
2	Group	$\beta = .09$ $p = .10$	$R^2\Delta = .03$; $p = .01$	Yes
	ALC	$\beta = .20$ $p = .01$		
3	Group	$\beta = .19$ $p = .02$	$R^2\Delta = .001$; $p = .25$	No
	CORT	$\beta = .10$ $p = .24$		

Note: Results from stepwise regression models shown; minority status and sibling number covaried.
[a]$R^2\Delta$ is significant at $p < .05$, df = (1119).
Group (1 = OA, 0 = OC); PT (1 = preterm, 0 = term); $R^2\Delta = R^2$ change from Model 1.



One Degree-of-freedom Nested Model Comparisons:
Model 1: Free Model: $\chi^2(6) = .59$; RMSEA = .03; GFI = .98, p = .98.
Model 2: equating ALC ⟶ PT across groups: $\chi^2(1) = 5.43$, p = .03.
Model 3: equating CORT ⟶ PT across groups: $\chi^2(1) = .88$, n.s.
Model 4: equating ALC ⟷ CORT: $\chi^2(1) = 1.30$, n.s.

RMSEA, root mean square residual; GFI, goodness of fit index;
$\chi^2(1)\Delta$, chi-square change from Model 1 (i.e., fit degradation).
*P < .05

Figure 2. Multiple group path analysis showing differential pathways to preterm status (PT) for offspring born to abused mothers (OA) and offspring born to comparison mothers (OC). Maternal alcohol use (ALC) is a significant group moderator. Maternal cortisol (CORT) is a significant predictor of PT for the OC group, but is not a *significant* group moderator. One Degree-of-freedom Nested Model Comparisons: Model 1: Free Model: $\chi^2(6) = .59$; RMSEA = .03; GFI = .098, p = 98. Model 2: equating ALC → PT across groups: $\chi^2(1)\Delta = 5.43$, p = .03. Model 3: equating CORT → PT across groups: $\chi^2(1)\Delta = .88$, n.s. Model 4: equating ALC → CORT: $\chi^2(1)\Delta = 1.30$, n.s. RMSEA, root mean square residual; GFI, goodness of fit index; $\chi^2(1)\Delta$, chi-square change from Model 1 (i.e., fit degradation).

OA versus OC group status and preterm delivery, but CORT did not. Thus, ALC was instrumental in helping to explain the higher rate of preterm delivery in the OA group.

Moderator Analysis

As seen in the above analysis, it is possible for some variables to act as mediators in that they account for, or help to explain, group differences. Still other variables may act as moderators, operating differently across groups to explain individual variation in outcomes—for example, variables related to outcome in one group, but not in the other group. It is often advantageous to examine both mediating and moderating relationships, especially when multivariate systems may impinge on outcome. Figure 2 depicts the statistical tests for group moderators via a stepwise multiple group path analysis (Loehlin, 1987) performed using the LISREL (V8.54) software (Joreskog & Sorbom, 1993). In Model 1, all pathways are freely estimated across groups. This free model fits the data well with low residual values, a high goodness of fit, and respectable R^2 values for each group. Model 2 shows the change in Model 1 χ^2 that is due to placing equality constraints on the ALC→PT pathway across groups. Utilizing one degree-of-freedom nested χ^2 difference tests [i.e., $\chi^2(1)\Delta$], results indicated that constraining the ALC→PT β parameters to be equal across the OA and OC groups produced a significant change in free model χ^2 indicative of significant overall fit degradation. Such fit degradation signifies that the magnitude difference in parameter estimates across groups is sufficiently larger than zero and that the best fitting model is one where equality parameter constraints are lifted. Neither equating the CORT→PT β parameters (Model 3) nor equating the ALC↔CORT correlational ρ parameters (Model 4) across groups resulted in significant χ^2 change or fit degradation from Model 1 suggesting that the magnitude difference in these parameters across groups is negligible. Hence, these results indicate that maternal prenatal alcohol use was a significant group moderator. Maternal cortisol did not function as a group moderator, however higher maternal cortisol was a significant predictor of PT in the OC group.

Discussion

This study provides the first evidence from a prospective study of the impact of early childhood abuse that maternal childhood sexual abuse is a significant risk-factor for preterm delivery. The study employed significant methodological improvements over past research

such as childhood sexual abuse being objectively obtained and substantiated, preterm delivery gleaned from medical records, prospective design, high retention rates, and the assessment of plausible mediators. Findings suggest, however, that the mechanisms associated with preterm delivery may be different for women with and without traumatic histories. Although abused females may not exhibit the particular underlying stress biology thought to place ''normally'' developing women at risk for preterm delivery (e.g., high basal cortisol concentrations), the likelihood of having an infant born prematurely remains significant for these women, perhaps due to a number of alternative behavioral traits and environmental conditions that are also associated with surviving severe childhood adversity. Women with abusive or violent pasts may require increased support and some additional monitoring during pregnancy. Hence, results presented underscore how the experience of childhood abuse may impinge on normal development for some females, and that outcomes for victims are likely realized via a variety of developmental pathways (Noll, Trickett, & Putnam, 2000; Noll et al., 2003; Trickett, Noll, Reiffman, & Putnam, 2001). These results also provide increased impetus for examining the various aspects of intergenerational transmission of the effects of childhood abuse.

Our results showed that maternal prenatal alcohol use was particularly important in explaining preterm deliveries for offspring born to abused mothers. These results suggest that child abuse survivors who have histories of alcohol abuse may benefit from education regarding the specific effects of prenatal alcohol use on pregnancy outcome. The pathophysiologic mechanism for alcohol to induce preterm delivery is related to an increase in the production of prostaglandins in accordance with alcohol intake. Labor records did not include variables related to the level of alcohol consumption during pregnancy so it is not possible from these data to make inferences about relative risk based on usage. However, the largest epidemiological study to date reported that as few as seven drinks per week during pregnancy is associated with increased risk for preterm delivery regardless of the type of intake be it beer, wine, or spirits (Albertsen et al., 2004). It is not well known how the timing of alcohol consumption might contribute to the risk for preterm delivery and data analyzed here do not lend clarification. The reported usage was obtained at the time of birth and is an indicator for the *general* level of exposure during pregnancy. This may imply misclassification for women who change their alcohol habits during pregnancy. Our results do not lead to inferences regarding the degree to which alcohol use is a causal agent in preterm delivery or whether it is indicative of alternative risk factors that may also be related to gestational age (Dole et al., 2003). For example, alcohol use may be a proxy for poor quality, erratic or delayed prenatal care, additional psychosocial stressors, or alternative high-risk lifestyles which may help explain the pathway between early sexual abuse and subsequent preterm delivery. Hence, these relationships remain difficult to tease apart.

The link between maternal stress biology and preterm delivery was not upheld in the sample of offspring born to abused mothers. This null finding may be indicative of the fact that in our data (Table I), and in several studies of adults retrospectively reporting childhood abuse (Heim et al., 2001; King et al., 2001), basal cortisol concentrations are consistent with various degrees of HPA axis *attenuation* (cortisol hyposecretion). These findings are consistent with the theory that individuals with initial chronic hyperarousal may develop a diminished capacity to sustain cortisol hypersecretion and, in order to avoid allostatic overload, adapt to a compensatory downward regulation of the HPA system (Schulkin, McEwen, & Gold, 1994; Susman, 2006). Attenuated cortisol secretion may be an adaptive response to allostatic overload and, thus, may not constitute a particular risk for preterm delivery in these women.

However, a significant positive correlation between maternal basal cortisol and preterm delivery *was* observed in our comparison sample. Thus our results may provide some hints into the connection between maternal stress biology and preterm delivery for women who have not experienced substantial childhood trauma. In general, environmental and behavioral manifestations of chronic or high stress such as anxiety disorders, highly stressful lifestyles, and inordinate succession of stressful life-events may predispose women to preterm delivery. A women's propensity to successfully manage stress prior to pregnancy may have implications for how she will fare when faced with stressors during pregnancy (Narendran, Nagarathna, Narendran, Gunasheela, & Nagendra, 2005). Managing maternal stress may have far-reaching benefits potentially extending to the next generation as higher levels of prenatal stress and stress hormones have been significantly associated with infant temperamental difficulties (Wadhwa, 2005), infant neurodevelopmental disorders (McIntosh, Mulkins, & Dean, 1995), and childhood psychopathology (Ward, 1991). These results underscore the compelling need to arrive at a better

understanding of the psychoneuroendocrine processes underlying preterm delivery.

The study has limitations that may limit generalization and/or suggest that results be replicated. Since a nonabusing caregiver was required to participate in the study, it is probable that the original abused sample overrepresents the population of sexually abused females who are believed and supported by their mother or other family members and underrepresents those who are not so believed and supported. This sample of offspring born to these women participating the longitudinal study is a convenience sample and was not randomly obtained from a larger population which may limit generalizability. Although maternal basal cortisol was averaged over at least two samples taken at the same time of day, there was no accounting for time since waking in our data thus resulting in intraindividual diurnal variation being relatively uncontrolled. There is the potential for misclassification of gestational age given possible errors in reporting of last menstrual period (Gjessing, Skjaerven, & Wilcox, 1999). Prenatal alcohol usage was crudely determined by self reports to physicians. As such, the completeness and accuracy of this measure is not known and results should be generalized accordingly. Finally, additional covariates that might constitute equally plausible explanations for preterm delivery (such as smoking during pregnancy) were not included in these analyses.

Although not directly addressed in the design of this study, findings reported here have implications for prevention and intervention. Risk for preterm delivery among women with histories of sexual abuse may be substantial, but the specific risk factors may not resemble those for women without such histories. The effects of childhood abuse may have lasting effects on female development, suggesting that interventions and treatment should continue throughout development and/or be revisited at critical points in development. With adequate child advocacy support, intake prenatal procedures could be made more comprehensive with inquiry about traumatic childhood abuse histories. After ruling out standard risk-factors commonly associated with normally developing females, practitioners might also attend to alternative prenatal risks for women with abuse histories and any comorbid conditions that may be indirectly associated with these risks such as alcohol dependence.

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