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The Differential Impacts of Early Physical and Sexual Abuse and Internalizing Problems on Daytime Cortisol Rhythm in School-Aged Children

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Abstract

The impact of early physical and sexual abuse (EPA/SA) occurring in the first five years of life was investigated in relation to depressive and internalizing symptomatology and diurnal cortisol regulation. In a summer camp context, school-aged maltreated (n = 265) and nonmaltreated (n = 288) children provided morning and late afternoon saliva samples on five consecutive days. Child self-report and adult observer reports of child internalizing and depressive symptoms were obtained. Children experiencing EPA/SA and high depressive or internalizing symptoms uniquely exhibited an attenuated diurnal decrease in cortisol, indicative of neuroendocrine dysregulation. These results were specific to EPA/SA, rather than later onset physical or sexual abuse or early occurring neglect or emotional maltreatment.

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What happens to biological and psychological functioning when there are severe dysfunctions in the early caregiving environment? The study of child maltreatment provides researchers with an excellent opportunity to address this question guided by preclinical research on the impact of early life stress on the development of fear and neuroendocrine dysregulation and by human research on maltreatment, depression and activity of the hypothalamic-pituitary-adrenocortical (HPA) system. The present report focuses on the impact of physical and sexual abuse (often in combination with neglect) during the first five years of life as an example of severe dysfunction of the early caregiving environment.

Among the array of difficulties exhibited by maltreated children, one of the most prevalent and widely documented is an increased risk for internalizing problems and depression in childhood and major depressive disorder in adulthood (Gibb, Wheeler, Alloy, & Abramson, 2001; Kaplow & Widom, 2007; Keiley, Howe, Dodge, Bates, & Pettit, 2001; Manly, Kim, Rogosch,

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& Cicchetti, 2001; Toth, Manly, & Cicchetti, 1992; Widom, DuMont, & Czaja, 2007). A variety of factors have been posited to mediate the impact of child maltreatment on depression. These include subtype of maltreatment, genetic vulnerability, the quality of children's relationships with their mothers, social support, stressful life events, attributional styles, social competence, self-esteem, and stress hormone activity (Cicchetti & Valentino, 2006). Regarding stress hormones, it has been argued that, for those at genetic risk of affective disorder, elevations in stress hormones during episodes of abuse may shape the development of more reactive fear and neuroendocrine systems, and hence increase the risk of developing internalizing disorders (Heim & Nemeroff, 2001).

The animal literature provides support for this hypothesis (Heim, Owen, Plotsky, & Nemeroff, 1997). Chronic elevations in glucocorticoids (cortisol in humans and other primates), produced by the HPA axis, increase the production of fear-mediating neurochemicals in the central nucleus of the amygdala (Makino, Gold, & Schulkin, 1994). In non-human primates, individuals carrying gene variants associated with risk for depression in abused children (e.g., Caspi et al., 2003), exhibit larger cortisol responses to psychosocial stressors (Barr et al., 2004; Sanchez et al., 2005). Furthermore, a large body of animal studies shows that adverse parental care during early development increases fearful, anxious behavior and shapes increased reactivity in neurobiological systems involved in defensive behaviors and physiological stress reactions (see Gunnar & Quevedo, 2007, and Gunnar & Vazquez, 2006, for reviews). In rodents and non-human primates, this has been demonstrated through studies of fear behavior and activity of the HPA axis (Levine, 2005; Maestripieri, 1999; Meaney & Szyf, 2005; Sanchez, McCormack, & Maestripieri, in press).

The neurobiological signature of adverse early care has been shown to be highly consistent at the systems and molecular levels to neurobiological findings in the study of adult mood disorder (see Heim, Newport, Mletzko, Miller, & Nemeroff, 2008; Heim, Plotsky, & Nemeroff, 2004). It has been argued that these early effects reflect both the dependence of the very young on maternal care for survival and the rapid development of the brain during the early period of life (Gunnar, 2003; Gunnar & Vazquez, 2006; Heim & Nemeroff, 2001). Thus, these animal studies suggest that inadequate or abusive parenting early in life persistently sensitizes the neurocircuits that are involved in the regulation of stress and emotion, while also suggesting that individuals at genetic risk for depression may be the most susceptible to these early experience effects (Heim & Nemeroff, 2001).

The HPA system has figured prominently in research linking early adverse care to later internalizing and mood disorders for a number of reasons. First, this neuroendocrine system is frequently dysregulated in adults with clinical depression (Holsboer, 2000). Second, both the releasing hormone (corticotropin-releasing hormone, CRH) and the final hormonal product of this system (cortisol in humans) play critical roles in brain development, affecting neurotrophic factors and regulating gene transcription (Gunnar & Vazquez, 2006). Extremely adverse early care has been shown in animal models to increase cell death in the infant brain (Zhang et al., 2002). During adverse early care, CRH produced in the hippocampus disturbs its normal development (Fenoglio, Brunson, & Baram, 2006) and chronic early stress produces increases in CRH production in the amygdala and a shift in the expression of receptors towards those supporting heightened fear and anxiety (Sanchez, Ladd, & Plotsky, 2001). Finally, heightened fear and activation of the HPA system as a function of early adverse care are then expected to produce an increase in allostatic load over the life course, increasing vulnerability to mood disorders in response to stressors experienced later in development (McEwen, 2003; McEwen & Stellar, 1992).

Models of the impact of early caregiving adversity typically focused on *increased* reactivity of the HPA axis as a likely outcome. However, particularly as the research has been extended

to human and non-human primates, it has become clear that a more accurate prediction is that the HPA axis will likely exhibit evidence of *dysregulation* and that this dysregulation may involve both hyper- and hypo-functioning (McEwen, 2000; Heim et al., 2004, 2008). Although not yet fully understood, a number of factors have been shown to affect whether elevated or suppressed activity is noted. The HPA axis tends to down-regulate in response to prolonged hyper-activation; thus in response to chronic stressors first hyper- and later hypo-activity of the axis may be noted (see for review, Miller, Chen and Zhou, 2007). With regards to basal cortisol, chronic stress and negative emotionality tend to be associated with slight elevations in the late afternoon and evening, but often lower than typical levels in the early morning (e.g., DeSantis, Adam, Doanne, Mineka, Zinbarg, & Craske, 2007; Gunnar & Quevedo, 2007). Thus, whether elevations or suppressions in baseline levels are reported may depend on time of measurement both in terms of time of day and time since the onset of a chronic stressor. Furthermore, in part because of counter-regulation within the axis, cortisol measures (baseline or reactivity) may appear normal, even when assessments higher up in the axis (e.g., ACTH response to CRH challenge or pharmacological probes of feedback sensitivity) may reveal dysfunction (see, for example, Heim et al., 2008). Finally, there is some speculation that individuals at genetic risk for antisocial personality disorder will exhibit attenuation or down-regulation of cortisol activity (Susman, 2006), while individuals at risk for depression will exhibit elevations or hyper-activity of the HPA axis following adverse early life conditions (Heim & Nemeroff, 2001; Heim et al., 2004). Overall, predicting that the measures of the HPA system will reflect dysregulation of the axis, rather than predicting that any given measure will be elevated or suppressed, reflects the current status of knowledge in the field. This is especially true in research on young children from vulnerable populations, where researchers are often limited to assessing basal measures of cortisol at only one or a few times during the day.

With these caveats in mind, we review the literature on adverse early caregiving, including maltreatment, and its association with HPA axis dysregulation in humans. Just as not all individuals who experience abuse and neglect as young children grow up to develop clinical depression (Cicchetti & Rogosch, 1996), not all go on to exhibit patterns of HPA axis dysregulation. There is increasing evidence that adults, who were abused as children, exhibit dysregulation of the HPA axis only or primarily if they have major depression and that adults with major depression in the absence of early child abuse do not show evidence of neuroendocrine dysregulation (Heim et al., 2004, 2008). The results of Heim and colleagues (2001) are consistent with studies of children and adolescents with depression. Non-maltreated children with early onset depression rarely display evidence of regulatory dysfunction of the HPA axis (Feder, Coplan, Goetz, Mathew, Pine, Dahl, Ryan, Greenwald, & Weissman, 2004). Evidence of dysregulation has, however, been noted for depressed children who also were sexually abused and suicidal (Dahl & Ryan, 1996) and for children who were abused and who have chronic posttraumatic stress disorder (PTSD; Carrion et al., 2002; DeBellis, Baum, et al., 1999). Similarly, two previous studies of maltreated children have shown that evidence of diurnal dysregulation of the HPA axis (slightly lower morning and slightly elevated afternoon cortisol levels) tends to be observed only in maltreated children who are also suffering from depression (Hart, Gunnar & Cicchetti, 1996; Kaufman, 1991).

Evidence of dysregulation extends to studies of pharmacological challenge. Specifically, Kaufman et al. (1997) administered a CRH challenge test to a sample of depressed abused, depressed nonabused, and normal control group children. Compared to the depressed nonabused and the normal control group children, the depressed abused children exhibited significantly greater peak, total, and net ACTH secretion post-CRH infusion. Finally, Cicchetti and Rogosch (2001b) found that compared to symptomatic nonmaltreated children, maltreated children with clinical-level internalizing problems were distinguished by higher morning, afternoon, and average daily cortisol levels across a 5-day period, suggestive of HPA axis dysregulation.

While dysregulation of the HPA axis appears to be associated with maltreatment for children who are high in internalizing problems or who are clinically depressed, it remains unclear whether this is due to the type, severity, or developmental timing of the maltreatment. To date, the relevant published studies have either focused on physical or sexual abuse (DeBellis, Baum, et al., 1999; Heim & Nemeroff, 2001; Kaufman et al., 1997) or have not examined a specific subtype of maltreatment. However, most maltreated children experience multiple types of maltreatment, and the previous studies have not attempted to differentiate among them (Cicchetti & Barnett, 1991; Cicchetti & Rizley, 1981). In particular, abuse and neglect frequently co-occur. Moreover, these maltreatment subtypes have different onsets, occur with varying severities, and can recur (Bolger, Patterson, & Kupersmidt, 1998; English et al., 2005; Manly et al., 2001). In addition, children experience maltreatment during different developmental periods. Thus, maltreatment is a heterogeneous phenomenon, and researchers and clinicians must consider the diversity in the type(s), perpetrator(s), onset, frequency, chronicity, and timing of maltreatment experiences. Even more striking, few such investigations also include a measure of timing, a critical variable in neuroendocrinology (Tarullo & Gunnar, 2006). In particular, inclusion of the temporal dimension is especially important because animal studies have revealed that timing is a crucial factor. Indeed the empirical evidence gleaned from rodent and nonhuman primate investigation indicates that adverse or maltreating (i.e., neglectful and abusive) care early in an animal's life has long-lasting effects. Attempts to translate the animal research on the criticality of timing have utilized a range of time periods (i.e., infancy through adolescence) to specify what constitutes early adverse care. Thus, while there is some evidence that HPA axis dysregulation is associated with maltreatment in the context of depression, little is known about whether the type, timing, or duration of maltreatment is relevant to these findings.

Because, based on the animal literature, dysfunction in the caregiving system early in life is believed to increase the risk of depression in genetically vulnerable individuals (e.g., Heim et al., 2004, 2008), the literature on maltreatment and risk of depression might provide guidance. However, similar problems exist in reviewing the literature on maltreatment and internalizing disorders. Most studies examining maltreatment and depression risk have not examined maltreatment subtypes. Those studies that have examined subtypes often focus on one (e.g. sexual abuse) and not others. Not surprisingly, the results of these studies are inconsistent; some investigations reveal that physically abused and sexually abused children have the greatest likelihood of developing depression, whereas others have shown this is the more likely outcome for neglected and emotionally maltreated children (Cicchetti & Valentino, 2006). Likewise, few prospective studies have examined the interaction between timing and type(s) of maltreatment on depression outcome.

One study that did address all subtypes of maltreatment found that serious neglect experienced during the preschool period was associated with a higher rate of internalizing symptoms (Manly et al., 2001). Another investigation indicated that the experience of harsh physical punishment before age five was related to higher teacher ratings of internalizing behaviors in kindergarten children (Keiley et al., 2001). Unfortunately, all types of maltreatment were not addressed in this study. In a study of adults, however, Kaplow, Dodge, Amaya-Jackson, and Saxe (2005) discovered that an earlier age of sexual abuse onset predicted higher levels of anxiety years later. Similarly, Kaplow and Widom (2007) found that individuals whose age of onset of maltreatment (i.e., physical abuse or neglect) was prior to 5 years of age reported higher levels of depressive and anxiety symptoms later in life than did individuals with a later onset of maltreatment (i.e., sexual abuse). These studies suggest that early occurring maltreatment may be particularly important in the experience of later depression and anxiety. However, limitations in the range of subtypes examined within the studies or the extent to which specific subtypes were adequately represented in the sample preclude definitive conclusions

In the current investigation, we operationalized early maltreatment as any incident of physical abuse, sexual abuse, neglect, or emotional maltreatment occurring within the first 5 years of life. We chose the infancy, toddler, and preschool periods as our index of early maltreatment for two primary reasons. First, national epidemiological studies of child maltreatment highlight this period as a critical time for the emergence of neglect and abuse (U. S. Department of Health and Human Services, Administration on Children, Youth, and Families, 2000). Second, despite the fact that the neural systems underlying stress reactivity/regulation and internalizing problems undergo a prolonged period of development, components of the limbic system such as the hippocampus, amygdala, and the pathways to the prefrontal cortex from these structures develop rapidly over the first years of life (Thompson & Nelson, 2001).

We also chose to focus on physical and sexual abuse experienced early in the child's life (in the first 5 years). In order to extend the work of Heim (see, e.g., Heim et al., 2008), we utilized prospective reporting of abuse, an earlier age of abuse onset, and evidence for depressive and internalizing symptomatology and cortisol dysregulation occurring earlier in development during the school-age years.

Hypotheses and research questions

This study was designed to examine the hypothesis that children who were physically abused, sexually abused, or both prior to age 5 (early physical abuse or sexual abuse; EPA/SA) and who expressed high depressive and internalizing symptoms would exhibit a more dysregulated pattern of diurnal cortisol activity (less decline; flatter slope) than would other maltreated children (no early physical or sexual abuse; NEPA/SA) or nonmaltreated comparison (NC) children. Prior to testing this hypothesis, we first examined whether EPA/SA children were at higher risk for developing high depressive or internalizing symptoms than were NEPA/SA or NC children. We also explored the specificity of the finding by contrasting cortisol patterns of the high depressive or internalizing symptom EPA/SA children and subsets of the other maltreated children. First we examined the contrast with high and low symptom children who experienced other forms of maltreatment (neglect, emotional maltreatment) *prior* to age 5. Next we examined the contrast with high and low symptom children whose physical or sexual abuse began after age 5. Finally, we examined whether other parameters of maltreatment (onset, recency, chronicity, diversity, and perpetrators) might be alternative processes that could account for the atypical diurnal regulation for high symptom EPA/SA children.

METHOD

Participants

Participants included 553 children aged seven to thirteen (*M* age = 10.02, *SD* = 1.87) who attended a summer day camp research program designed for school-aged low-income children. The sample included both maltreated children (*n* = 265) and nonmaltreated children (*n* = 288). All of the children were in their first year of camp attendance. The children were not in foster care placements and were residing with their biological mothers. Demographic characteristics are presented in Table 1 for the EPA/SA, NEPA/SA, and NC groups. The proportion of boys differed between the NC and both the EPA/SA and NEPA/SA groups, χ^2 (2) = 17.86, p < .001; thus, gender was controlled in analyses. The groups did not differ in age, race/ethnicity, or in family history of receiving welfare benefits.

Recruitment and Classification Procedures

Parents of all children provided informed consent for their child's participation, as well as for examination of Department of Human Services (DHS) records pertaining to the family. Children in the maltreated group had been identified by the county DHS as having experienced child abuse or neglect, and the sample was representative of the children in families receiving

services from the DHS. A recruitment liaison from DHS contacted eligible maltreating families, explained the study, and if parents were interested, then their names were released to the project team for recruitment. Families were free to choose whether or not to participate. Comprehensive searches of DHS records were completed and maltreatment information was coded utilizing operational criteria from the maltreatment nosology specified in the *Maltreatment Classification System* (MCS: Barnett, Manly, & Cicchetti, 1993), as discussed below.

Consistent with national demographic characteristics of maltreating families (NIS-3; Sedlack & Broadhurst, 1996), the maltreated children were predominantly from low socioeconomic status families. Consequently, demographically comparable nonmaltreated children were recruited from families receiving Temporary Assistance for Needy Families (TANF). A DHS recruitment liaison contacted eligible nonmaltreating families, described the project, and if interested, parents signed a release for their names to be given to the project for recruitment. DHS record searches were completed for these families to verify the absence of any record of child maltreatment. Trained research assistants also interviewed mothers of children recruited for the nonmaltreatment group to confirm a lack of DHS involvement and prior maltreatment experiences utilizing the *Maternal Maltreatment Classification Interview* (Cicchetti, Toth, & Manly, 2003). Subsequently, record searches were conducted in the year following camp attendance to verify that all available information had been accessed. Only children from families without any history of documented abuse or neglect were retained in the nonmaltreatment group. In addition, families who had received preventive services through DHS due to concerns over risk for maltreatment were excluded from the sample to reduce the potential for unidentified maltreatment existing within this group.

Classification into maltreatment subgroups was accomplished through use of the *Maltreatment Classification System* (MCS: Barnett et al., 1993). The MCS is a reliable and valid method for classifying maltreatment (Bolger et al., 1998; English et al., 2005) that utilizes DHS records detailing investigations and findings involving maltreatment in identified families over time. Rather than relying on official designations and case dispositions, the MCS codes all available information from DHS records, making independent determinations of maltreatment experiences. Based on operational criteria, the MCS designates all of the subtypes of maltreatment children have experienced (i.e., neglect, emotional maltreatment, physical abuse, sexual abuse) and the severity of each type on scales that range from 1 (least) to 5 (most) severe. Based on documented records, the MCS also determines when in the course of development maltreatment events occurred, providing indices of timing. Events were coded as occurring during five developmental periods, including infancy (0–12 months), toddlerhood (13–36 months), preschool (36 to 60 months), early school age (age 5 to 7), and later school age (age 8 to 12). The timing information allows for the determination of whether maltreatment occurred within each of the developmental periods. The developmental period of *onset* of maltreatment and the *recency* of maltreatment can be determined, and the number of developmental periods of maltreatment serves as an index of *chronicity*. Moreover, the developmental timing of each of the maltreatment subtypes also can be specified.

Coding of the DHS records was conducted by trained research assistants, doctoral students, and clinical psychologists. Coders were required to meet acceptable reliability with criterion standards before coding actual records for the study. Coders demonstrated acceptable reliability (weighted κ‘s with the criterion ranging from .86 to .98). Reliabilities for the presence vs. absence of maltreatment subtypes (κ's of .90 to 1.00) and for individual subtypes severities (weighted κ's of .75 to 1.00) also were established.

In terms of the subtypes of maltreatment, *neglect* involves failure to provide for the child's basic physical needs for adequate food, clothing, shelter, and medical treatment. In addition to

inadequate attention to physical needs, forms of this subtype include lack of supervision, moral-legal neglect, and education neglect. *Emotional maltreatment* involves extreme thwarting of children's basic emotional needs for psychological safety and security, acceptance and self-esteem, and age-appropriate autonomy. Examples of emotional maltreatment of increasing severity include belittling and ridiculing the child, extreme negativity and hostility, exposure to severe marital violence, abandoning the child, and suicidal or homicidal threats. *Physical abuse* involves the non-accidental infliction of physical injury on the child (e.g., bruises, welts, burns, choking, broken bones). Injuries range from minor and temporary to permanently disfiguring. Finally, *sexual abuse* involves attempted or actual sexual contact between the child and a family member or person caring for the child for purposes of that person's sexual satisfaction or financial benefit. Events range from exposure to pornography or adult sexual activity, to sexual touching and fondling, to forced intercourse with the child.

Children in the maltreatment group all had histories of abuse, neglect, or both occurring in their families according to DHS records. Among the maltreated children, 79.2% had experienced neglect (*M* neglect severity for those with neglect = 3.42, *SD* = 1.13), 67.8% had experienced emotional maltreatment (*M* severity = 3.44, *SD* = 1.24), 32.6% had experienced physical abuse (*M* severity = 2.56, *SD* = 1.04), and 8.3% (*M* severity = 2.86, *SD* = 0.94) had experienced sexual abuse. The majority of children had experienced multiple subtypes of maltreatment. Specifically, 65.2% of the maltreated children had experienced two or more maltreatment subtypes, and 13 of the 15 possible combinations of presence vs. absence of each of the four maltreatment subtypes were observed in the sample. On average, the maltreated children had experienced 1.89 (*SD*= .78) subtypes of maltreatment. The number of subtypes of maltreatment provides an index of maltreatment *diversity*.

Based on the MCS, we identified a group of children (*n* = 60) who had experienced *early abuse*, i.e., physical or sexual abuse or both occurring prior to age 5 in the infancy, toddler, or preschool developmental periods (EPA/SA). In primary analyses, these children were contrasted with the remaining maltreated children (*n* = 205) who had *not* experienced early physical or sexual abuse (NEPA/SA), e.g., experienced neglect or emotional maltreatment only or later onset of physical or sexual abuse.

Day Camp Procedures

Children attended a week-long day camp program and participated in research assessments (see Cicchetti & Manly, 1990, for detailed descriptions of camp procedures). Children were transported by bus to the camp each day, with travel time averaging 45 minutes. At the camp, children were assigned to groups of eight (4 maltreatment, 4 control) same-age and same-sex peers. Each group was led by three trained camp counselors, who were unaware of the maltreatment status of children and the hypotheses of the study. Camp lasted 7 hrs/day for five days, providing 35 hours of interaction between children and counselors. In addition to the recreational activities, after providing assent, children participated in various research assessments and provided morning and afternoon saliva samples. Trained research assistants, who also were unaware of research hypotheses and maltreatment status, conducted individual research sessions with children, in which questionnaires and other research measures were administered. Clinical consultation and intervention occurred if any concerns over danger to self or others emerged during research sessions. The counselors, who had been trained extensively for two weeks prior to the camp, also completed assessment measures on individual children, based on their 35 hours of observations and interactions with children in their respective groups. Counselors were unaware of the research hypotheses and children's maltreatment status.

Day Camp Measures

Saliva Collection and Preparation—The camp context allowed for a setting where a consistent collection of saliva samples could occur at uniform times across the camp week. Each day upon arrival to the camp at 9:00 a.m., trained research assistants obtained saliva samples from each child. Given the approximate 45-minute transportation time and the time spent being greeted by camp staff, children had been awake at least one hour prior to providing the morning saliva samples, thus avoiding the period of the dynamic cortisol awakening response (cf. Susman, Dockray, Dorn, Schiefelbein, Herwehe, & Heaton, 2007). Samples also were obtained each day prior to the children's departure from the camp at 4:00 p.m.

The children had not consumed food or drink for at least 30 minutes before each saliva sample was obtained. Samples were collected following the method recommended by Granger et al. (1999). The children were asked to chew Trident® sugarless original flavor gum to stimulate saliva flow and then passively drool though a short drinking straw into a 20 ml plastic vial. The samples were immediately frozen and stored at −40° C. Each week, the samples were shipped overnight on dry ice for next day delivery to Salimetrics Laboratories (State College, PA) for assay. Cortisol was assayed in duplicate by Salimetrics, Inc using an enzyme immunoassay (Salimetrics, State College, PA). This assay has a lower limit of sensitivity of 0.007 μg/dl (range up to 1.8 μg/dl), and average intra-and inter-assay coefficients of variation less than 5.0% and 10.0%, respectively. Units of cortisol are expressed in μg/dl (micrograms per deciliter). Across camp days, the mean morning cortisol level was .19 (*SD* = .11), and the mean late afternoon level was .11 (*SD* = .07). As cortisol values were positively skewed and leptokurtotic, logarithmic transformations (log10) of cortisol values were used for all statistical analyses. Values were averaged across days to yield one morning and one afternoon cortisol measure for each child.

Internalizing and Depressive Symptoms

Children's Depression Inventory (CDI; Kovacs, 1982, 2004)—The CDI is a widely used self-report questionnaire to assess depressive symptomatology in school-age children. For each item, children chose from among three option statements, depicting increasing levels of depressive symptoms, in order characterize their experiences in the past two weeks. Kovacs (2004) reports that internal consistency for the total scale has ranged from .71 to .89, and validity has been well established. In the current investigation, internal consistency was .85. A total score of 19 and above has been established as a cutpoint for clinical level depressive symptoms (Kovacs, 2004). In the current sample, 11.4% (*n* = 63) of the children reported clinical level depressive symptoms.

Teacher Report Form (TRF; Achenbach, 1991)—Behavioral symptomatology was evaluated at the end of each week by counselors' completion of the TRF. The TRF is a widely used and validated instrument to assess behavioral disturbance from the perspective of teachers, and the measure was used in the present study, because camp counselors are able to observe similar behaviors to that of teachers. The TRF, containing 118 items rated for frequency, assesses two broadband dimensions of child symptomatology, internalizing and externalizing, as well as total behavior problems. In the present investigation, we focus on the internalizing dimension, which includes subscales of withdrawn, somatic problems, and anxiety/depression. Interrater reliability for the internalizing scale was based on average intraclass correlations among pairs of raters. Across the years, reliabilities for internalizing ranged from .56 to .84 (*M* = .68), consistent with the greater difficulty in observers rating internalizing symptoms (Achenbach, 1991). Internal consistency was .84. The counselors' scores for each child were averaged to obtain individual child scores for the internalizing dimension. Utilizing the T-Scores for the internalizing scale, a cutpoint of 60T was used to designate the presence or

absence of high internalizing symptoms (Achenbach, 1991). In the sample, 9.9% of the participants (n = 55) were classified as having high level internalizing problems.

Summary Measure of Internalizing—To yield a multi-informant index of children displaying high depressive and internalizing symptomatology, child and counselor measures were combined. Overall, the CDI and TRF internalizing scores were significantly but not strongly correlated, r = .20, p < .001. Similarly, the high symptom level groups for the CDI and TRF were significantly but weakly associated, χ^2 (1, N = 553) = 4.48, p = .03. This level of correspondence is consistent with the degree of agreement typically found between different sources of report for child internalizing symptoms. To combine both perspectives, the presence of high symptoms on either the CDI or the TRF was determined and used as an index of high depressive and internalizing symptomatology, resulting in 19.3% of the children being classified as symptomatic.

RESULTS

Plan of Analysis

The analysis was complicated by the complexity of maltreatment subgroup categorization, based on maltreatment subtype and developmental timing of individual subtypes, and concomitant cell sizes, particularly when participants were further classified by the presence of high vs. low depressive or internalizing symptoms. We, therefore, pursued a layered approach to these analyses. First, maltreatment parameters of the early physical or sexual abuse group (EPA/SA) were examined relative to characteristics of other maltreated children (NEPA/SA) for descriptive purposes. Next, we examined whether the EPA/SA group differed from the NEPA/SA and NC children on depressive and internalizing symptoms, as well as morning and afternoon cortisol levels. Third, we examined main and interaction effects of early abuse and depressive or internalizing symptoms on cortisol levels and change from morning to late afternoon. Gender and age effects also were probed. Then, using only the maltreated children, we examined the specificity of early physical and sexual abuse: first, relative to *early* neglect or emotional maltreatment without early abuse, and next, relative to *later onset* of physical or sexual abuse at age 5 or older. Finally, other aspects of maltreatment timing, diversity, and perpetrator were considered to determine whether these features of maltreatment influenced cortisol regulation in the context of high symptomatology.

Characteristics of the Early Physical or Sexual Abuse Group

Within the EPA/SA group, 80% had experienced physical abuse and 28.3% had experienced sexual abuse; five children had experienced both maltreatment subtypes. Physical and sexual abuse often occurred in the presence of neglect and emotional maltreatment. In fact, 73.3% of the children in the EPA/SA group had experienced neglect, and 73.3% had also experienced emotional maltreatment. Overall, the EPA/SA group had been exposed to an average of 2.55 subtypes of maltreatment (SD = .70). The individual subtype severities for children who had experienced respective subtypes did not differ between the EPA/SA and NEPA/SA groups (p's ranged from .15 to .77).

Among the maltreated children 89.8% had experienced some form of maltreatment from their mothers, and this rate did not differ for the EPA/SA (91.7%) versus NEPA/SA children (89.3%) groups, χ^2 (1, N = 265) = .29, p = .81. The rate of fathers as perpetrator also was comparable by group, χ^2 (1, N = 265) = .04, p = .88, 43.3% versus 42.0%. However, the rate of others as perpetrator was higher in the EPA/SA group (58.3%) than the NEPA/SA group (35.1%), χ^2 (1, N = 265) = 10.39, p = .002.

For the EPA/SA group, the onset of maltreatment had occurred in infancy for 43.3%, in toddlerhood for 25.0%, and in preschool for 31.7% of the early abuse group. Moreover, 63.3 % of the children had been maltreated during the toddler period and 93.3% had been maltreated in the preschool period. However, in terms of recency, only 18.4% had been maltreated beyond the preschool period. Chronicity of maltreatment, as indexed by the number of developmental periods of maltreatment, averaged 2.22 developmental periods (*SD* = .92).

Group Differences on Depressive and Internalizing Symptoms and Cortisol

ANOVAs were conducted to contrast children in the EPA/SA, NEPA/SA and NC groups on CDI and TRF internalizing scores (see Table 2). Results differed depending on whether child self-report or counselor reports were used. For child self reported symptoms on the CDI, the main effect of group was marginally significant, $F(2,550) = 2.62$, $p = .07$. In contrast, significant differences for group classification were found for counselor-reported TRF internalizing symptoms, $F(2,550) = 9.14$, $p < .001$. Tukey post hoc tests indicated that the EPA/SA group had higher internalizing scores than the NEPA/SA and NC children.

As described in the methods, the presence of high symptoms on the CDI or the TRF was used as an index of high depressive and internalizing symptomatology. Significant group differences were observed on the presence or absence of high symptoms, χ^2 (2, $N = 553$) = 11.71, $p = .003$. Follow-up contrasts indicated that the EPA/SA group (33.3%) had significantly higher rates of symptoms than the NC children (14.9%), χ^2 (1, $N = 348$) = 11.34, $p = .001$, and marginally higher rates than the NEPA/SA children (21.5%), χ^2 (2, $N = 265$) = 3.57, $p = .06$. Finally, the NEPA/SA maltreated children had marginally higher rates of elevated symptoms than the nonmaltreated children, χ^2 (2, $N = 493$) = 3.52, $p = .06$. Finally, as shown in Table 2, the groups did not differ on average morning, $F(2,550) = 1.52$, $p = .22$, or afternoon cortisol levels, $F(2,550) = .07$, $p = .94$.

Diurnal Cortisol Regulation: The Interaction of Early Physical and Sexual Abuse with Depressive and Internalizing Symptomatology

A 3 (early physical/sexual abuse groups) by 2 (depression/internalizing level) by 2 (AM/PM cortisol) repeated measures ANCOVA was conducted, covarying age and gender (see Table 3). In addition to significant gender and age effects, a significant main effect was found for symptom level and a marginally significant main effect was obtained for early physical or sexual abuse classification group. However, these main effects were qualified by a significant interaction among group, symptom level, and AM/PM, $F(2,545) = 6.07$, $p = .002$. Follow-up tests for simple effects were conducted separately within the high and low symptom levels. As shown in Figure 1a, for children who were not high on depressive or internalizing symptoms, no effect of group on diurnal variation in cortisol was obtained, $F(2,441) = .81$, $p = .45$. As shown in Figure 1b, for children with high depressive or internalizing problems, group status was a significant predictor of diurnal cortisol activity, $F(2,104) = 5.94$, $p = .004$. To further pinpoint the effect for the high symptom children, we computed the AM to PM decline (AM minus PM) and subjected these scores to an ANCOVA now entering morning cortisol, gender and age as covariates. The effect of group was significant, $F(2,101) = 4.27$, $p = .017$. Bonferroni-corrected post hoc comparisons indicated that the EPA/SA group had a significantly lower diurnal decline in cortisol (0.12) than the NEPA/SA group (0.23) and the NC group (0.25). The morning and afternoon scores also were examined separately. Although the group differences were in the expected direction (see Figure 1b), the groups did not differ significantly at either time of day. Thus, the EPA/SA group with high depressive or internalizing symptoms was distinguished by the smaller decrease or attenuated slope of the daytime diurnal rhythm of cortisol, due to slightly but not significantly lower AM and slightly but not significantly higher afternoon levels than the other children.

The effects of the covariates of gender and age on the within subjects diurnal decrease in cortisol are represented in the repeated measures ANCOVA by the respective interactions of the covariates with time. To interpret the effects of the covariates, the difference between morning and afternoon cortisol was computed, and *t*-tests were conducted for gender and age groups (< 10 and ≥ 10). Gender differences were found, t (551) = 2.83, p = .005, such that girls had a significantly greater cortisol decrease across the day (M = .27, SD = .17) than boys (M = .22, SD = .19). Although morning cortisol levels did not differ by gender, t (551) = 0.74, p = .46, afternoon levels did, t (551) = 2.41, p = .02, with boys having higher cortisol (M = −1.02, SD = .16) than girls (M = −1.05, SD = .16). Interpretation of the covariate effect of age involved a significant effect of age group on diurnal cortisol decrease, t (551) = 6.12, p < .001, with older children evincing a steeper decrease (M = .29, SD = .19) than younger children (M = .19, SD = .17). Older children had significantly higher morning cortisol levels than younger children, t 20 (550.9) = 5.46, p < .001, M older = −.74, SD = .20; M younger = −.83, SD = .17). Afternoon cortisol did not differ, t (551) = .56, p = .58.

Because puberty is associated with a rise in basal and reactivity measures of the HPA axis (Adam, 2006; Gunnar, Wewerka, Frenn, Long, & Griggs, in press; Stroud, Foster, Handwerger, Papandonatos, Granger, et al., in press) and is highly correlated with age, and because age was a significant covariate in the above analyses, we examined whether age might interact with the group by symptom level effects on the diurnal decline in cortisol reported above. To examine this question, we used a regression analysis with diurnal decrease as the dependent factor. EPA/SA versus others, age group, and symptom level were entered as dummy variables, followed two-way interaction terms, and finally the three-way interaction of age, symptom level, and group. Importantly, the results did not yield a significant three-way interaction, β = .01, p = .73, indicating that age did not moderate the overall findings observed involving attenuated diurnal decrease in children with early abuse and high symptoms. Similarly, we considered whether gender would moderate the effects of early abuse and symptom level. An analogous regression analysis, as was conducted for age, also was carried out with gender; however, the three-way interaction of early abuse, symptom level, and gender also was not significant, β = −.01, p = .82, indicating that the effects were not operating differently among boys and girls.

Early Physical/Sexual Abuse versus Early Neglect/Emotional Maltreatment

To examine whether other forms of early maltreatment exerted an impact on the diurnal decrease in cortisol, we examined only the children whose maltreatment onset was prior to age 5. There were 151 children who experienced neglect or emotional maltreatment but not physical or sexual abuse, and these children were contrasted with the EPA/SA group. The severity of emotional maltreatment and of neglect did not differ between these two groups. In an ANCOVA using the decrease in cortisol as the dependent measure, the early maltreatment subtype designation and high vs. low symptom status were used as grouping factors, and morning level cortisol, age, and gender as covariates. A significant main effect was found for symptom status, F(1,204) = 13.13, p < .001; however, this effect was clarified by a significant interaction effect of symptom status and maltreatment type, F(1,204) = 7.29, p = .007. To probe this interaction effect, the four groups in the interaction were contrasted with Bonferroni post hoc tests. The interaction effect is shown in Figure 2, panel 1. The EPA/SA group with high symptoms (M = 0.13, SD = .23) showed a smaller diurnal decrease than any of the other groups. The difference was highly significant for each of the low depressive symptom groups: early neglect-emotional maltreatment (M = 0.24, SD = .17, p = .004) and early physical-sexual abuse (M = 0.27, SD = .17, p = .001), and was marginally significant for the early neglect, high depressive symptom group (M = 0.22, SD = .17; p = .10). No other contrast was significant.

The Effect of Early versus Late Onset of Physical/Sexual Abuse

We also sought to determine whether early physical or sexual abuse was responsible for the observed effects, or whether physical or sexual abuse with later onset also would demonstrate the attenuated diurnal decrease in cortisol. Accordingly, children who experienced the onset of physical or sexual abuse beyond the preschool period were identified (n = 41), and these children were contrasted with the EPA/SA group. The severity of physical abuse and of sexual abuse did not differ between these abuse onset groups. In an ANCOVA using the decrease in cortisol as the dependent measure, the abuse onset group and high vs. low symptom status were used as grouping factors, and morning level cortisol, age, and gender as covariates. As shown in Figure 2, a significant interaction between timing of abuse onset (< 60 mo., ≥ 60 mo.) and the presence of high depressive or internalizing symptoms was found, $F(1,94) = 11.87$, $p = .001$. The interaction was probed through Bonferroni-corrected post hoc comparisons. The EPA/SA group with high symptoms ($M = 0.12$, $SD = .24$ had a significantly lower decrease in cortisol than the later abuse/low level symptom group ($M = 0.29$, $SD = .20$; $p = .05$) and the EPA/SA group with low level symptoms ($M = 0.27$, $SD = .17$, $p = .001$); however, a significant contrast was not found with the later abuse and low level symptom group ($M = 0.21$, $SD = .20$; $p = .25$). Other contrasts were not significant. These results again highlight the EPA/SA - high symptom group as uniquely demonstrating a pattern of attenuated diurnal decrease in cortisol.

Features of Maltreatment Timing, Diversity, and Perpetrator

Finally, we considered whether the onset period of maltreatment, recency of maltreatment, chronicity of maltreatment (number of developmental periods of maltreatment), diversity of maltreatment experiences (number of subtypes of maltreatment), and mother, father, and others as perpetrators of maltreatment, provided more discriminating perspectives on the atypical pattern of cortisol change across the day observed in early physically or sexually abused - high symptom children. For each of these seven maltreatment features, analogous repeated measures ANCOVAs were conducted, as had been done with the early abuse group classification. High versus low depressive and internalizing symptom status was included as a second main effect, with age and gender as covariates. We determined if any significant interaction effects for time of day, symptom status, and the respective maltreatment feature were significant in relation to cortisol regulation across the day. None of the maltreatment parameters we considered resulted in a significant interaction effect (p's = .26, .46, .32, .19, .48, .16, and .99, for onset, chronicity, recency, diversity, and mother as perpetrator, father as perpetrator, and other as perpetrator of maltreatment, respectively).

DISCUSSION

In this investigation we sought to examine whether children experiencing physical or sexual abuse in the early years of life and exhibiting high internalizing symptomatology displayed a unique signature of cortisol levels across the day suggestive of neuroendocrine dysregulation. The results supported our predictions. First, we found that children experiencing abuse in the first five years of life exhibited more internalizing symptoms than maltreated children without early abuse and nonmaltreated children of the same socioeconomic level. Second, children experiencing early abuse who had high internalizing symptoms exhibited an atypical flattening of cortisol production over the daytime hours. Further analyses indicated that, even among children high on internalizing symptoms, other parameters of maltreatment, including developmental period of onset, recency, chronicity, diversity, and type of perpetrator, did not provide a better explanation of the phenomena than did the experience of early physical or sexual abuse.

The pattern of cortisol production noted for the early abused/high internalizing children provided consistent evidence of HPA axis dysregulation as noted in adults and children under chronic stress (Gunnar & Vazquez, 2001; Heim et al., 2000, 2008; Miller et al., 2007). That is, we obtained evidence of a less marked decline in cortisol over the daytime due to a slight lowering of AM and slight increase in PM levels. Given the times we were able to sample saliva, it is somewhat remarkable that we were able to detect this pattern of dysregulation. Cortisol is at its daily peak approximately 30 minutes after morning awakening, drops steeply for the next 30 minutes to an hour, and then declines gradually over the day, until it reaches almost a zero nadir soon after the onset of nighttime sleep (cf. Susman et al, 2007). Most studies that have identified this stress-related pattern of daytime cortisol dysregulation have sampled at morning wake up and at bedtime. In the present study, in contrast, while we sampled the children at 9 AM it is very likely that the children had been awake long enough so that we missed the early morning peak. Although 4 PM is late in the afternoon, levels at this time are not as low as the nadir that they will reach later in the evening. Thus, it is possible that the pattern we noted would have been even more marked had we been able to sample earlier and later in the day.

Whereas the pattern we noted is not the same as that seen among adults with clinical depression, the findings are consistent with those described in two other reports on depressed, maltreated children studied in a day camp setting (Hart et al., 1996; Kaufman, 1991). Hart and colleagues found that maltreated children who were also depressed showed a less marked diurnal decrease in cortisol from 9 AM to 4 PM, although no attempt was made in that study to differentiate patterns based on subtype of maltreatment. Kaufman (1991) noted that maltreated and depressed children in her sample were less likely than non-depressed maltreated children to show a decrease in cortisol from 9 AM to 4 PM. Notably similar to the present study, she also observed that the depressed maltreated children were more likely to have suffered abuse in addition to neglect, rather than solely neglect. Neither the Hart et al. study nor the Kaufman et al. study examined the influence of the developmental timing of maltreatment subtypes.

We also found that both gender and age were associated with morning to afternoon change in cortisol level. Whereas girls had significantly lower afternoon cortisol and greater diurnal decrease than boys, gender did not moderate the effects of early physical or sexual abuse and high symptoms. In terms of age, consistent with the literature, older children were found to have significantly higher morning cortisol levels and a greater morning to afternoon cortisol decrease than younger children. However, age did not moderate the overall effects of early physical or sexual abuse and high symptoms. Thus, the findings are generally applicable for boys and girls, as well as for children across the age range studied.

The results of this investigation also contribute to our knowledge of pathways to depressive and internalizing symptomatology. For example, Kaplow and Widom (2007) reported that an earlier occurrence of maltreatment, defined as onset of maltreatment in the first 5 years of life, predicted more symptoms of anxiety and depression in adulthood. Our findings suggest that the results of Kaplow and Widom (2007) may have been even more compelling if they had been able to examine physical abuse, sexual abuse, and neglect separately. However, both the Kaplow and Widom (2007) and the current investigation raise the question as to why an early onset of maltreatment, and in our case abuse, is more predictive than a later onset in increasing risk for internalizing and depressive symptomatology and, in the context of these symptoms, dysregulated patterns of diurnal cortisol production. Early abuse may be more damaging to developing emotion and stress systems because it occurs during periods of rapid neurodevelopment (Cicchetti & Walker, 2001; Gunnar & Vazquez, 2006). Very young children also may be less able than older children to discern the cues predictive of an abusive attack, and this lack of predictability may engender chronic stress and hypervigilance to aggression in these youngsters, even when abusive events are not occurring (Rieder & Cicchetti, 1989;

Teisl & Cicchetti, 2008). School-aged physically abused children, on the other hand, have been shown to be highly sensitive to even very degraded signals of threat, which although potentially maladaptive in some contexts, may provide some relief from constant vigilance in the abusive home (Pollak, Cicchetti, & Klorman, 1998; Pollak & Sinha, 2002; Shackman, Shackman, & Pollak, 2007). In addition, the impact of early physical and sexual abuse on developing brain systems may be especially pernicious because it occurs during a period when the child is nearly wholly dependent on parents for survival. For the abused infant, toddler, or preschooler, a hypervigilant state of mind and chronic stress with respect to unpredictable parental attacks may shift neurobiological development onto pathways leading to depression and neuroendocrine dysregulation (Cicchetti & Rogosch, 2001a; Tarullo & Gunnar, 2006).

Another question pertains to why early neglect in the absence of physical or sexual abuse did not have a significant impact on either internalizing and depressive symptoms or HPA axis activity. While early neglect has been shown to have marked impacts on young children's social and cognitive competence (Pears & Fisher, 2005), neglect experiences may not be as fear and threat arousing as are the experiences of being physically attacked and hurt. Although it is not uncommon to find early neglect in the absence of abuse, it is rare to find early abuse in the absence of neglect, especially in low-income samples such as the one we studied. Nearly three-fourths of our early physical and sexual abuse sample also was neglected and emotionally maltreated. We do not know whether their neglect and emotional maltreatment contributed to their increased internalizing symptomatology and HPA dysregulation; however, it is possible that neglect made these children even more vulnerable to the fear and stress engendered by early abuse.

It also is conceivable that our findings may have been even more striking if we had included maltreated children who were residing in foster care. Generally children who experience early and extreme maltreatment are removed from their homes and placed in foster care. Because we excluded children with out-of-home placements, it is likely that we did not include the most extreme cases. This is not to say that the maltreated children in our sample experienced only mild abuse. Indeed, the median physical or sexual abuse severity score as coded using the MCS for the early abused children was 3 on the scale of 5 points, corresponding to incidents involving the infliction of marks on a child's face, serious bruises, or fondling a child for sexual gratification. Rather, by excluding children with out-of-home placements, we avoided confounding the impact of maltreatment with the stress of transitions in and out of foster care homes. These changes may be important in the sequelae of emotion and neuroendocrine dysregulation for many maltreated children (Gunnar & Fisher, 2006; Kaufman, 1991). Studying pre-school children who had primarily experienced neglect, Bruce and colleagues (2008) found reduced morning cortisol in the first months following a new foster placement. A year or more later, however, whether or not the morning cortisol recovered, continued to be dysregulated, or became more dysregulated, depended on whether the children were randomly assigned to regular foster care or to a foster care intervention condition which stabilized their early care (Fisher, Stoolmiller & Gunnar, 2007). Thus, the Bruce and colleagues (2008) study revealed the sensitivity of cortisol regulation to transient impacts of neglect and relationship disruption, while the present study indicated the long-term impacts of early physical and sexual abuse in the absence of the child being removed from the home.

Consistent with the results of Heim et al. (2000), the combination of high depressive symptoms and early physical or sexual abuse appeared to be critical to dysregulation of the HPA axis. Early physically and sexually abused children who were low on depressive and internalizing symptoms had cortisol patterns that were comparable to other maltreated and nonmaltreated children. However, whereas Heim and colleagues examined reactions of the HPA axis to psychosocial stressors and pharmacological challenges, we addressed only the children's typical daytime pattern of cortisol production. We do not know whether the high internalizing,

early abused children also would show larger or more prolonged cortisol responses to a stressor task similar to the adult women in the Heim et al. (2000) study. This awaits further research. It also will be important to conduct longitudinal studies in order to ascertain whether the dysregulated pattern of cortisol production noted among these high internalizing/early abused children presages on-going patterns of neurobiological and neuroendocrine dysregulation as the children develop.

There are many parallels between the neurobiological effects of early abuse and those of MDD (Heim et al., 2004, 2008). It is likely that prior investigations of individuals with MDD have included persons who had experienced early abuse (Bemporad & Romano, 1992; Heim et al., 2008). As such, many of the established neurobiological findings in depression may be due to the undetected presence of early physical or sexual abuse (Heim et al., 2004). In the absence of early abuse, depression may not be related to changes in the HPA system. Thus, there may be different neurobiological subtypes of depression. Accordingly, a history of early abuse, as well as the clinical presentation, onset, and course of depression should be incorporated into the formulation of potential etiological models of mood disorder (Heim et al., 2004, 2008). For example, because cortisol dysregulation rarely occurs in depressed children who did not experience abuse early in life (Feder et al., 2004), childhood depression without early physical or sexual abuse may be caused by different processes and have different neurobiological features than childhood depression with early abuse. Our findings that nonmaltreated children with high levels of depressive or internalizing symptomatology did not exhibit atypical cortisol regulation attest to this divergence. There may be biologically distinct subtypes of depression as a function of the presence or absence of abuse early in life (cf. Heim et al., 2004, 2008). Thus, not all individuals with depression exhibited cortisol dysregulation; we found it only for the early physically or sexually abused children with high internalizing and depressive symptomatology – a group that is more likely to have dysfunctions in early neurobiological development through the high allostatic load caused by abuse.

It is also important to note that not all of the children who experienced early abuse had high depressive or internalizing symptoms. Why is the HPA axis functioning of this group of early abused children better regulated than that of early abused and depressed children with the attenuated diurnal decrease in cortisol? It is likely that there are genes that increase the probability of depression and internalizing problems under conditions of high stress such as early physical or sexual abuse. For example, a number of investigations have demonstrated that the s/s and s/l gentoypes of the serotonin transporter gene linked promoter region (5-HTTLPR), in interaction with severe child maltreatment, are associated with increased risk for depression in children, adolescents, and adults (see, e.g., Caspi et al., 2003; Cicchetti, Rogosch, & Sturge-Apple, 2007; Kaufman et al., 2004). Conversely, the l/l genotype of the 5-HTTLPR may confer a protective function by decreasing the probability that individuals experiencing severe maltreatment will develop depression (Cicchetti et al., 2007; Kaufman et al., 2004). Consistent with calls for a multiple-levels-of-analysis approach to investigating the determinants of resilience (Charney, 2004: Cicchetti & Curtis, 2007; Curtis & Cicchetti, 2003), future research will need to include assessments of potential genetic dispositions to depression to discover whether individuals experiencing early physical and sexual abuse may have the genes that confer vulnerability or protection against depression (see also Bradley et al., 2008). Similarly, it will be important in subsequent investigations to examine the contribution that the corticotropin releasing hormone (CRH) genes and the glucocorticoid receptor (GR) genes play in individual differences in HPA axis activity, regulation, and reactivity.

There are several limitations in the design that deserve discussion. First, it is impossible to isolate completely the effects of physical and sexual abuse from those of neglect. The clinical reality is that there is an extremely high overlap and that neglect is almost a consistent

experience for these children, particularly in low-income samples. Thus, some caution is warranted in interpreting the meaning of our subtype findings. It may be, as we argued above, that abuse (physical or sexual) has features (e.g., provoke feelings of fear) that are particularly potent in activating stress and emotion pathways, and thus in dysregulating rapidly developing stress and emotion neurobiological systems. However, it could also be that abuse in the context of neglect is a more severe form of maltreatment, and thus that "severity" rather than particular features (e.g. fear provoking) produced the effects we observe. As abuse and neglect are nearly impossible to disentangle in human populations, it may prove useful to translate the findings from the current study back into animal models that compare early abuse, early abuse and early neglect, early neglect only, and later abuse and neglect.

Another limitation in the study was our lack of information about the time when the children awoke. Cortisol levels change markedly in the first hour after morning awakening and then gradually decrease over the remainder of the day. Levels are more closely related to the individual's wake-up time than to clock time. We chose not to measure wake-up time because we anticipated errors in child reporting of this measure, given the high level of disorganization in many of the families' homes. However, in future studies determining a valid method of obtaining individual wake up times would be helpful. Note, however, that in order for differences in wake-up time to have explained our results, high symptom children with physical or sexual abuse prior to age 5 would have to have woken up earlier than all the other children. Although it is possible, it seems unlikely that this was the case.

A third limitation was that we did not obtain puberty stage data on these children. Puberty is associated with increases in cortisol levels and reactivity to stressors (Gunnar et al., 2008; Stroud et al., 2008). Undoubtedly some of the older children in our study were entering puberty and this may have influenced their cortisol levels. In the absence of pubertal measures, we used age group (under age 10, age 10 and older) as a proxy measure, and as reported earlier, we did not find that age level moderated the effects of early abuse and high symptoms. Thus, we do not have evidence that pubertal development in the current sample had a major impact on the findings.

Finally, we cannot ascertain whether differences in the diurnal decrease in cortisol of the magnitude we observed will be predictive of future physical or mental health risks in children. There is increasing interest in the impact of chronic stress on patterns of diurnal HPA axis activity. Several studies of adult cohorts now show that "flattening" of the diurnal decrease in the same range as we observed predicts heightened risk of impairment in cardiovascular and immune system functioning (Heim et al., 2000; Raison & Miller, 2003). Thus it is conceivable that variations in the diurnal decrease in the range we report reflect mechanisms that have health consequences. Nonetheless, we cannot conclude that this is the case without longitudinal studies.

Our findings possess significant implications for children within the child welfare population, particularly with respect to the importance of early preventive interventions for children who have been abused or neglected even in the absence of clinical levels of depressive or internalizing symptomatology. In addition, intervention, timed as closely as possible to the early abusive experience, could be implemented to prevent the recidivism of maltreatment, to redirect the abused children onto a positive developmental trajectory, and to prevent the emergence of depression (Cicchetti, Rogosch, & Toth, 2006; Toth & Cicchetti, 1993). Because HPA axis dysregulation in early-abused children with depression may reflect a unique subtype of depression, differential responses to treatment modalities may emerge, and this is an area requiring further investigation. Given what we know about the effects of cortisol dysregulation on a wide array of neurobiological systems, as well as on physical health and cognitive processes, the results of this investigation also have significant public health implications.

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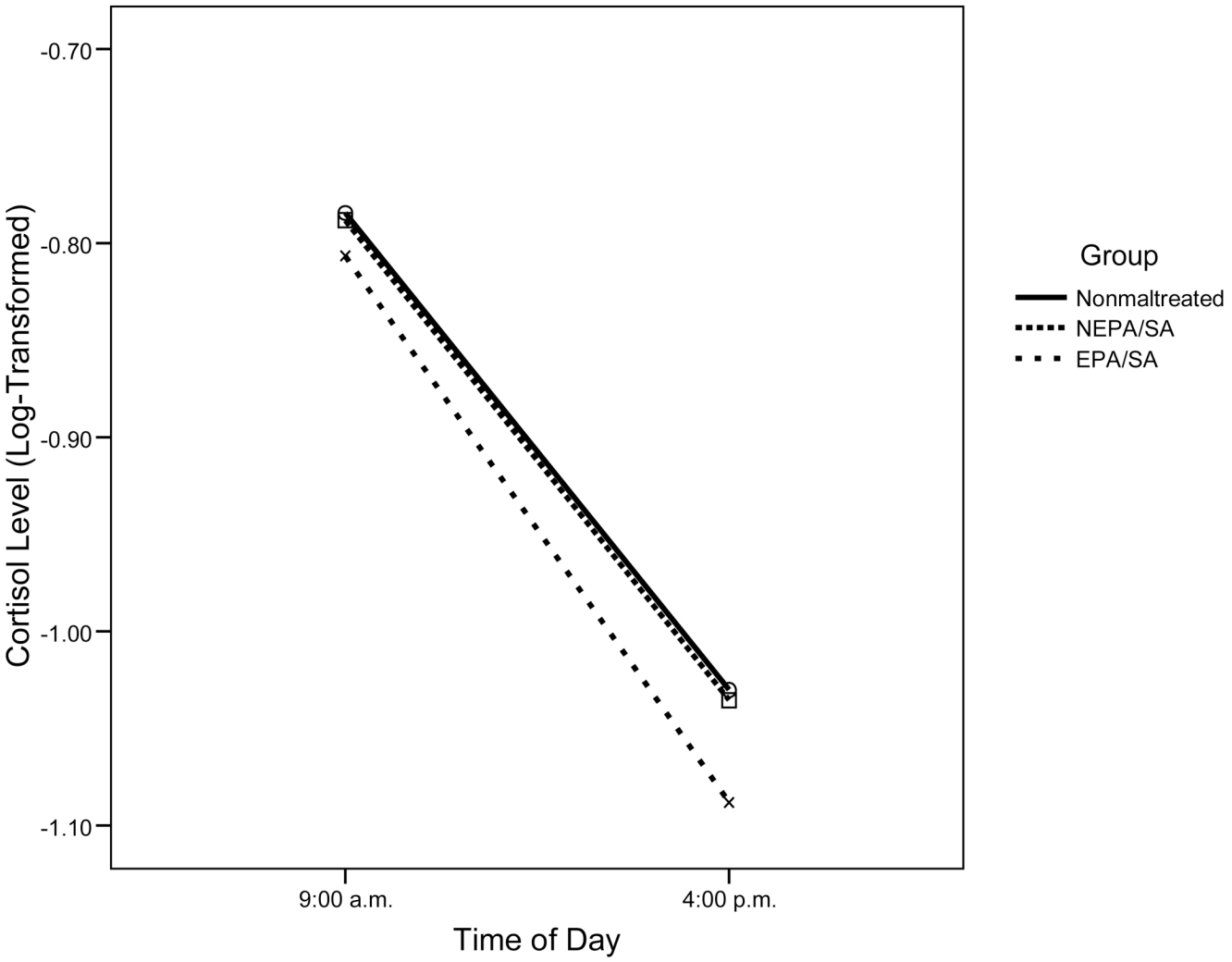

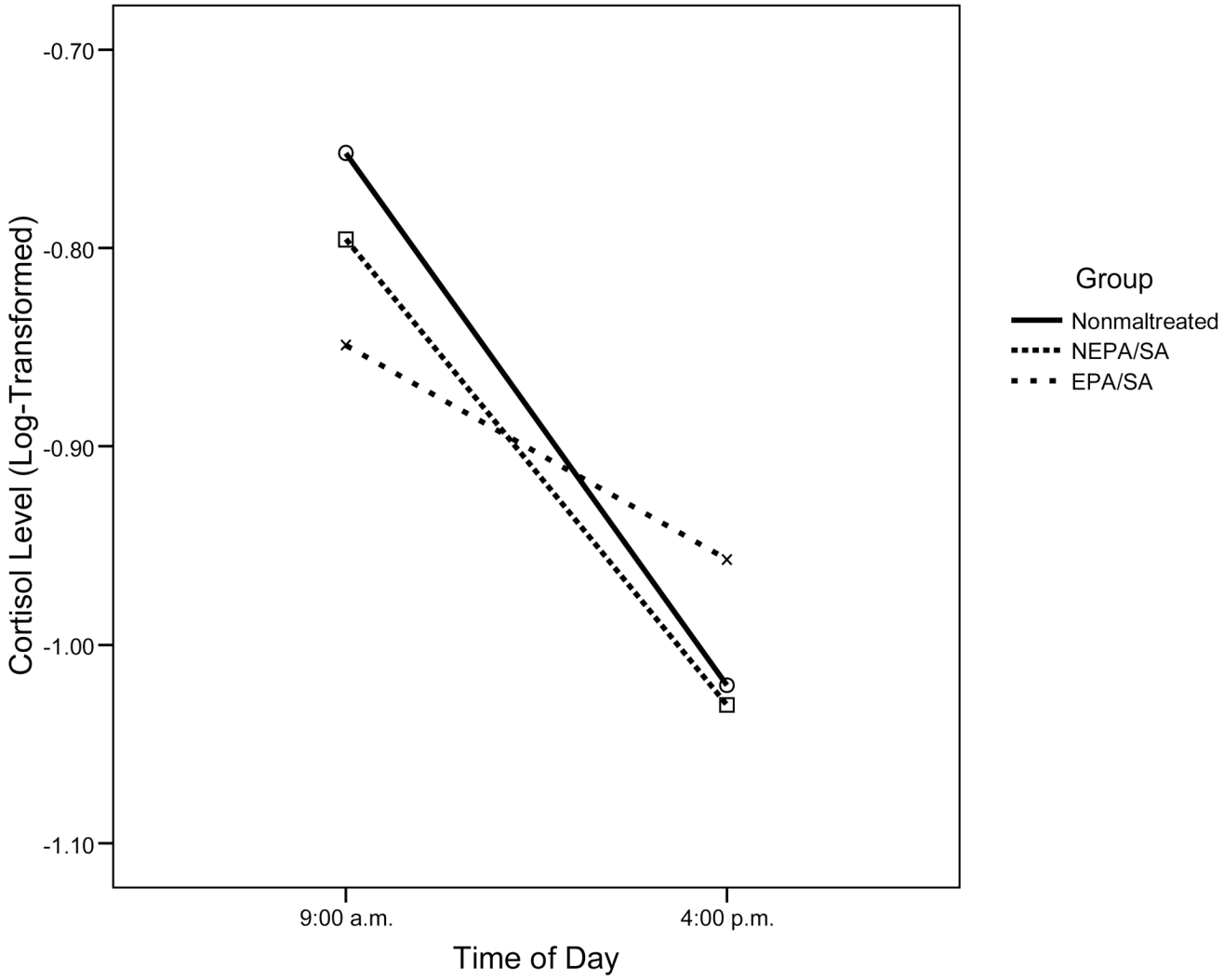

Figure 1.

Figure 1a. Children with *low* depressive and internalizing symptoms: Diurnal cortisol regulation in early abuse classification groups.

Note: Cortisol values are in μg/dl with log 10 transformation, adjusted for covariates. EPA/SA = early physical or sexual abuse, NEPA/SA = maltreated, but not early physical or sexual abuse.

Figure 1b. Children with *high* depressive or internalizing symptoms: Diurnal cortisol regulation in early abuse classification groups.

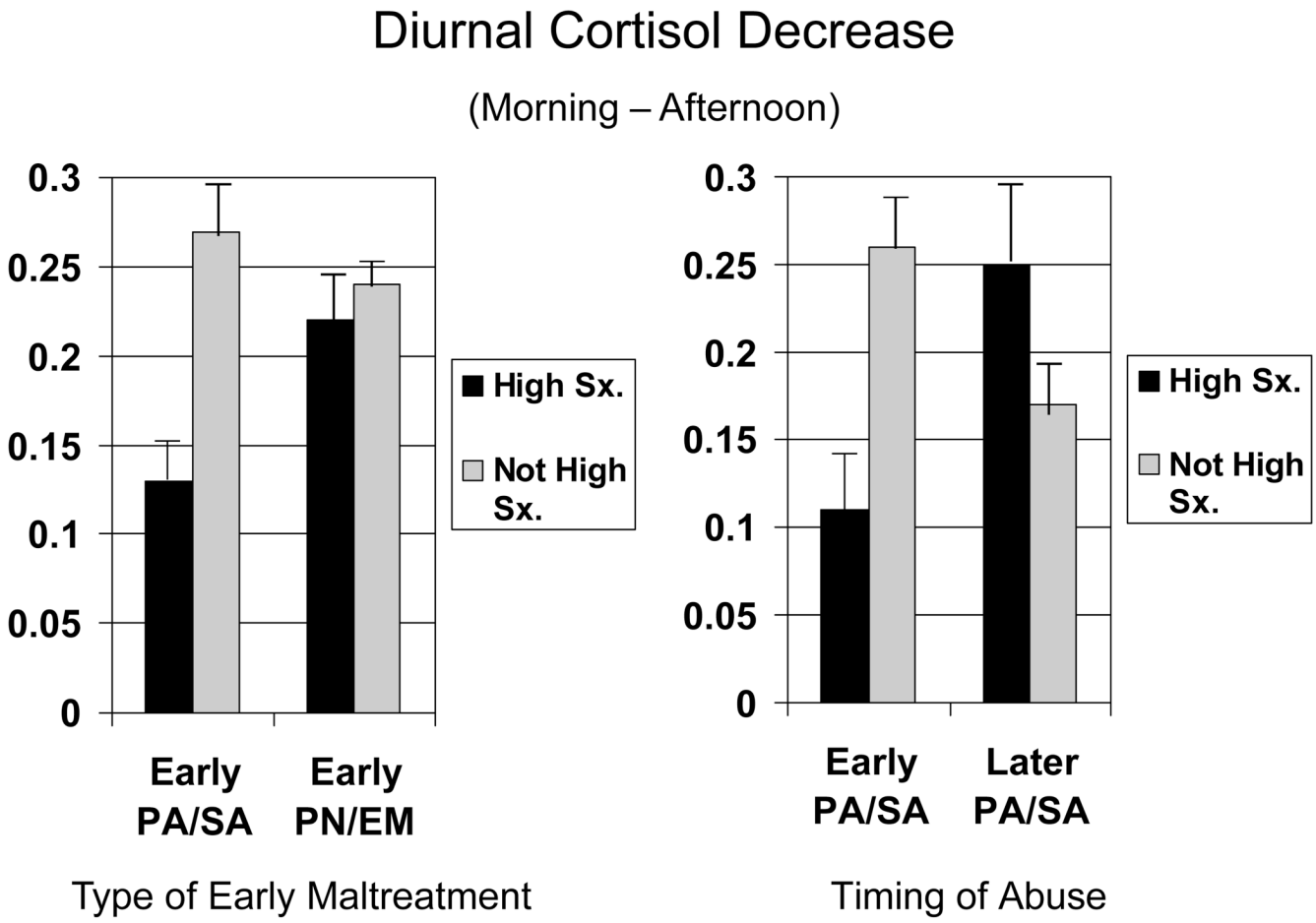

Figure 2.

Specificity of diurnal cortisol attenuation for early physical or sexual abuse and high symptoms: Comparisons with early neglect or emotional maltreatment and with later physical or sexual abuse

Note: Diurnal cortisol decrease values are adjusted for covariates; PA = physical abuse, SA = sexual abuse, PN = physical neglect, EM = emotional maltreatment, Sx. = internalizing and depression symptoms.

Table 1

Demographic Characteristics.

	Early Physical/ Sexual Abuse (***n*** **= 60)**	**Maltreated without Early Abuse** (***n*** **= 205)**	**Nonmaltreated** (***n*** **= 288)**
	M (*SD*) or %	*M* (*SD*) or %	*M* (*SD*) or %
Age	9.91 (1.91)	10.02 (1.85)	10.08 (1.89)
Gender (% male)	70.0 %	58.5 %	44.4 %
Race/Ethnicity			
African-American	50.0 %	64.9 %	65.3 %
Latino	23.3	11.7	17.4
Caucasian	25.0	21.0	14.9
Other	1.7	2.4	2.4
Family History of Welfare	96.7 %	95.0 %	96.0 %

Table 2

Comparison of Early Physical/Sexual Abuse Classification Groups on Depressive and Internalizing Symptoms and Cortisol.

	Early Physical/ Sexual Abuse (***n*** **= 60)**	**Maltreated without Early Abuse** (***n*** **= 205)**	**Nonmaltreated** (***n*** **= 288)**
	M (*SD*)	*M* (*SD*)	*M* (*SD*)
CDI total score	10.43 (7.84)	9.32 (7.75)	8.30 (.6.70)
TRF Internalizing T-score	$51.79\ (9.48)_{a}$	$48.91\ (7.99)_{b}$	$47.29\ (7.15)_{b}$
	% (*n*)	% (*n*)	% (*n*)
High CDI or TRF	$33.3\ (20)_{a}$	$21.5\ (44)_{ab}$	$14.9\ (43)_{b}$
	M (*SD*)	*M* (*SD*)	*M* (*SD*)
Average Morning Cortisol	−0.82 (.19)	−0.79 (.20)	−0.78 (.18)
Average Afternoon Cortisol	−1.04 (.16)	−1.03 (.17)	−1.03 (.15)

Notes: CDI = *Children's Depression Inventory*; TRF = *Teacher's Report Form*. Groups having the same subscript are not significantly different. Cortisol values are log transformed (log10).

Table 3

Repeated measures (morning and afternoon cortisol) analysis of covariance for high depressive or internalizing symptoms and early physical or sexual abuse classification groups.

Source	*df*	*F*	η^2	*p*
	Within subjects			
Time	1	.66	.001	.42
Sex X Time	1	5.87	.011	.016
Age X Time	1	28.28	.049	.000
Early Phys/Sexual Abuse X Time	2	2.36	.009	.095
High Symptoms X Time	1	6.46	.012	.011
Early Phys/Sexual Abuse X	2	6.07	.022	.002
High Symptoms X Time				
Error	545			