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The impact of child maltreatment and psychopathology on neuroendocrine functioning

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Abstract
Cortisol regulation was investigated in a sample of school-aged maltreated (n = 167) and demographically comparable low-income nonmaltreated (n = 204) boys and girls in the context of a day camp research program. The presence of clinical-level internalizing and clinical-level externalizing symptomatology was determined through adult report and child self report. Children who exhibited clinical-level internalizing problems only, clinical-level externalizing problems only, and comorbid clinical-level internalizing and externalizing problems were identified. Clinical-level cases were more prevalent among the maltreated children. Maltreated children with clinical-level internalizing problems were distinguished by higher morning, afternoon, and average daily cortisol levels across the week of camp attendance. In contrast, nonmaltreated boys with clinical-level externalizing problems emerged as distinct in terms of low levels of morning and average daily levels of cortisol. Maltreated children with comorbid clinical-level internalizing and externalizing problems were more likely not to show the expected diurnal decrease in cortisol. The findings are discussed in terms of the joint impact of maltreatment and different forms of psychopathology on neuroendocrine regulation.

Child maltreatment may represent the greatest failure of the caregiving environment to provide many of the expectable experiences that are necessary in order to facilitate normal developmental processes (Cicchetti & Lynch, 1995). Maltreating parents may be viewed as an aberration of the supportive, nurturant, sensitive, and protective adults that are expected by children in the evolutionary context of species-typical development (Belsky, 1984; Cicchetti & Lynch, 1995; Rogosch, Cicchetti, Shields, & Toth, 1995). In addition, numerous investigations reveal that maltreating families characteristically provide fewer opportunities for positive experiences and growth outside the family than are expected from an environment that can help to promote normal developmental outcomes (Belsky, 1993; Belsky & Vondra, 1989; Cerezo, 1997; Cicchetti & Lynch, 1995; Howes, Cicchetti, Toth, & Rogosch, 2000).

From the perspective of a dynamic systems theory view, reciprocal coactions among multiple levels of a developing system are thought to cause development to occur (Gottlieb, 1992). Maltreatment experiences provide serious challenges to the species-typical child–environment coactions that play important roles in the emergence and timing of normal developmental change (Aber & Cicchetti, 1984; Cicchetti & Lynch, 1995; Cicchetti & Toth, 2000; Trickett & McBride–Chang, 1995).

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In contrast to what is expected in response to an average expectable environment, the ecological, social, biological, and psychological conditions that are associated with maltreatment set in motion a probabilistic path of epigenesis for maltreated children characterized by an increased likelihood of failure and disruption in the successful resolution of major stage-salient issues of development, resulting in grave implications for functioning across the life span (Cicchetti, 1989; Cicchetti & Lynch, 1993, 1995; Cicchetti & Toth, 1995; Malinosky–Rummell & Hansen, 1993). These repeated developmental disruptions create a profile of relatively enduring vulnerability factors that increase the probability of the emergence of maladaptation and psychopathology as negative transactions between the child and the environment continue (Cicchetti, 1989; Cicchetti & Rizley, 1981).

The notion that an average expectable environment is required for species-typical development suggests that competent outcomes in maltreated children should be highly improbable due to wide-ranging disturbances in the maltreatment ecology (Cicchetti & Lynch, 1993; Cicchetti & Rizley, 1981). However, despite the fact that there is documented risk for maladaptation associated with maltreatment, the absence of an average expectable environment does not necessarily condemn all maltreated children to negative developmental outcomes later in life (Cicchetti & Rogosch, 1997; Cicchetti, Rogosch, Lynch, & Holt, 1993; McGloin & Widom, 2001).

Although not all maltreated children will develop maladaptation and psychopathology, nonetheless later disturbances in their functioning are likely to occur over the course of epigenesis. However, the diversity of developmental pathways that maltreated children take suggests that various forms of behavioral disturbance and psychopathology may emerge. In fact, maltreated children and adolescents have evidenced a wide range of disturbances and mental disorders (Cohen, Brown, & Smailes, 2001; Famularo, Kinscherff, & Fenton, 1992; Kashani, Shekim, Burk, & Beck, 1987). Specifically, child maltreatment has been linked with clinical levels of internalizing and externalizing symptomatology, as well as with higher rates of depression and anxiety disorders, conduct disorder and delinquency, and posttraumatic stress disorders (PTSD; Aber, Allen, Carlson, & Cicchetti, 1989; Allen & Tarnowski, 1989; De Bellis & Putnam, 1994; Famularo et al., 1992; Kaufman, 1991; Kaufman & Cicchetti, 1989; Kazdin, Moser, Colbus, & Bell, 1985; Pynoos, Steinberg, & Wraith, 1995; Shields & Cicchetti, 1998; Smith & Thornberry, 1995; Sternberg et al., 1993; Toth, Manly, & Cicchetti, 1992).

In addition to the burgeoning number of investigations on psychopathology in maltreated children that have occurred, work on the biological correlates and consequences of maltreatment has grown over the past decade (see, e.g., Bremner et al., 1997; DeBellis, Baum, et al., 1999; DeBellis, Keshavan, et al., 1999; DeBellis & Putnam, 1994; Hart, Gunnar, & Cicchetti, 1995, 1996; Ito, Teicher, Glod, & Ackerman, 1998; Pollak, Cicchetti, Klorman, & Brumaghim, 1997; Pollak, Klorman, Thatcher, & Cicchetti, 2001). Now that it is apparent that the mechanisms of neural plasticity cause the brain's anatomical differentiation to be dependent on stimulation from the environment (Cicchetti, in press; Cicchetti & Tucker, 1994), it is not surprising that maltreatment experiences impact on both psychological and biological processes. Although it has been demonstrated that gene expression alters social behavior (Young, Nilson, Waymore, MacGregor, & Insel, 1999), the study of child maltreatment, an "experiment of nature," can be utilized to illustrate how social experiences can alter gene expression, as well as brain structure, function, and organization (Bremner et al., 1997 Cicchetti, in press; Francis, DiOrio, Liu, & Meaney, 1999; McEwen, 2000; Meaney et al., 1996).

Although at present we possess only rudimentary knowledge about the molecular genetics and neurobiology of children growing up in maltreating homes, a number of investigations of the hypothalamic–pituitary–adrenal (HPA) axis have been conducted with maltreated children. Child abuse and neglect are stressful and threatening experiences that pose adaptational challenges, and the HPA axis is one of the physiological systems that has

evolved in mammals to help focus and sustain cognitive, emotional, behavioral, and metabolic activity in response to conditions of threat (Lopez, Akil, & Watson, 1999; Vazquez, 1998). Basal activity of this neuroendocrine system follows a circadian rhythm such that the highest levels appear around the time of awakening and then decline to low levels near the onset of sleep (Kirschbaum & Hellhammer, 1989). Basal levels of cortisol are essential to ensure normal brain growth and to support the metabolic activity necessary to sustain general functioning (McEwen, 1998).

The capacity to elevate cortisol in response to acute trauma is necessary for survival. Although brief elevations in glucocorticoids subsequent to acute stressors appear to improve the individual's ability to manage stressful experiences successfully, chronic hyperactivity of the HPA axis may bring about neuronal loss in the hippocampus, inhibit the process of neurogenesis, slow down the development of myelination, eventuate in abnormalities in synaptic pruning, and contribute to impairments in affective and cognitive functioning (DeBellis, Keshavan, et al., 1999; Gould, Tanapat, McEwen, Flugge, & Fuchs, 1998; Sapolsky, 1992; Todd, Swarzenski, Rossi, & Visconti, 1995). Moreover, hypocortisolism can also cause damage to neurons (Gunnar & Vazquez, 2001; Heim, Ehlert, & Hellhammer, 2000). Thus, some individuals who experience chronic stressors, such as ongoing physical abuse, may manifest reduced adrenocortical secretion, decreased adrenocortical reactivity, or enhanced negative feedback of the HPA axis (Cicchetti & Rogosch, 2001). Accordingly, the avoidance of both chronic glucocorticoid hypersecretion and hyposecretion is in the best interests of all individuals (Sapolsky, 1996).

A number of investigations have been conducted that indicate that there is altered HPA axis functioning in maltreated children. Disparate methodological approaches have been taken in these studies, including differences in the operationalization of the construct of maltreatment, diverse sampling strategies (e.g., officially indicated cases of maltreatment in community samples, children hospitalized in a psychiatric ward who had histories of maltreatment), varied experimental procedures and contexts (e.g., pharmacological challenge tests conducted in a laboratory or clinic as opposed to salivary cortisol assessments collected in a day camp), and differing techniques used to collect (e.g., use of Kool-Aid crystals vs. sugarless gum) and to assay saliva. Not unexpectedly, then, there is not a consensus among the results of these investigations. Impressively, however, all studies cohere in the conclusion that the HPA axis of maltreated children is often characterized by regulatory difficulties.

Neuroendocrine investigations of more heterogeneous groups of maltreated children have been conducted in complex social settings, such as a preschool environment and a summer day camp program. These contexts do not allow the same level of experimental control as that available in a scientific laboratory, though they do offer some advantages that accompany research conducted in more naturalistic settings.

One such investigation, conducted by Hart et al. (1995), examined the salivary cortisol concentrations and social behavior of maltreated and nonmaltreated children. The maltreated youngsters were studied while attending a therapeutic preschool for abused and neglected children, and the nonmaltreated children from a comparable social class background were studied while they were enrolled in a preschool that served economically disadvantaged families. Each child's cortisol values over a number of weeks were used to compute measures of basal activity and reactivity.

Hart and colleagues (1995) discovered that maltreated preschoolers exhibited less cortisol reactivity than did the comparison youngsters. Maltreated children also scored lower in social competence than did their nonmaltreated counterparts. Taken together, these results suggest a reduction in cortisol reactivity in maltreated children related to the impairment in social competence found in these children, whereas the nonmaltreated children exhibited higher cortisol reactivity and higher social competence.

Cicchetti and Rogosch (2001) also investigated cortisol regulation in a sample of

school-aged maltreated and demographically comparable nonmaltreated children in the context of a summer day camp research program. Group differences between maltreated and nonmaltreated children were not obtained for either average morning or average afternoon cortisol levels. However, significant variation was revealed based on the subtypes of maltreatment children had experienced. Maltreated children who had been both physically and sexually abused (as well as neglected or emotionally maltreated) exhibited substantial elevations in morning cortisol levels across the camp week, suggestive of hypercortisolism; children who manifested higher cortisol levels in both the morning and afternoon also were overrepresented in this multiple abuse group. Developmental timing of maltreatment did not account for these group differences, whereas severity of sexual abuse was implicated. In contrast to the multiple abuse group, a subgroup of physically abused children evidenced a marginally significant lower level of morning cortisol relative to nonmaltreated children. Moreover, these physically abused children displayed a significantly smaller decrease in cortisol level from morning to afternoon. Taken in tandem, these results suggest that the subgroup of physically abused children exhibited hypocortisolism.

Cicchetti and Rogosch (2001) concluded that high levels of cortisol do not uniformly characterize the day to day functioning of maltreated relative to nonmaltreated children. Rather, within the maltreatment group, differential patterns of cortisol regulation were observed. Moreover, the neglected and the emotionally maltreated subgroups of children did not appear to manifest abnormalities of the HPA axis, although it is conceivable that comparing these groups of children to a high risk comparison group of children from the lower-SES may have masked any HPA dysregulation that might exist (cf. Lupien, King, Meaney, & McEwen, 2001).

A number of investigations also have examined the neuroendocrine functioning of maltreated children who have been diagnosed with psychopathological disorders. These studies are instructive in that they also can provide insight into how the experience of child maltreatment affects the typical patterns of HPA axis regulation observed among nonmaltreated persons with mental disorders.

DeBellis, Baum, and colleagues (1999) examined the relations among trauma, psychiatric symptoms, and urinary free cortisol and catecholamine (i.e., epinephrine, norepinephrine, and dopamine) excretion in prepubertal maltreated children with PTSD. The maltreated children with PTSD were compared to a group of nontraumatized children with overanxious disorder (OAD) and to a normal control group of children.

The maltreated children with PTSD excreted significantly greater concentrations of urinary dopamine and norepinephrine over a 24-hr period than did the children with OAD or those in the normal control group. In addition, the maltreated children with PTSD excreted greater concentrations of 24-hr urinary free cortisol than did the normal control group children. Moreover, the maltreated children with PTSD excreted significantly greater concentrations of urinary epinephrine than did the children with OAD. Furthermore, urinary catecholamines and urinary free cortisol concentrations were found to be positively correlated with both the duration of the PTSD trauma and with the severity of the PTSD symptoms.

Interestingly, a substantial percentage of the maltreated children with PTSD in the DeBellis, Baum, et al. (1999) investigation had comorbid mood disorders. Although cortisol hypersecretion is not generally found in studies of children and adolescents with major depressive disorder or dysthymia (Dahl & Ryan, 1996), maltreated children with PTSD and comorbid depressive disorder manifested increased cortisol secretion. Thus, the findings of DeBellis, Baum, et al. (1999) provide further evidence that maltreated children with internalizing pathology manifest dysregulation of the HPA axis.

In the context of a winter day camp program, Kaufman (1991) examined salivary cortisol secretion in a group of maltreated children, a subgroup of whom had major depressive disorder (MDD), dysthymia (DD), or both (i.e., "double depression"). Although there was not a normal demographically comparable com-

parison group in this study, the maltreated children who met diagnostic criteria for MDD or DD were less likely than maltreated children who did not meet these criteria to display the expected diurnal rhythm decrease in afternoon cortisol secretion. Consistent with the findings of the DeBellis, Baum, et al. (1999) study reviewed previously, the results of Kaufman's (1991) study suggest that maltreated children who have a depressive disorder are characterized by dysregulation of the HPA axis.

In a related vein, Hart et al. (1996) examined the effects of maltreatment on adrenocortical functioning in a large sample of low-socioeconomic-status (low-SES) school-age maltreated and nonmaltreated children who were attending a summer day camp research program. These investigators discovered that depressed maltreated children displayed lower morning cortisol concentrations compared with nondepressed maltreated children. Moreover, consistent with the findings of Kaufman (1991), depressed maltreated children were more likely to show a rise rather than the expected diurnal decrease in cortisol from early morning to late afternoon, a finding suggestive of a regulatory dysfunction of the HPA axis. Additionally, there was no evidence that depressed, nonmaltreated children exhibited this aberration in diurnal cortisol activity.

Pharmacological challenges also have been utilized in investigations of HPA axis functioning in maltreated children with psychopathology. For example, augmented mean morning serial plasma cortisol levels have been found in sexually abused girls, implicating altered glucocorticoid functions in the HPA axis (Putnam et al., 1991). Similarly, in another investigation with sexually abused girls, the majority of whom had histories of severely depressed mood with suicidal behavior, an attenuated plasma adrenocorticotropic hormone (ACTH) response and normal levels of cortisol secretion were found in response to the ovine corticotropin-releasing hormone (CRH) stimulation test (DeBellis et al., 1994). These findings provide further evidence for dysregulation of the HPA axis in sexual abuse.

In another investigation of neuoendocrine functioning in maltreated children, Kaufman et al. (1997) administered a CRH challenge to a sample of depressed abused, depressed nonabused, and normal control children. Blood samples for ACTH and cortisol were drawn at 15-min intervals, with three occurring pre-CRH infusion and six occurring post-CRH infusion. Compared to the depressed nonabused and the normal control children, the depressed abused children exhibited significantly greater peak, total, and net ACTH secretion post-CRH infusion. However, there was variability within the depressed abused group of children. Specifically, the increased ACTH secretion post-CRH administration was obtained only in the depressed abused children who were experiencing ongoing chronic adversity (e.g., domestic violence, emotional maltreatment, poverty). In contrast, those depressed abused children whose trauma had occurred in the past did not manifest the increased ACTH and the HPA axis dysregulation characteristic of the chronic adversity subgroups of depressed abused children. Unfortunately, Kaufman and colleagues (1997) did not include an abused, nondepressed group of children in their study. The absence of this group prevents the ability to disentangle the effects of abuse and depression alone from those accompanying the interaction of depression and abuse.

In contrast with the growing number of investigations of the HPA axis in maltreated children with depression, there is a paucity of research on the neuroendocrine functioning of maltreated children with clinical levels of externalizing problems. In the largest study conducted to date, Hart and colleagues (1996) obtained a marginally significant effect that suggested that maltreated boys and girls with clinical levels of externalizing problems had lower cortisol levels averaged across the day than did maltreated children whose externalizing scores were below the clinical level. However, because the Hart et al. (1996) investigation did not examine the relation among maltreatment subtype, psychopathology, and cortisol, and because these researchers did not differentiate a separate group of maltreated and nonmaltreated children who had clinical levels of internalizing *and* externalizing symptomatology, it is important to conduct another

study that addresses the limitations of the Hart et al. (1996) study in order to ascertain whether maltreated children with externalizing disorders exhibit lower cortisol levels.

Several studies of HPA axis functioning have been conducted with children and adolescents with disruptive behavioral disorders (DBD) that provide insight into the hypocortisolism observed in some maltreated children (Cicchetti & Rogosch, 2001). For example, van Goozen, Matthys, Cohen–Kettenis, Buitelaar, and van Engeland (2000) investigated HPA axis functioning in a sample of male and female normal control children and children with disruptive behavior disorder in baseline, nonstressful, and stressful conditions. The cortisol levels of the children with DBD did not differ from those of the normal controls at baseline; however, the cortisol levels of the children with DBD were lower than were those of the control group children during conditions of interpersonal stress. The differences may have been more dramatic if the cortisol samples had not been obtained in the afternoon. Thus, lower levels of HPA activity appear to be associated with clinical levels of disruptive behaviors in school-age children. Due to the small number of girls in their study, van Goozen et al. (2000) could not investigate whether there were any gender differences in HPA axis functioning in children with DBD.

McBurnett, Lahey, Rathouz, and Loeber (2000) examined the relations between salivary cortisol levels and aggression in a group of school-aged boys who were followed up into adolescence. In contrast to children who had higher concentrations of cortisol at a single sampling time, children with low levels of cortisol across time evidenced persistence and early onset of aggression, as well as 3 times the number of aggressive nominations from their peers. Moreover, low cortisol at multiple time period assessments appeared to be more strongly related to aggression than was a low concentration of cortisol at one point in time.

Pajer, Gardner, Rubin, Perel, and Neal (2001) obtained several morning plasma samples for cortisol from a group of adolescent girls with conduct disorder and a normal control group. These investigators found that girls with conduct disorder had significantly lower cortisol levels than did girls in the normal control group at all morning sampling times. Additionally, girls with conduct disorder who had no other psychiatric disorders were more likely to show decreased cortisol levels than were girls with conduct disorder who had comorbid disorders. Although girls with the aggressive type of conduct disorder exhibited lower mean cortisol levels than did girls with nonaggressive conduct disorder, the difference was not statistically significant, in part due to the fact that only a small percentage of the adolescent girls with conduct disorder were nonaggressive. Thus, it remains to be answered whether low cortisol is a marker for aggressive conduct disorder in girls similarly to what it appears to be in boys.

Research Questions

The current investigation was conducted to expand upon prior studies that examined the relations among child maltreatment, psychopathology, and neuroendocrine functioning. Through enlisting the participation of a large representative sample of maltreated and nonmaltreated comparison children, a more thorough comparison of maltreated and nonmaltreated children, with and without different forms of psychopathology, and their adrenocortical functioning can be made.

We hypothesized that maltreated children would have higher rates of clinical-level internalizing and externalizing problems than would the demographically comparable groups of nonmaltreated children. Moreover, we expected that maltreated children would be more likely to exhibit comorbid clinical-level internalizing *and* externalizing problems than would nonmaltreated comparison children.

In addition, we predicted that maltreated children with clinical-level internalizing symptomatology would exhibit increased basal levels of cortisol compared to maltreated children who did not have clinical-level internalizing problems, as well as nonmaltreated children who had or did not have clinical-level internalizing problems. We also sought to determine if maltreated children with clinical-level internalizing problems would be less likely to

exhibit the expected decrease in cortisol over the course of the day. Furthermore, we hypothesized that the children with clinical-level externalizing symptomatology would manifest lower basal levels of cortisol than would children without externalizing problems. Additionally, we predicted that this result would be intensified for the maltreated externalizing cases. Moreover, because children with comorbid internalizing and externalizing clinical-level problems have not been distinguished in prior research on neuroendocrine functioning, and because of the hypothesized differential cortisol regulation patterns anticipated for children with internalizing as opposed to externalizing psychopathology, we sought to explore the effects of these comorbid problems on HPA axis regulation.

Finally, because most studies that have investigated maltreatment and adrenocortical functioning either have controlled for gender, employed small sample sizes that precluded testing for gender effects, or utilized samples of only boys or girls, we will explore gender as a possible moderator of the links between maltreatment, psychopathology, and cortisol regulation.

Method

Participants

The participants in this investigation included 371 children from an upstate New York urban setting who attended a day camp research program in the years 1997–2000. These children comprised 97.6% of the sample examined in Cicchetti and Rogosch (2001) for whom psychopathology data were available; the children with missing data did not differ from the larger group on any demographic indicators or cortisol variables.

The research camp program was designed for maltreated and nonmaltreated low-income disadvantaged children (Cicchetti & Manly, 1990). During each of these camp years, saliva samples were obtained from camp attendees. We chose to study all children who received their first cortisol assessment in camp during 1 year out of a consecutive 4-year period. In the camp, the children did not know what to expect from the adults and children they encountered and whom they had never previously met. An advantage of the naturalistic camp setting was that it permitted the collection of saliva from the children during uniform time periods. Saliva samples were obtained twice daily from the children at the same time in the morning (i.e., at 9:00 a.m., as soon as the children arrived at camp in a bus) and in the afternoon (i.e., at 4:00 p.m., shortly before being bused home at the end of the day). Thus, the morning samples were obtained during the peak period of the diurnal cortisol cycle, which occurs between 8:00 and 9:00 a.m. (Vinson, Whitehouse, & Hinson, 1998).

Children were on average 9.24 years of age (SD = 2.33); 225 of the children were boys, and 146 were girls. The majority of the children in the sample (80.8%) were of minority racial or ethnic backgrounds: 62.4% African American, 16.8% Latino American, 19.2% Euro-American, and 1.6% other racial–ethnic groups. The sample consisted of both maltreated (n = 167) and nonmaltreated (n = 204) children.

Parents of all maltreated and nonmaltreated children provided informed consent for their child's participation, as well as consent for examination of any Department of Social Services (DSS) records pertaining to the family. Children in the maltreatment group had been identified by DSS as having experienced child abuse or neglect. All existing DSS records for these families were screened and coded by raters, utilizing the Barnett, Manly, and Cicchetti (1993) nosological classification system for child maltreatment, described below.

The abuse and neglect experiences of the maltreated sample were diverse; 74.3% had been emotionally maltreated, 79.6% had been neglected, 37.1% had been physically abused, and 17.4% had been sexually abused. Consequently, the majority of maltreated children (68.9%) had experienced multiple subtypes of maltreatment, consistent with other samples of maltreated children and the nature of child maltreatment (Barnett, Manly, & Cicchetti, 1993; Cicchetti & Barnett, 1991). The multiplicity of subtypes experienced was particu-

larly true for children who had been physically abused or sexually abused. Only 4.8% of children experiencing physical abuse were solely physically abused, and only 3.4% of children experiencing sexual abuse were exclusively sexually abused.

Children were categorized into subtype groups based on the very high prevalence rates for neglect and emotional maltreatment relative to sexual and physical abuse, as well as the degree to which these forms of maltreatment violate cultural standards. First, all children who had been *sexually abused and physically abused* were identified (n = 16). Each of the children in this group also had experienced either neglect or emotional maltreatment in addition to sexual and physical abuse. In fact, 75% of the children in this group had experienced all four subtypes of child maltreatment. Next, all children who had been *sexually abused* but not physically abused were selected (n = 13); 92.3% of these children also had been neglected or emotionally maltreated. Children who were *physically abused* but not sexually abused were then identified (n = 46); 93.5% of these children had been neglected or emotionally maltreated. Children who had been *neglected* but not physically or sexually abused were then grouped (n = 72); 61.1% of these children also had been emotionally maltreated. Finally, the remaining children who had only experienced emotional maltreatment were categorized in the *emotionally maltreated* group (n = 20).

In order to obtain a demographically comparable comparison group, nonmaltreated children were recruited from families receiving public assistance. DSS record searches were conducted for these families to verify the absence of any child maltreatment. Families were excluded from the nonmaltreatment group if there was any family history of DSS involvement or if the family had received services to prevent foster care placement.

As was demonstrated in Cicchetti and Rogosch (2001), the maltreated and nonmaltreated samples were comparable on a number of demographic variables. For example, parents were currently married in 17.5% of the maltreating families and 20.7% of the nonmaltreating families. In 45.4% of the maltreating families and 44.9% of the nonmaltreating families, there was only one adult in the home, typically a single mother. The families of both groups tended to be large; 63.2% of the maltreating families and 61.6% of nonmaltreating families had three or more children. In terms of socioeconomic indicators, 65.1% of the mothers in the maltreating families had a high school education or less, compared to 60.1% of the mothers in nonmaltreating families. A history of receipt of public assistance was characteristic of both groups (i.e., 95.1% of maltreating and 90.9% of nonmaltreating families). Per capita income for the maltreating families (including welfare benefits) was $4,851, compared to $5,221 for the nonmaltreating families. All of these demographic indicators did not differ significantly between the maltreated and nonmaltreated groups. Thus, based on these demographic features, the sample as a whole was characterized by substantial disadvantage, with low SES, utilization of public assistance, large families, and frequent single parenthood.

Procedure

Each year of the research program, families were approached and asked if they would agree to have their child attend a week-long day camp and participate in the research. At the camp, the children participated in a variety of recreational activities in groups of six to eight same-age and same-sex peers. Half of the children in each group were maltreated; the other half were nonmaltreated. Each camp day lasted for 7 hr, providing 35 hr of interaction between children and adult camp counselors. In addition to participating in the camp recreational activities, the children also took part in a variety of research assessments (Cicchetti & Manly, 1990) and provided morning and afternoon saliva samples. At the end of the camp week, the camp counselors completed assessment measures on the individual children in their respective groups. The camp counselors were not aware of the maltreatment status of the various children in their group.

Measures

Salivary cortisol. Saliva samples were obtained daily at 9:00 a.m. and 4:00 p.m. Two

different collection procedures were used. In 1997 and 1998, children were given a small amount of Kool-Aid crystals to stimulate saliva flow. Then they mouthed a sterile cotton dental role to soak up the saliva. The wetted portion of the cotton was placed in a needleless syringe, and the saliva was expressed into a vial for storage at −80°C until it was shipped on dry ice to the University of Minnesota for analysis. The Ciba Corning Magic Cortisol kit was the assay procedure utilized, and assays were completed without knowledge of the children's maltreatment status. Samples were assayed in duplicate with all samples from the same subjects assayed within the same batch to prevent interassay variation from obscuring within-subject regulation patterns. The assay yields values down to 0.02 μg/dl. The returned values were examined for physiologically improbable values (i.e., > 4.0 μg/dl), and such values were not retained. Intra- and interassay coefficients of variation are 5.31 and 13.10.

Because of the potential for assay interference resulting from use of drink mix crystals as a saliva stimulant (Schwartz, Granger, Susman, Gunnar, & Laird, 1998), in 1999 and 2000 an alternate procedure was utilized. Children were given a piece of Trident original flavor sugarless gum to chew in order to stimulate saliva flow. Children then expurgated through a plastic straw directly into a vial, which was stored at −80°C until shipment on dry ice to Pennsylvania State University for analysis. Cortisol assays were conducted without awareness of the maltreatment status of participating children. The saliva samples were assayed in duplicate using a high-sensitivity enzyme immunoassay (Salimetrics, State College, PA). In each assay batch, analytical controls representing low and high cortisol levels were included. The test has a lower limit of sensitivity of .007 μg/dl, and average intraassay and interassay coefficients of variation of 4.13 and 8.89, respectively. Method accuracy, determined by spike recovery, and linearity, determined by serial dilution, are 105% and 95%. Values from matched serum and saliva samples show the expected strong linear relation ($r = .94$, $p < .0001$).

The distributions of cortisol assay results evidenced variability across years in mean μg/dl (1997: $M = .28$, $SD = .18$; 1998: $M = .41$, $SD = .26$; 1999: $M = .14$, $SD = .11$; and 2000: $M = .17$, $SD = .14$). Furthermore, the distributions of obtained values contained high skew and negative kurtosis. Accordingly, the data for each year were transformed using log10 to reduce skew and kurtosis. Then, within each camp year, all log10 transformed values were converted to Z scores. Because children were selected from different camp years based on the first time they provided saliva samples, the standardized scores allowed relative comparability in cortisol level across camp years (Granger, personal communication, April 27, 2000).

Maltreatment classification. The Maltreatment Classification System (MCS; Barnett et al., 1993) was used to delineate diverse features of child maltreatment that individual children had experienced. Cicchetti and Rizley (1981) argued that the phenomenology of child maltreatment is heterogeneous, involving different types and patterns of maltreatment with different etiologies, developmental sequelae, and response to treatment. Accordingly, Cicchetti and Barnett (1991) advocated the development of a nosological system to characterize the substantial variability in experience among maltreatment children. The MCS utilizes DSS records detailing investigations and findings regarding maltreatment occurrences in identified families. Rather than relying on official designations and case dispositions, the MCS codes all information available on a designated family from DSS records, making independent determinations of maltreatment experiences. In particular, the MCS codes all incidents that have been documented in DSS records and, based on operational criteria, designates the subtypes of maltreatment individual children have experienced (i.e., emotional maltreatment, neglect, physical abuse, sexual abuse), the severity of each type of maltreatment, the frequency of occurrence, the developmental periods in which maltreatment occurred, and the perpetrators of maltreatment. Coding of DSS records was conducted by trained research assistants, doctoral students, and PhD-level psychologists; adequate reliability has been obtained (weighted

kappas ranging from .86 to .98; Manly, Kim, Rogosch, & Cicchetti, 2001). Other investigators have utilized the MCS and have demonstrated the system to be reliable and valid in classifying maltreatment incidents (Bolger, Patterson, & Kupersmidt, 1998; Manly, Cicchetti, & Barnett, 1994; Smith & Thornberry, 1995).

In terms of the operationalization of subtypes of maltreatment, *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 in terms of increasing severity may involve belittling, ridiculing the child, using fear and intimidation, blaming the child inappropriately, extreme negativity and hostility, exposure to severe marital violence, abandoning the child, confining the child in an enclosed space, and suicidal and homicidal threats. *Neglect* involves failure to provide for children's basic physical needs (i.e., for adequate food, clothing, shelter, medical treatment), lack of supervision (leaving child without adult supervision, exposure to firearms, leaving child in the care of dangerous caregivers), or moral–legal or educational neglect (e.g., exposing the child to criminal activity, failing to send a child to school). *Physical abuse* involves the infliction of physical injury on a child other than by accidental means (e.g., beating the child, causing bruises, welts, broken bones, burns, choking). Injuries may range from minor and temporary to permanently disfiguring. Finally, *sexual abuse* involves any attempted or actual sexual contact between a child and caregiver for purposes of the caregiver's sexual satisfaction or financial benefit. Acts vary from exposure to pornographic material or adult sexual activity to sexual touching and fondling, attempted penetration, forced intercourse, and prostitution of the child.

Internalizing and externalizing psychopathology. Two measures were used to assess clinical levels of psychopathology in the participants. These measures included the *Teacher Report Form of the Child Behavior Checklist* (TRF; Achenbach, 1991) and the child-report *Children's Depression Inventory* (CDI; Kovacs, 1985).

TRF. After observing and interacting with children in their respective groups over the course of the camp week, camp counselors completed the TRF on individual children in their group. The camp counselors were unaware of the maltreatment status of the individual children in their group, as well as the research hypotheses. The TRF is a widely used and validated instrument to assess symptomatology by teachers. In the present study, because counselors are able to observe similar behavior to that of teachers, the camp counselors' ratings were used to provide an analogous assessment of child functioning by an adult external to the family. The TRF contains 118 items that are rated for frequency that assess two broadband dimensions of child psychopathology, including externalizing behavior problems (delinquent behavior, aggressiveness) and internalizing behavior problems (withdrawal, somatic complaints, anxiety–depression). Interrater reliabilities among three raters for each child (average alphas) for multiple years of camp have been reported to average .84 for externalizing and .76 for internalizing (Cicchetti & Rogosch, 1997). The counselors' scores for each child were averaged to obtain individual children's scores raw and *T* scores for the externalizing and internalizing dimensions. Given the interest in clinical levels of psychopathology, children were categorized as to whether they met threshold criteria for clinical levels of psychopathology for the internalizing and the externalizing dimensions. Achenbach's (1991) recommended clinical cut point of a *T* score of 60 or greater was used to designate clinical-level problems.

CDI. Because adults may be less sensitive to internalizing difficulties children are experiencing as compared to more overt behavioral problems (Achenbach, McConaughy, & Howell, 1987), children's own perspectives on their internalizing distress also were assessed with the CDI for children age 7 years and older. The CDI is a self-report questionnaire widely used to assess depressive symptomatology in school-age children. For each item, children chose from among three option

statements, depicting increasing levels of symptoms of depression experienced in the past 2 weeks. Reliability and validity data are reported by Saylor, Finch, Spirito, and Bennett (1984) and Kovacs (1992). A total CDI score of 19, as recommended by Kovacs (1992), was used as the clinical cut point to categorize children as exhibiting clinical-level depressive symptomatology.

Defining clinical-level case groups. The clinical cutpoints from the TRF and CDI were used to categorize children into clinical case groups. Children were identified as exhibiting clinical-level internalizing only psychopathology if they had clinical-level scores on the TRF internalizing scale or the CDI and did not exhibit clinical-level problems on the TRF externalizing scale (n = 38). A clinical-level externalizing only psychopathology group was defined as children who had clinical-level scores on the TRF externalizing scale but did not exhibit clinical-level symptoms on the TRF internalizing scale or the CDI (n = 65). A third group was identified who exhibited clinical levels of both internalizing and externalizing psychopathology (n = 31). These children had clinical-level scores on the TRF externalizing scale, as well as clinical-level scores on the TRF internalizing scale or clinical-level CDI scores. Finally, children who did not exhibit clinical-level difficulties on the CDI and the TRF internalizing and externalizing scales were designated as belonging to the no clinical problem group (n = 237).

Results

Maltreatment and clinically significant behavior problems

The rate of clinically elevated behavior problems was contrasted between the maltreated and nonmaltreated groups. Overall, maltreated children evidenced substantially higher rates of clinical case-level disturbance (49.7%), as compared to nonmaltreated children (25.1%), χ^2 (3) = 38.25, $p < .0001$. Although the rate of clinical-level internalizing problems was comparable for maltreated (9.6%) and nonmaltreated children (10.8%), maltreated children exhibited higher rates of clinical-level externalizing problems (24.0%) and comorbid clinical-level internalizing and externalizing problems (16.2%) than nonmaltreated children (12.3% and 2.0%, for externalizing and for comorbid internalizing and externalizing problems, respectively). The rate of comorbid clinical-level internalizing and externalizing problems was particularly low among nonmaltreated children. When children with any clinical-level internalizing problems were examined (the internalizing and comorbid internalizing and externalizing groups), the majority of maltreated children with clinically significant internalizing problems was more likely to evidence clinical-level externalizing difficulties also (62.8%), compared with 15.4% for nonmaltreated children, χ^2 (1) = 14.72, $p < .0001$. Thus, maltreatment was related to significantly higher rates of diverse forms of clinically significant psychopathology.

Within the maltreatment group, subtype group membership was not significantly differentially related to exhibiting a specific form of clinical-level behavior problems, χ^2 (14) = 14.47, *ns*. Children exhibiting each form of elevated psychopathology were found in all maltreatment subtype groups, and no form of disturbance predominated in a specific subtype group.

Cortisol regulation, maltreatment, and clinically significant psychopathology

To examine patterns of cortisol regulation in relation to maltreatment and manifestations of clinical case-level psychopathology, initially a repeated measures analysis of variance (ANOVA) was conducted, with maltreatment status, gender, and clinical case group membership as independent variables. Morning and afternoon mean cortisol levels served as the dependent variables. The analysis resulted in a significant within-subjects effect for gender, F (1, 355) = 14.07, $p < .001$, a marginal maltreatment by gender interaction, F (1, 355) = 2.98, $p = .085$, and a maltreatment by gender by case membership group interaction, F (3, 355) = 5.46, $p < .001$. Thus, this analysis revealed that complex relations were operating across maltreatment, gender, and case groups

in terms of differential patterns of cortisol regulation. However, this analysis was compromised by the small size of the comorbid internalizing and externalizing group in the nonmaltreated group (n = 4). Moreover, one girl in this group evidenced very high cortisol levels and appeared to be an influential outlier. Accordingly, the repeated measures analysis was conducted again, excluding the children with both internalizing and externalizing clinical-level problems from the analysis.

The new repeated measures ANOVA again revealed a significant within-subjects main effect for gender, F (1, 328) = 8.061, $p < .005$, as well as a significant within-subjects interaction of maltreatment, gender, and case membership group, F (2, 328) = 5.62, $p <$ .004). Moreover, significant between-subjects effects also were obtained. Significant main effects were found for maltreatment status, F (1, 328) = 4.78, p = .029, and clinical case group, F (2, 328) = 4.10, p = .017. Although the maltreatment by case group interaction effect was marginally significant, F (2, 328) = 2.49, p = .085, the three-way interaction of maltreatment by gender by case group, F (2, 328) = 3.39), p = .035, was significant. Thus, this analysis indicated that complex relations existed for cortisol regulation, even with the exclusion of children with comorbid clinical-level internalizing and externalizing problems. Subsequent analyses were undertaken to delimit how these effects were operating.

First, the clinical case groups (none, internalizing only, and externalizing only) were examined for differences in morning and afternoon cortisol levels. In terms of morning cortisol levels, an ANOVA revealed a significant effect for group, F (2, 337) = 3.10, p = .046. Tukey HSD post hoc tests at the .05 level indicated that the clinical-level internalizing group (M = .51, SD = .82) exhibited higher morning cortisol than the clinical-level externalizing group (M = .18, SD = .70). The children with no case level problems (M = .33, SD = .62) exhibited morning cortisol levels between the internalizing and the externalizing clinical-level groups, although they did not differ significantly from either group. In contrast, no significant group differences were obtained for afternoon levels of cortisol, F (2, 337) = .98, *ns*, or for average day level of cortisol, F (2, 337) = 2.68, *ns*. Thus, children with clinical-level internalizing problems exhibited relatively higher cortisol levels in the morning, contrasting with children with clinical-level externalizing problems, who exhibited relatively lower morning cortisol.

Given the maltreatment by gender by clinical case group within-subjects and between subjects interactions, the clinical case group effect was examined further to elucidate different patterns of cortisol regulation exhibited by nonmaltreated and maltreated boys and girls with follow-up ANOVAs. For morning cortisol levels, significant differences were found for gender, F (1, 328) = 5.317, p = .022, clinical case group, F (2, 328) = 3.60, p = .028, and the three-way interaction of maltreatment by gender by case group, F (2, 328) = 7.95, $p < .001$. Group patterns are depicted in Figure 1. Less variation was observed for afternoon cortisol levels (see Figure 2). The only effect to approach significance was an interaction of maltreatment and clinical case group, F (2, 328) = 2.52, p = .08. Finally, Figure 3 depicts differences for average daily cortisol levels. The results of this ANOVA for average daily cortisol yielded a significant main effect for clinical case group, F (2, 328) = 3.06, p = .048, a maltreatment by case group interaction, F (2, 328) = 3.06, p = .048, and a maltreatment by gender by case group three-way interaction, F (2, 328) = 3.386, p = .035.

Inspection of Figure 1 suggests greater uniformity among nonmaltreated children across the gender–case groups in morning cortisol level, with the exception of externalizing boys. In contrast, more variability is observed for the gender–case groups among the maltreated children. A similar pattern of greater variability among subgroups of maltreated children is seen in Figure 2 for afternoon cortisol and in Figure 3 for average daily cortisol. Examination of these subgroup differences will be subsequently organized around the clinical-level internalizing and the externalizing case groups.

Clinical case-level internalizing problems

To examine the interaction effects further with a focus on cases exhibiting clinically sig-

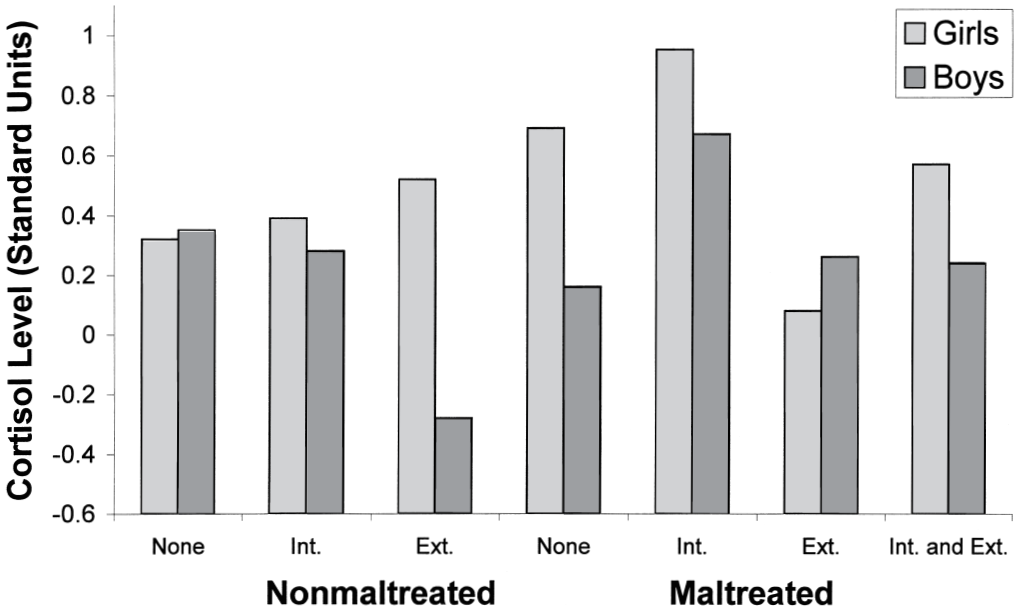

Figure 1. Morning cortisol levels for maltreated and nonmaltreated boys and girls within different clinical case groups. Int., internalizing case only; Ext., externalizing case only; Int. and Ext., internalizing and externalizing case.

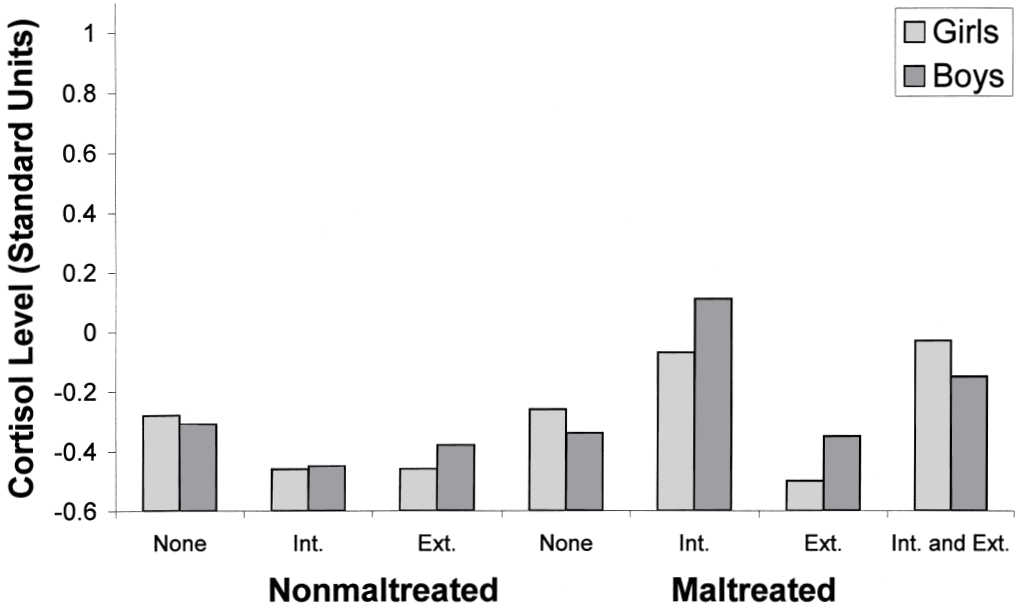

Figure 2. Afternoon cortisol levels for maltreated and nonmaltreated boys and girls within different clinical case groups. Int., internalizing case only; Ext., externalizing case only; Int. and Ext., internalizing and externalizing case.

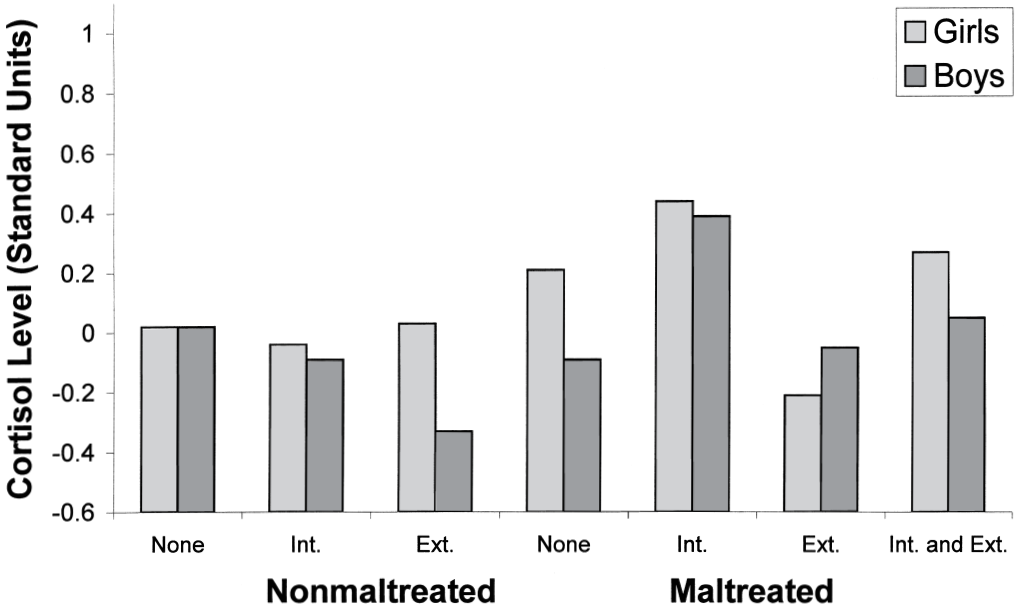

Figure 3. Average daily cortisol levels for maltreated and nonmaltreated boys and girls within different clinical case groups. Int., internalizing case only; Ext., externalizing case only; Int. and Ext., internalizing and externalizing case.

Table 1. *Cortisol levels, child maltreatment, and case-level internalizing problems*

	Nonmaltreatment Internalizing Case				Maltreatment Internalizing Case				
	Absence (1)		Presence (2)		Absence (3)		Presence (4)		
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	Contrasts
Morning cortisol	.31	.64	.33	.66	.28	.64	.75	.96	4 > 1, 2, 3
Afternoon cortisol	−.31	.61	−.45	.60	−.34	.65	.05	.78	4 > 2
Average daily cortisol	.00	.51	−.06	.54	−.03	.54	.40	.82	4 > 1, 2, 3

nificant internalizing problems, ANOVAs were conducted with presence or absence of clinical case-level internalizing problems, maltreatment, and gender as independent variables. For morning cortisol levels, a significant main effect for gender was obtained, F (1, 332) = 4.82, p = .029, as well as a significant maltreatment by internalizing clinical case-level interaction, F (1, 332) = 3.87, p = .05. The three-way interaction was not significant. Post hoc Tukey HSD tests were used to examine the maltreatment by internalizing case-level interaction in more detail. As shown in Table 1, maltreated children with clinical-level internalizing problems had significantly higher morning cortisol (M = .75, SD = .96) than each of the other groups: nonmaltreated children with clinical-level internalizing problems (M = .33, SD = .66), maltreated children without internalizing problems (M = .28, SD = .64), and nonmaltreated children without internalizing problems (M = .31, SD = .64). These three latter groups did not differ from each other. Thus, maltreated children with clinically sig-

nificant internalizing problems were differentiated by their high elevations of morning cortisol relative to the other maltreated and nonmaltreated children.

Similar findings were obtained for afternoon cortisol levels. An ANOVA with presence or absence of significant internalizing problems, maltreatment, and gender yielded a similar significant interaction of maltreatment and internalizing case group membership, F (1, 332) = 5.92, p = .016. Post hoc tests revealed that the afternoon cortisol levels of the maltreated children with clinical-level internalizing problems (M = .05, SD = .78) were significantly higher than those afternoon cortisol levels for the nonmaltreated children with clinical-level internalizing problems (M = −.45, SD = .60). No other group differences were found.

Average daily cortisol also was examined and likewise distinguished the maltreated children with clinical-level internalizing problems. The ANOVA resulted in a significant maltreatment by internalizing clinical case interaction, F (1, 332) = 6.60, p = .011. Post hoc Tukey HSD tests also indicated that the maltreated children with clinical-level internalizing problems (M = .40, SD = .82) differed significantly from each of the other groups: nonmaltreated children with clinical-level internalizing problems (M = −.06, SD = .54), maltreated children who did not have internalizing problems (M = −.03, SD = .54), and nonmaltreated children who did not have internalizing problems (M = .00, SD = .51).

Thus, across these three measures—morning cortisol level, afternoon cortisol level, and average daily cortisol level—maltreated children with clinical-level internalizing problems evidenced significant cortisol elevations. Gender did not appear to play an influential role in these results.

Given the within-subjects effects of the repeated measures ANOVA for morning and afternoon cortisol levels, diurnal variation was further investigated in a follow-up repeated measures ANOVA, with the independent variables of maltreatment, gender, and presence or absence of clinical-level internalizing problems. No significant within-subjects effects were obtained. Thus, there was no indication that internalizing problems in maltreated children were related to a differential pattern of morning to afternoon decrease in cortisol level.

Finally, given the distinguishing features of high levels of morning and afternoon cortisol for the group of maltreated children with internalizing problems, we examined how many children in the sample evidenced cortisol levels in the top quartile of the morning and of the afternoon cortisol distributions. Across the children, 4.7% evidenced these high cortisol levels in both the morning and the afternoon. High morning and afternoon cortisol was found more often among children having clinical case level internalizing problems (13.2%), χ^2 (1) = 6.82, p = .009, as compared to children who did not have internalizing problems (3.6%). Although this pattern was not found among nonmaltreated children, the pattern was significant for maltreated children, χ^2 (1) = 8.68, p = .003; 25% of maltreated children with internalizing problems had top quartile level cortisol in both the morning and afternoon, compared to 4.8% of maltreated children without internalizing problems.

Clinical case-level externalizing problems

Children with clinically elevated externalizing problems also were examined in more detail, contrasting them with children who did not have clinical-level externalizing problems. In a series of ANOVAs, with maltreatment, gender, and presence or absence of clinical-level externalizing problems as independent variables, differences in morning cortisol, afternoon cortisol level, and average daily cortisol levels were examined.

For morning cortisol levels, a significant maltreatment by gender by clinical-level externalizing group interaction was obtained, F (1, 332) = 15.25, $p < .001$. To explore this interaction effect, post hoc Tukey tests indicated that nonmaltreated boys with clinical case-level externalizing problems dominated this interaction effect (see Table 2). Morning cortisol levels of the nonmaltreated boys with clinical-level externalizing problems (M = −.28, SD = .81) were significantly lower than

Table 2. *Cortisol levels, child maltreatment, gender, and case-level externalizing problems*

	Nonmaltreated Externalizing Case				Maltreated Externalizing Case				
	Absence		Presence		Absence		Presence		
	Girls (1)	Boys (2)	Girls (3)	Boys (4)	Girls (5)	Boys (6)	Girls (7)	Boys (8)	Contrasts
Morning	.33	.34	.52	−.28	.73	.24	.08	.26	4 < 1, 2, 3, 5
	(.56)	(.67)	(44)	(.81)	(.75)	(.63)	(.61)	(.70)	7 < 5
Afternoon	−.30	−.32	−.46	−.38	−.23	−.27	−.50	−.35	
	(.54)	(.69)	(.46)	(.59)	(.67)	(.72)	(.58)	(.64)	
Average daily	.01	.01	.03	−.33	.25	−.02	−.21	−.05	4 < 5
	(.45)	(.56)	(.36)	(.56)	(.56)	(.60)	(.53)	(.57)	

Note: Values in columns numbered 1–8 are means with standard deviations in parentheses.

all other nonmaltreatment groups, including nonmaltreated boys without externalizing problems (M = .34, *SD* = .67), nonmaltreated girls with clinical-level externalizing problems (*M* = .52, *SD* = .44), and nonmaltreated girls who did not have externalizing problems (*M* = .33, *SD* = .56). The nonmaltreated boys with clinical-level externalizing problems also were significantly lower than maltreated girls who did not have externalizing problems (*M* = .73, *SD* = .75).

In addition to the contrasts involving nonmaltreated boys with externalizing problems, other significant cortisol differences were found among maltreated girls. Maltreated girls with clinical-level externalizing problems had significantly lower morning cortisol (*M* = .08, *SD* = .61) than maltreated girls who did not have externalizing problems (*M* = .73, *SD* = .75). Maltreated boys with clinical-level externalizing problems (*M* = .26, *SD* = .70) and maltreated boys without externalizing problems (*M* = .24, *SD* = .63) did not differ from any group.

No significant effects were found in the ANOVA for afternoon cortisol levels. However, for average daily cortisol level a significant three-way interaction effect for maltreatment, gender, and externalizing problem group was obtained, F (1, 332) = 6.15, p = .014. Post hoc tests indicated that nonmaltreated boys with clinical-level externalizing problems (*M* = −.33, *SD* = .56) had the lowest average daily cortisol levels and were significantly lower than maltreated girls who did not have externalizing problems (*M* = .25, *SD* = .56). All other groups ranged between these two extremes.

Diurnal change from morning to afternoon cortisol levels was further examined in a repeated measures ANOVA, with maltreatment, gender, and externalizing group as independent variables. A significant within-subjects three-way interaction effect was obtained, F (1, 332) = 11.33, $p < .001$. This three-way interaction effect was driven by the nonmaltreated boys with clinical-level externalizing problems. Given their very low morning and afternoon cortisol levels, their diurnal change was relatively flat, whereas all other groups evidenced the expected drop in cortisol levels from morning to afternoon.

A pattern of atypical cortisol regulation also was investigated in relation to clinically elevated externalizing problems. Specifically, children who had morning cortisol levels in the lowest quartile and who also did not decrease or who increased in the afternoon were identified. This group consisted of 6.5% of the sample. However, this pattern was significantly more prominent, χ^2 (1) = 7.09, p = .008, among children with clinical-level externalizing problems (13.8%), as compared to those without externalizing problems (4.9%). The percentage of maltreated children with clinical-level externalizing problems display-

ing this pattern (15.0%) was comparable to that of nonmaltreated children (12.0%).

Maltreated children with comorbid clinical-level internalizing and externalizing problems

Although it was not feasible to include the children with comorbid clinical-level internalizing and externalizing problems in the multivariate analyses because of the very small number of nonmaltreated children with both types of psychopathology, the maltreated children with comorbid clinical-level internalizing and externalizing problems were included in the figures for comparison purposes. Maltreated girls with comorbid clinical-level internalizing and externalizing problems appeared to have morning cortisol levels between that of girls with clinical-level internalizing problems and girls with clinical-level externalizing problems (see Figure 1). In contrast, maltreated boys with comorbid problems appeared very comparable to maltreated boys with clinical-level externalizing problems and maltreated boys with no clinical problems.

Another pattern of diurnal cortisol variation was investigated in relation to maltreated children with both internalizing and externalizing clinical-level problems. Specifically, children who did not evidence a decrease from morning to afternoon cortisol levels were identified. Across the entire sample, 19.4% of children showed this pattern, and the pattern was equally prevalent among nonmaltreated (19.4%) and maltreated (19.2%) children. Within the maltreated group, children who had comorbid clinical-level internalizing and externalizing psychopathology versus those who did not were grouped. Lack of a decrease in afternoon cortisol was more prominent, χ^2 (1) = 4.17, p = .04, among maltreated children with comorbid clinical-level internalizing and externalizing problems (33.3%) than it was among other maltreated children who did not have comorbid internalizing and externalizing psychopathology (16.4%).

Discussion

In the present study, we investigated whether a large representative sample of maltreated children from low socioeconomic backgrounds would exhibit a greater percentage of internalizing and externalizing clinical-level problems than would a demographically comparable nonmaltreated comparison sample of children. Because negative social experiences such as child maltreatment have been demonstrated to impact adversely on neuroendocrine functioning (Cicchetti & Rogosch, 2001; DeBellis, Baum, et al., 1999; Hart et al., 1996; Kaufman et al., 1997), we wished to determine whether homogeneous groups of maltreated children with clinical levels of problems would manifest differential patterns of adrenocortical regulation. Since we possess evidence that individuals with comorbid forms of psychopathology often have worse long-term prognoses than do persons with singular disorders (Birmaher, Ryan, Williamson, Brent, & Kaufman, 1996; Harrington, Rutter, & Fombonne, 1996; Hinshaw, Lahey, & Hart, 1993; Kovacs, 1996), it is conceivable that comorbid clinical levels of problems in maltreated children may reveal important information about these children's neuroendocrine functioning. Prior research in our laboratory has discovered that not all maltreated children exhibit the same pattern of neuroendocrine regulation (Cicchetti & Rogosch, 2001). Hence, we reasoned that it would be very likely that maltreated children with differing subtypes of maltreatment and combinations of clinical-level internalizing and externalizing problems would manifest varying patterns of adrenocortical regulation.

Not unexpectedly, we found that there were higher rates of clinical-level externalizing problems and clinical-level comorbid internalizing and externalizing problems in the maltreated children than there were in the nonmaltreated comparison youngsters. Specifically, maltreated children were found to have higher rates of case-level disturbances; 49.7% of the maltreated children exhibited clinical-level problems compared to 25.0% of the nonmaltreated comparison youngsters. Moreover, 24% of maltreated children exhibited externalizing problems and 16.2% displayed comorbid problems compared to 12.3% and 2% for the nonmaltreated children, respectively. The rates of clinical-level internalizing disorder were comparable between groups, with

9.6% of the maltreated and 10.8% of the nonmaltreated children manifesting internalizing problems. Within the maltreatment group, subtype group membership was not found to be significantly related to the manifestation of a specific form of clinical-level behavior problem. These findings suggest that children with common patterns of psychopathological disturbance had not encountered similar experiences of maltreatment.

Although prior investigations have reported increased depression and other internalizing problems in maltreated than in nonmaltreated children, in the present study there were no differences in clinical-level internalizing problems between maltreated and nonmaltreated children. However, actual differences in such case-level internalizing problems may have been masked by the fact that many maltreated children with internalizing clinical-level difficulties were in the comorbid internalizing and externalizing group. These results possess implications for clinical diagnosis and intervention, as it is possible that the internalizing disorders of maltreated children could be overlooked due to an emphasis on more overt and disruptive externalizing behavior problems.

Our finding that, independent of gender, maltreated children with clinical levels of internalizing problems exhibited increased morning and across the day average levels of cortisol compared to the other groups of maltreated and nonmaltreated children suggests that the presence of maltreatment moderated the impact of case-level internalizing problems on neuroendocrine functioning. Likewise, the discovery that maltreated children with case-level internalizing problems displayed higher afternoon cortisol than nonmaltreated children with clinical-level internalizing problems further suggests that maltreatment serves as a moderator of the impact of internalizing problems on adrenocortical regulation.

No differences in diurnal variation of cortisol were found across any of the groups with respect to internalizing case-level problems. Both the maltreated and the nonmaltreated children with internalizing problems exhibited the expected drop in cortisol from morning to late afternoon levels.

Furthermore, in an effort to discover differential patterns of cortisol regulation in relation to internalizing psychopathology, we examined children whose level of cortisol was in the top quartile (i.e., 25%) in both the morning and late afternoon. Children displaying this pattern were more prevalent in the group of maltreated children with clinical-level internalizing problems than in the nonmaltreated comparison internalizing group or in any other group.

The increased levels of cortisol found in the maltreated children with clinical levels of internalizing problems are extremely interesting. The results differ from those generally obtained with clinically diagnosed samples of depressed children and adolescents, who rarely show the increase in cortisol that is characteristic of a regulatory dysfunction of the HPA axis (Dahl & Ryan, 1996). The nonmaltreated children with case-level internalizing problems in our sample likewise did not exhibit increased levels of cortisol and an HPA axis dysregulation. However, the HPA axis abnormalities of the maltreated children with clinical levels of internalizing problems are strikingly similar to those obtained with maltreated children with PTSD and major depressive disorder (DeBellis, Baum, et al., 1999) and with depressed adults who were physically or sexually abused during their childhood (Heim, Newport, Bonsall, Miller, & Nemeroff, 2001; Heim et al., 2000; Lemieux & Coe, 1999). Thus, it appears that the experience of maltreatment intensifies the typical effects of depressive disorder in childhood on neuroendocrine functioning.

The increased morning, afternoon, and average daily levels of cortisol manifested by the maltreated children with case-level internalizing problems are suggestive of hypercortisolism. Consequently, maltreated children with significant internalizing psychopathology may be at risk for developing neurobiological anomalies, including neuronal loss in the hippocampus, inhibition of neuronal development, delays in the process of myelination, aberrations in synaptic pruning, and impairments in cognitive and affective functioning (Sapolsky, 1996; Vasquez, 1998).

The case-level internalizing problems in

our sample are derived from both the TRF and the CDI. The TRF measure contains anxiety items, in addition to those denoting depression. Thus, it is not possible to ascertain whether the increased levels of cortisol in maltreated children with internalizing problems are due either to maltreatment moderating depression, anxiety, or a combination of both anxiety and depression internalizing problems. Future investigations must be undertaken to differentiate depression, anxiety, and comorbid depression and anxiety, as these disorder presentations relate to neuroendocrine functioning in maltreated and nonmaltreated children.

With respect to clinical-level externalizing problems, we found that the nonmaltreated boys had lower levels of morning cortisol than all of the other nonmaltreated groups. Within the maltreated group, maltreated girls with case-level externalizing problems had lower morning cortisol than did maltreated girls without externalizing problems. These differences were not observed among nonmaltreated girls. Although there were no effects found for afternoon cortisol levels, the nonmaltreated boys with clinical-level externalizing problems exhibited the lowest average daily cortisol and were significantly lower than the maltreated girls without externalizing problems, the group that manifested the highest levels of average daily cortisol. The apparently limited effects of maltreatment in relation to externalizing problems and to cortisol secretion, especially for boys, were surprising. Lower levels of cortisol have been found to be associated with early onset aggressive conduct disorder (McBurnett et al., 2000). Moreover, it has been demonstrated that children with early onset conduct disorder evidence mild neuropsychological deficits (Moffitt, 1993a, 1993b), as well as more early family dysfunction, including child maltreatment (Aguilar, Sroufe, Egeland, & Carlson, 2000). We expected that maltreated children with clinical-level externalizing problems would exhibit even lower levels of cortisol than their nonmaltreated counterparts.

Differential patterns of adrenocortical regulation were further examined in relation to externalizing psychopathology. Children whose cortisol levels were in the lowest quartile and who did not decrease their cortisol levels through the afternoon were identified. More children with clinical-level externalizing problems than those children without externalizing problems exhibited this pattern (13.8% vs. 4.9%, respectively). However, there were no differences between maltreated children with case-level externalizing problems and nonmaltreated children with externalizing problems. These subgroups of children evidenced a pattern of cortisol regulation suggestive of hypocortisolism (cf. Cicchetti & Rogosch, 2001; Gunnar & Vazquez, 2001; Heim et al., 2000).

Despite the fact that the small number of nonmaltreated children with clinical levels of comorbid internalizing and externalizing problems precluded the inclusion of these youngsters in the statistical analyses, we examined the maltreatment group and searched for children who did not exhibit a decrease in cortisol levels across the day. Interestingly, a lack of decrease or a rise from morning to afternoon cortisol levels was found to be twice as common in maltreated children with clinical levels of comorbid problems than in maltreated children without comorbid problems.

The rise in afternoon levels of cortisol in the group of maltreated children with clinical levels of comorbid internalizing and externalizing problems calls to mind the results of Hart and colleagues (1996), who discovered that four out of nine maltreated children with clinical levels of depression evidenced a rise in afternoon cortisol rather than the expected diurnal drop. We think it is conceivable that the small group of maltreated children with clinical levels of depression in the Hart et al. (1996) investigation who evidenced increased afternoon cortisol secretion may have in fact had clinical levels of comorbid internalizing and externalizing problems.

In this investigation it has been conclusively demonstrated that not all maltreated children display the same pattern of cortisol regulation. Differential patterns of cortisol regulation were observed, providing further evidence that the neurobiological functioning of all maltreated children is not affected in a similar fashion by the experience of maltreatment (cf. Cicchetti, in press; Cicchetti & Ro-

gosch, 2001). Although the utilization of two different methods of collecting salivary samples and two different assay techniques may pose some limitations to the findings of the current study, the results of this investigation expand and elaborate upon the existing knowledge base of neuroendocrine functioning in maltreated children. Future longitudinal follow-up investigations of this sample of children, including the linking of maltreatment not only to neuroendocrine functioning, but also to neuropsychological and other psychological outcomes, will shed continued light on the processes underlying HPA axis dysregulation in maltreated children.

References

Aber, J. L., Allen, J. P., Carlson, V., & Cicchetti, D. (1989). The effects of maltreatment on development during early childhood: Recent studies and their theoretical, clinical, and policy implications. In D. Cicchetti & V. Carlson (Eds.), *Child maltreatment: Theory and research on the causes and consequences of child abuse and neglect* (pp. 579–619). New York: Cambridge University Press.

Aber, J. L., & Cicchetti, D. (1984). Socioemotional development in maltreated children: An empirical and theoretical analysis. In H. Fitzgerald, B. Lester, & M. Yogman (Eds.), *Theory and research in behavioral pediatrics* (Vol. 2, pp. 147–205). New York: Plenum Press.

Achenbach, T. M. (1991). *Manual for the Teacher's Report Form and 1991 Profile.* Burlington, VT: University of Vermont, Department of Psychiatry.

Achenbach, T. M., McConaughy, S. H., & Howell, C. T. (1987). Child/adolescent behavioral and emotional problems: Implications of cross-informant correlations for situational specificity. *Psychological Bulletin, 101,* 213–232.

Aguilar, B., Sroufe, L. A., Egeland, B., & Carlson, E. (2000). Distinguishing the early onset/persistent and adolescence-onset antisocial behavioral types: From birth to 16 years. *Development and Psychopathology, 12,* 109–132.

Allen, D., & Tarnowski, K. (1989). Depressive characteristics of physically abused children. *Journal of Abnormal Child Psychology, 17,* 1–11.

Barnett, D., Manly, J. T., & Cicchetti, D. (1993). Defining child maltreatment: The interface between policy and research. In D. Cicchetti & S. L. Toth (Eds.), *Child abuse, child development, and social policy* (pp. 7–73). Norwood, NJ: Ablex.

Belsky, J. (1984). The determinants of parenting: A process model. *Child Development, 55,* 83–96.

Belsky, J. (1993). Etiology of child maltreatment: A developmental–ecological analysis. *Psychological Bulletin, 114,* 413–434.

Belsky, J., & Vondra, J. (1989). Lessons from child abuse: The determinants of parenting. In D. Cicchetti & V. Carlson (Eds.), *Child maltreatment: Theory and research on the causes and consequences of child abuse and neglect* (pp. 153–202), New York: Cambridge University Press.

Birmaher, B., Ryan, N., Williamson, D., Brent, D., & Kaufman, J. (1996). Childhood and adolescent depression: A review of the past 10 years. Part II. *Journal of the American Academy of Child & Adolescent Psychiatry, 35,* 1575–1583.

Bolger, K. E., Patterson, C. J., & Kupersmidt, J. B. (1998). Peer relationships and self-esteem among children who have been maltreated. *Child Development, 69,* 1171–1197.

Bremner, J. D., Randall, P., Vermetten, E., Staib, L., Bronen, R. A., Mazure, C., Capelli, S., McCarthy, G., Innis, R. B., & Charney, D. S. (1997). Magnetic resonance imaging–based measurement of hippocampal volume in posttraumatic stress disorder related to childhood physical and sexual abuse. *Biological Psychiatry, 41,* 23–32.

Cerezo, M. A. (1997). Abusive family interaction: A review. *Aggression and Violent Behavior, 2,* 215–240.

Cicchetti, D. (1989). How research on child maltreatment has informed the study of child development: Perspectives from developmental psychopathology. In D. Cicchetti & V. Carlson (Eds.), *Child maltreatment: Theory and research on the causes and consequences of child abuse and neglect* (pp. 377–431). New York: Cambridge University Press.

Cicchetti, D. (in press). How a child builds a brain: Insights from normality and psychopathology. In W. Hartup & R. Weinberg (Eds.), *Minnesota Symposia on Child Psychology: Vol. 35. Child psychology in retrospect*. Mahwah, NJ: Erlbaum.

Cicchetti, D., & Barnett, D. (1991). Toward the development of a scientific nosology of child maltreatment. In W. Grove & D. Cicchetti (Eds.), *Thinking clearly about psychology: Essays in honor of Paul E. Meehl: Vol. 2. Personality and psychopathology* (pp. 346–377). Minneapolis, MN: University of Minnesota Press.

Cicchetti, D., & Lynch, M. (1993). Toward an ecological/transactional model of community violence and child maltreatment: Consequences for children's development. *Psychiatry, 56,* 96–118.

Cicchetti, D., & Lynch, M. (1995). Failures in the expectable environment and their impact on individual development: The case of child maltreatment. In D. Cicchetti & D. Cohen (Eds.), *Developmental psychopathology: Vol. 2. Risk, disorder and adaptation* (pp. 32–71). New York: Wiley.

Cicchetti, D., & Manly, J. T. (1990). A personal perspective on conducting research with maltreating families: Problems and solutions. In G. Brody & I. Sigel (Eds.), *Methods of family research: Vol. 2: Families at risk* (pp 87–133). Hillsdale, NJ: Erlbaum.

Cicchetti, D., & Rizley, R. (1981). Developmental perspectives on the etiology, intergenerational transmission, and sequelae of child maltreatment. *New Directions for Child Development, 11,* 32–59.

Cicchetti, D., & Rogosch, F. A. (1997). The role of self-organization in the promotion of resilience in mal-

treated children. *Development and Psychopathology, 9,* 799–817.

Cicchetti, D., & Rogosch, F. A. (2001). Diverse patterns of neuroendocrine activity in maltreated children. *Development and Psychopathology, 13,* 677–693.

Cicchetti, D., Rogosch, F. A., Lynch, M., & Holt, K. (1993). Resilience in maltreated children: Processes leading to adaptive outcome. *Development and Psychopathology, 5,* 629–647.

Cicchetti, D., & Toth, S. L. (1995). A developmental psychopathology perspective on child abuse and neglect. *Journal of the American Academy of Child & Adolescent Psychiatry, 34,* 541–565.

Cicchetti, D., & Toth, S. L. (2000). Developmental processes in maltreated children. In D. Hansen (Ed.), *Nebraska Symposium on Motivation: Vol. 46. Child maltreatment* (pp. 85–160). Lincoln, NE: University of Nebraska Press.

Cicchetti, D., & Tucker, D. (1994). Development and self-regulatory structures of the mind. *Development and Psychopathology, 6,* 533–549.

Cohen, P., Brown, J., & Smailes, E. (2001). Child abuse and neglect and the development of mental disorders in the general population. *Development and Psychopathology, 13,* 981–999.

Dahl, R., & Ryan, N. (1996). The psychobiology of adolescent depression. In D. Cicchetti & S. L. Toth (Eds.), *Rochester Symposium on Developmental Psychopathology: Vol. 7. Adolescence: Opportunities and challenges* (pp. 197–232). Rochester, NY: University of Rochester Press.

DeBellis, M. D., Baum, A. S., Birmaher, B., Keshavenm, M. S., Eccard, C. H., Boring, A. M., Jenkins, F. J., Ryan N. D. (1999). Developmental traumatology Part I: Biological stress systems. *Biological Psychiatry, 45,* 1259–1270.

DeBellis, M. D., Chrousos, G., Dorn, L., Burke, L., Helmers, K., Kling, M., Trickett, P., & Putnam, F. (1994). Hypothalamic–pituitary–adrenal axis dysregulation in sexually abused girls. *Journal of Clinical Endocrinology and Metabolism, 77,* 1–7.

DeBellis, M. D., Keshavan, M., Clark, D. B., Casey, B. J., Giedd, J., Boring, A. M. Frustaci, K., & Ryan, N. D. (1999). Developmental Traumatology Part II: Characteristics of trauma and psychiatric symptoms and adverse brain development in maltreated children and adolescents with PTSD. *Biological Psychiatry, 45,* 1271–1284.

DeBellis, M. D., & Putnam, F. W. (1994). The psychobiology of childhood maltreatment. *Child and Adolescent Psychiatric Clinics of North America, 3,* 663–678.

Famularo, R., Kinscherff, R., & Fenton, T. (1992). Psychiatric diagnoses of maltreated children: Preliminary findings. *Journal of the American Academy of Child & Adolescent Psychiatry, 31,* 863–867.

Francis, D., DiOrio, J., Liu, D., & Meaney, M. (1999). Nongenomic transmission across generations of maternal behavior in the rat. *Science, 286,* 1155–1158.

Gottlieb, G. (1992). *Individual development and evolution: The genesis of novel behavior*. New York: Oxford University Press.

Gould, E., Tanapat, P., McEwen, B. S., Flugge, G., & Fuchs, E. (1998). Proliferation of granule cell precursors in the dentate gyrus of adults monkeys is diminished by stress. *Proceedings of the National Academy of Sciences, 95,* 3168–3171.

Gunnar, M. R., & Vazquez, D. M. (2001). Low cortisol and a flattening of expected daytime rhythm: Potential indices of risk in human development. *Development and Psychopathology, 13,* 515–538.

Harrington, R., Rutter, M., & Fombonne, E. (1996). Developmental pathways in depression: Multiple meanings, antecedents, and endpoints. *Development and Psychopathology, 8,* 601–616.

Hart, J., Gunnar, M. & Cicchetti, D. (1995). Salivary cortisol in maltreated children: Evidence of relations between neuroendocrine activity and social competence. *Development and Psychopathology, 7,* 11–26.

Hart, J., Gunnar, M., & Cicchetti, D. (1996). Altered neuroendocrine activity in maltreated children related to depression. *Development and Psychopathology, 8,* 201–214.

Heim, C., Ehlert, U., & Hellhammer, D. (2000). The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuroendocrinology, 25,* 1–35.

Heim, C., Newport, D. J., Bonsall, R., Miller, A. H., & Nemeroff, C. B. (2001). Altered pituitary–adrenal axis responses to provocative challenge tests in adult survivors of childhood abuse. *American Journal of Psychiatry, 158,* 575–581.

Heim, C., Newport, D. J., Heit, S., Graham, Y. P., Wilcox, M., Bonsall, R., Miller, A. H., & Nemeroff, C. B. (2000). Pituitary–adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. *Journal of the American Medical Association, 284,* 592–597.

Hinshaw, S. P., Lahey, B. B., & Hart, E. L. (1993). Issues of taxonomy and comorbidity in the development of conduct disorder. *Development and Psychopathology, 5,* 31–49.

Howes, P. W., Cicchetti, D., Toth, S. L., & Rogosch, F. A. (2000). Affective, structural, and relational characteristics of maltreating families: A systems perspective. *Journal of Family Psychology, 14,* 95–110.

Ito, Y., Teicher, M. H., Glod, C. A., & Ackerman, E. (1998). Preliminary evidence for aberrant cortical development in abused children: A quantitative EEG study. *The Journal of Neuropsychiatry and Clinical Neuroscience, 10,* 298–307

Kashani, J. H., Shekim, W. O., Burk, J. P., & Beck, N. C. (1987). Abuse as a predictor or psychopathology in children and adolescents. *Journal of Clinical Child Psychology, 138,* 143–153.

Kaufman, J. (1991). Depressive disorders in maltreated children. *Journal of the American Academy of Child & Adolescent Psychiatry, 30,* 257–265.

Kaufman, J., Birmaher, B., Perel, J., Dahl, R. E., Moreci, P., Nelson, B., Wells, W., & Ryan, N. D. (1997). The corticotropin-releasing hormone challenge in depressed abused, depressed nonabused, and normal control children. *Biological Psychiatry, 42,* 669–679.

Kaufman, J., & Cicchetti, D. (1989). The effects of maltreatment on school-aged children's socioemotional development: Assessments in a day camp setting. *Developmental Psychology, 25,* 516–524.

Kazdin, A. E., Moser, J., Colbus, D., & Bell, R. (1985). Depressive symptoms among physically abused and psychiatrically disturbed children. *Journal of Abnormal Psychology, 94,* 298–307.

Kirschbaum, C., & Hellhammer, D. H. (1989). Salivary cortisol in psychobiology research: An overview. *Neuropsychobiology, 22,* 150–169.

Kovacs, M. (1985). The children's depression inventory. *Psychopharmacology Bulletin, 21,* 995–998.

Kovacs, M. (1992). *Children's Depression Inventory Manual.* North Tonawanda, NY; Multi-Health Systems.

Lemieux, A. M., & Coe, C. L. (1995). Abuse-related posttraumatic stress disorder: Evidence for chronic neuroendocrine activation in women. *Psychosomatic Medicine, 57,* 110–115.

Lopez, J. F., Akil, H., & Watson, S. J. (1999). Neural circuits mediating stress. *Biological Psychiatry, 46,* 1461–1471.

Lupien, S. J., King, S., Meaney, M. J., & McEwen, B. S. (2001). Can poverty get under your skin? Basal cortisol levels and cognitive function in children from low and high socioeconomic status. *Development and Psychopathology, 13,* 653–676.

Malinosky–Rummell, R., & Hansen, D. (1993). Long-term consequences of childhood physical abuse. *Psychological Bulletin, 114,* 68–79.

Manly, J., Cicchetti, D., & Barnett, D. (1994). The impact of subtype, frequency, chronicity, and severity of child maltreatment on social competence and behavior problems. *Development and Psychopathology, 6,* 121–143.

Manly, J., Kim, J., Rogosch, F. A., & Cicchetti, D. (2001) Dimensions of child maltreatment and children's adjustment: Contributions of developmental timing and subtype. *Development and Psychopathology, 13,* 759–782.

McBurnett, K., Lahey, B. B., Rathouz, P. J., & Loeber, R. (2000). Low salivary cortisol and persistent aggression in boys referred for disruptive behavior. *Archives of General Psychiatry, 57,* 38–43.

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *Seminars in Medicine of the Beth Israel Deaconess Medical Center, 338,* 171–179.

McEwen, B. S. (2000). Effects of adverse experiences for brain structure and function. *Biological Psychiatry, 48,* 721–731.

McGloin, J. M., & Widom, C. (2001). Resilience among abused and neglected children grown up. *Development and Psychopathology, 13,* 1021–1038.

Meaney, M. J., DiOrio, J., Francis, D., Widdowson, J., LaBlante, P., Caldji, C., Sharma, S. Seckl, J. R., & Plotsky, P. M. (1996). Early environmental regulation of forebrain, glucocorticoid receptor gene expression: Implications for adrenocortical response to stress. *Developmental Neuroscience, 18,* 49–72.

Moffitt, T. E. (1993a). Life course persistent and adolescence limited antisocial behavior: A developmental taxonomy. *Psychological Review, 100,* 674–701.

Moffitt, T. E., (1993b). The neuropsychology of conduct disorder. *Development and Psychopathology, 5,* 135–151.

Pajer, K., Garner, W., Rubin, R. T, Perel, J., & Neal, S. (2001). Decreased cortisol levels in adolescent girls with conduct disorder. *Archives of General Psychiatry, 58,* 297–302.

Pollak, S. D., Cicchetti, D., Klorman, R., & Brumaghim, J. T. (1997). Cognitive brain event-related potentials and emotion processing in maltreated children. *Child Development, 68,* 773–787.

Pollak, S. D., Klorman, R., Brumaghim, J., & Cicchetti, D. (2001). P3b reflects maltreated children's reactions to facial displays of emotion. *Psychophysiology, 38,* 267–274.

Putnam, F. W., Trickett, P. K., Helmers, K., Dorn, L., & Everett, B. (1991). Cortisol abnormalities in sexually abused girls. *Proceedings of the 144th Annual Meeting of the American Psychiatric Association* (p. 107). Washington, DC: American Psychiatric Press.

Pynoos, R., Steinberg, A., & Wraith, R. (1995). A developmental model for childhood traumatic stress. In D. Cicchetti & D. Cohen (Eds.), *Developmental psychopathology: Vol. 2. Risk, disorder, and adaptation* (pp. 72–95). New York: Wiley.

Rogosch, F., Cicchetti, D., Shields, A., & Toth, S. L. (1995). Parenting dysfunction in child maltreatment. In M. H. Bornstein (Ed.), *Handbook of parenting* (Vol. 4, pp. 127–159). Hillsdale, NJ: Erlbaum.

Sapolsky, R. M. (1992). *Stress, the aging brain and the mechanisms of neuron death.* Cambridge, MA: MIT Press.

Sapolsky, R. M. (1996). Stress, glucocorticoids, and damage to the NS: The current state of confusion. *Stress, 1,* 1–19.

Saylor, C., Spirito, A., Fince, A., & Bennett, B. (1984). The Children's Depression Inventory: A systematic evaluation of psychometric properties. *Journal of Consulting and Clinical Psychology, 52,* 955–967.

Schwartz, E. B., Granger, D. A., Susman, E. J., Gunnar, M. R., & Laird, B. (1998). Assessing salivary cortisol in studies of child development. *Child Development, 69,* 1503–1513.

Shields, A., & Cicchetti, D. (1998). Reactive aggression among maltreated children: The contributions of attention and emotion dysregulation. *Journal of Clinical Child Psychology, 27,* 381–395.

Smith, C. A., & Thornberry, T., (1995). The relationship between child maltreatment and adolescent involvement in delinquency. *Criminology, 33,* 451–481.

Sternberg, K., Lamb, M., Greenbaum, C., Cicchetti, D, Dawud, S., Cortes, R., & Krispin, O. (1993). Effects of domestic violence on children's behavior problems and depression. *Developmental Psychology, 29,* 44–52.

Todd, R. D., Swarzenski, B., Rossi, P., & Visconti, P. (1995). Structural and functional development of the human brain. In D. Cicchetti & D. Cohen (Eds.), *Developmental psychopathology: Vol. 1. Theory and methods* (pp. 161–194). New York: Wiley.

Toth, S. L., Manly, J. T., & Cicchetti, D. (1992). Child maltreatment and vulnerability to depression. *Development and Psychopathology, 4,* 97–112.

Trickett, P., & McBride–Chang, C. (1995). The developmental impact of different types of child abuse and neglect. *Developmental Review, 15,* 311–337.

Van Goozen, S. H., Matthys, W., Cohen–Kettenis, P., Buitelaar, J., & van Engelard, H. (2000). Hypothalamic–pituitary–adrenal axis and autonomic nervous system activity in disruptive children and matched controls. *Journal of the American Academy of Child & Adolescent Psychiatry, 39,* 1438–1445.

Vazquez, D. M. (1998). Stress and the developing limbic–hypothalamic–pituitary–adrenal axis. *Psychoneuroendocrinology, 23,* 663–700.

Vinson, G. P., Whitehouse, B. J., & Hinson, J. P. (1998). Structure and function of the adrenal cortex. In A. Grossman (Ed.), *Clinical endocrinology,* 2nd ed., pp. 401–430. London: Blackwell Science.

Young, L. J., Nilson, R., Waymore, K., MacGregor, G. R., & Insel, T. R. (1999). Increased affiliative response to vasopressin in mice expressing the V1a receptor from a monogamous male. *Nature, 400,* 766–768.