Reciprocal Influences among Adrenocortical Activation, Psychosocial Processes, and the Behavioral Adjustment of Clinic-Referred Children

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GRANGER, DOUGLAS A.; WEISZ, JOHN R.; MCCRACKEN, JAMES T.; IKEDA, SANDRA C.; and DOUGLAS, PAMELA. *Reciprocal Influences among Adrenocortical Activation, Psychosocial Processes, and the Behavioral Adjustment of Clinic-Referred Children.* CHILD DEVELOPMENT, 1996, **67**, 3250–3262. The reciprocal effects among cognitive-behavioral, environmental, and biological influences on clinic-referred children's (N = 64; 34 boys; M age 12.71 years) short-term psychological and psychiatric adjustment were studied. At clinic intake and 6 months later, standardized measures of adjustment and control-related beliefs were assessed. Before and after conflict-oriented parent-child interaction tasks the children's saliva was sampled. Adrenocortical responses (i.e., increases in salivary cortisol) to the social conflict task predicted children's internalizing problem behaviors and anxiety disorders at follow-up. Consistently high adrenocortical reactivity at intake *and* follow-up was associated with deflated social competence over the 6-month period. Also, specific patterns of discontinuity in children's internalizing behavior problems predicted individual differences in their subsequent adrenocortical responsiveness. Specifically, rising behavior problem levels across time predicted higher and declining behavior problem levels predicted lower adrenocortical reactivity at follow-up. Findings are among the first to suggest links among internalizing behavior problems, adrenocortical responsiveness to social challenge, and clinic-referred children's short-term cognitive-behavioral and emotional adjustment.

For several decades, researchers have debated whether the reciprocal influences among social processes, environmental events, and the activity of the hypothalamic-pituitary-adrenocortical (HPA)[1] axis affect individual differences in health and human development (e.g., Kling et al., 1989; Levine, 1970; Mason, 1968; McEwen & Schmeck, 1994; Selye, 1950). Recently, advances in the neurosciences have eliminated

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[1] The terms HPA axis activation or reactivity and adrenocortical activation or reactivity are used interchangeably throughout.

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much of the doubt (Chrousos & Gold, 1992; Gold, Goodwin, & Chrousos, 1988; Sapolsky, 1992). Studies reveal hormone secretion evoked on an individual basis according to life events can have substantial effects on developmental processes (e.g., Meaney et al., 1991).

Not surprisingly, the implications of these findings for child development have become a new focus for research (Gunnar, 1986; Susman et al., 1989). Some studies report modest associations between children's HPA axis activation and behavioral reactivity to physical, social, and cognitive challenges (e.g., Kagan, Reznick, & Snidman, 1987; Tennes & Kreye, 1985; Tennes, Kreye, Avitable, & Wells, 1986). There is also scattered evidence that the biobehavioral processes that regulate HPA activation may contribute to the prediction of children's subsequent behavioral adjustment (e.g., Granger, Stansbury, & Henker, 1994; Gunnar, 1993a; Kagan, Snidman, & Reznick, 1989). Thus far, however, there have been few attempts to link individual differences in HPA axis and behavioral responsiveness to environmental challenge among children who have behavioral difficulties severe enough to warrant clinic-referral. Furthermore, no studies (to our knowledge) have addressed the extent to which patterns of children's adrenocortical activation and problem behavior covary longitudinally. The latter represents a substantial gap in our knowledge. It seems plausible that the frequency of negative personal life events, or exaggerated psychological responses to those events, or both, experienced by children with behavioral and emotional problems may evince irregular, or perhaps chronic, HPA axis activation.

The handful of relevant studies of clinic samples of children have produced invaluable information, but they have significant limitations. For instance, many of the data have been derived from studies that measure basal levels of HPA axis hormones (i.e., ACTH, cortisol), the circadian profile of cortisol production, or changes in HPA axis products in response to pharmacological challenge (e.g., Dorn et al., in press; Kruesi, Schmidt, Donnelly, Hibbs, & Hamburger, 1989; Pfeffer et al., 1989; Puig-Antich et al., 1989). The emerging literature of studies with normally developing children suggests that cortisol-behavior associations are more likely to be detected, and cortisol-behavior relations may have more significant developmental implications, when the HPA axis measurements represent reactivity to social-ecologically valid environmental challenges, than when they index basal activity or pharmacologically induced reactivity (e.g., Gunnar, Marvinney, Isenee, & Fisch, 1989). Thus, it is somewhat surprising that no studies have explored the effects of psychosocial challenge on cortisol-behavior relations in clinic populations of children.

Additionally, few studies with clinic-referred children have incorporated measures of control-related constructs in their designs. This may also be an important gap, in that animal-model studies describe control as a significant mediator of HPA axis activation to environmental challenge (Levine, 1980; Rose, 1980), and some findings support a linkage between adrenocortical activation and perceived control among adult samples (Breier, 1989). Given recent findings suggesting that control-related beliefs may be related to patterns of child psychopathology (Weisz, Sweeney, Proffitt, & Carr, 1993; Weisz, Weiss, Wasserman, & Rintoul, 1987), studies that integrate assessments of both control-related beliefs and HPA axis hormones in clinic samples of children would seem to be an appropriate next step.

Finally, most previous studies have focused on single dimensions of child psychopathology (e.g., depression, aggression). In the process, important but more complex or additive relations may have been missed. For example, McBurnett et al. (1991) reported that only the *combination* of anxiety and conduct problems was linked to high salivary cortisol, and in a preliminary study, our group (Granger, Weisz, Kauneckis, & Rudolph, 1992) observed that the combination of anxiety *and* depression was associated with children's adrenocortical activation. Such findings suggest that the most fruitful approach to inquiry in this area would sustain a focus on multiple rather than single dimensions of psychopathology.

As an initial step toward addressing some of these limitations, we explored relations among clinic-referred children's behavior problems, control-related beliefs, and adrenocortical activation (i.e., increases in salivary cortisol levels) to a developmentally appropriate social challenge—a parent-child conflict discussion task (see Granger, Weisz, & Kauneckis, 1994). The study yielded several potentially important findings. In comparison to other subjects, children who showed a rise in salivary cortisol level in re-

sponse to the challenge were more socially withdrawn, more socially anxious, and had more somatic complaints and social problems. They also were likely to attribute the outcomes of their personal successes and failures to external sources and less likely to perceive the social outcomes that children experience as contingent on the children and what they do.

The present study was designed to test four alternative hypotheses regarding the direction of effect between measures of behavior problems and adjustment, and assessments of adrenocortical activation. The data were collected as part of the intake and 6-month follow-up assessments for the families studied in our previous report (Granger, Weisz, & Kauneckis, 1994). If aspects of the biobehavioral processes regulating HPA axis activation are affecting children's psychological adjustment, one would expect that adrenocortical activation measured at intake would predict subsequent levels of internalizing problem behavior and/or patterns of control beliefs, but that measures of psychological adjustment at intake would not predict subsequent levels of HPA axis activation. Conversely, if patterns of children's internalizing behavior problems and control-related beliefs are affecting the biobehavioral processes moderating HPA axis activation, one would expect that measures of these constructs at intake would predict adrenocortical activation 6 months later, and that adrenocortical activation measured at intake would not predict subsequent levels of behavior problems or control-related beliefs. Alternatively, it may be the pattern (hyper- vs. hyporeactivity) or pervasiveness (i.e., cross-time or cross-situational consistency), of HPA axis activation that has the most developmental significance. If this were true, individuals who exhibit consistently high or consistently low adrenocortical activation across the intake and follow-up assessments should have different levels of psychological adjustment at follow-up. Of course, it is also possible that the cross-sectional correlates of HPA axis activation observed in our previous report were related to transient problems leading to the children's clinic referral. If this were the case, cortisol-behavior relations would not be detected 6 months later, and measures of adrenocortical activation and psychological adjustment would be unrelated across time. Evidence supporting any one of these hypothetical outcomes would help refine working theoretical models regarding biobehavioral processes that mediate individual development (e.g., Gottlieb, 1992).

Method

Overview

The data were collected as part of a multisite study of youngsters receiving mental health care services at community-based clinics in California. Children and their mothers were interviewed within days of the families' clinic intake assessment and then again 6 months later. Each interview involved assessments of children's behavioral and emotional problems, and psychiatric symptoms and diagnoses; control, competence, and contingency beliefs; and the collection of children's saliva before and after their participation in a parent-child interaction task. The children and parents individually completed the paper and pencil questionnaires over the course of 2–3 hours. To avoid the possibility of carryover effects from measure to measure, participating families were randomly assigned to complete the questionnaires and interaction tasks in several fixed orders. Families were selected from consecutive clinic referrals. Conditions likely to interfere with the detection of the relations of interest (i.e., mental retardation, organic impairment interfering with cognitive function, and childhood onset psychosis) were excluded.

Subjects

The data reported here were from families available 6 months after the initial interview for a follow-up assessment. There were no differences between the 75 youngsters completing and the 27 youngsters failing to complete the follow-up assessments with respect to age, gender, ethnicity, family income levels, or intake levels of behavior problems. In this report, we restricted the sample further. Participants who at intake were less than 9 years old were excluded to reduce the possibility that reliability problems associated with younger children's self-report would compromise the validity of the assessments.

At intake, the average age of the 64 children (34 boys) was (M = 12.71, SD = 2.15). Twenty-eight children (16 boys) ranged in age from 9 to 11.99 years (M = 10.60, SD = .80), and 36 children (18 boys) ranged in age from 12 to 16.45 years (M = 14.34, SD = 1.23). The children were 45.3% Caucasian, 26.6% Latino, 18.8% African-American, and 9.3% mixed/other ethnic backgrounds. The median annual family income was approxi-

mately $22,000, and the average parent education level corresponded to completion of 1 year of college.

As is typical in outpatient clinics, the youngsters were referred for a variety of problems. Using the parent version of the Diagnostic Interview Schedule for Children (DISC-2.3; National Institute of Mental Health, 1991) with DISC-2.3c scoring logic (Piacentini & Fisher, 1992) multiple DSM-III-R (American Psychiatric Association, 1987) diagnoses were common. The mean number of diagnoses per child at intake was 2.3 (SD = 2.17); the median was 2.0, with 25% of the sample meeting criteria for three or more diagnoses. With respect to diagnostic categories, 54.7% received diagnoses for one or more Anxiety Disorder, 59.4% diagnoses for one or more Disruptive Behavior Disorder, and 26.6% for one or more Affective Disorder.

Behavioral Assessments

Assessing problem behavior.—During both assessments children completed the Youth Self-Report and parents filled in the Child Behavior Checklist (YSR, CBCL; Achenbach, 1991a, 1991b). These standardized measures list 118 specific child behavior problems. They generate *T* scores that reflect a child's status relative to others of the same sex and age in Internalizing, Externalizing, and Total Problems, as well as in eight narrow-band syndromes: Anxiety/Depression (e.g., complains of loneliness; feels worthless or inferior; unhappy, sad, or depressed), Somatic Complaints (e.g., physical problems without known medical cause, such as stomachaches or cramps, rashes or skin problems, and headaches), Social Problems (e.g., clings to adults or too dependent; doesn't get along with other children; gets teased a lot), Aggression (e.g., argues a lot; gets in many fights; stubborn, sullen, or irritable), Delinquency (e.g., lying or cheating, vandalism, stealing), Attention Problems (e.g., impulsive, can't concentrate, daydreams), Social Withdrawal (e.g., likes to be alone; shy or timid; withdrawn, doesn't get involved with others), and Thought Problems (e.g., hears or sees things that aren't there, repeats certain acts over and over). Validity, internal consistency, and test-retest reliability have been extensively documented (Achenbach, 1991a, 1991b). Cross-informant scores, computed for the initial and follow-up assessments separately, were used in the analyses. The composite scores were computed by converting parent and child raw scores for the three summary and eight syndrome scales to Z scores and then averaging across information source. Raw-to-Z score transformations were used because some of the children were younger than the established age and gender norms used to generate YSR *T* scores.[2]

On each occasion, the children completed the Social Anxiety Scale for Children (SASC; La Greca, Dandes, Wick, Shaw, & Stone, 1988) and the Children's Depression Inventory (CDI; Kovacs, 1983). The SASC is a 10-item self-report measure of children's feelings of social anxiety. Items of the SASC represent three components of social anxiety: Fear of negative evaluation, social avoidance, and social distress. La Greca et al. (1988) report internal consistency as measured by Cronbach's alpha as .76, and test-retest reliability over a 2-week period as .67. The validity of the SASC is supported by findings linking scores to related constructs, such as chronic anxiety measured by the Revised Children's Manifest Anxiety Scale (Reynolds & Richmond, 1978) and peer neglect (La Greca et al., 1988). The 27 items of the CDI are designed to survey major depressive symptoms (Kovacs, 1983). The CDI has well-documented psychometric properties. Internal consistency measured by coefficient alpha ranges from 0.84 to 0.94 (Kovacs, 1980; Saylor, Finch, Spirito, & Bennett, 1984; Smucker, Craighead, Craighead, & Green, 1986). Test-retest reliability over periods of 1, 3, 4, and 6 weeks ranges from 0.38 to 0.87 (Kovacs, 1980; Smucker et al., 1986).

Psychiatric diagnoses.—As noted above, parents also completed the Diagnostic Interview Schedule for Children (DISC-2.3; NIMH, 1991). The DISC-2.3c scoring logic (Piacentini & Fisher, 1992) was used to determine DSM-III-R diagnoses within each of the broad categories of Dis-

[2] Consistent with previous findings regarding the magnitude of cross-informant associations for the Achenbach scales (Achenbach, McConaughy, & Howell, 1987), correlations between parent (CBCL) and child (YSR) summary and narrow-band raw scores were on average .28 and .25, respectively ($dfs = 62$, $ps < .05$). The procedure used to compute cross-informant scores was developed in consultation with David Jacobowitz, Department of Psychiatry, University of Vermont. All questions were administered to the children in an interview format by trained interviewers.

ruptive Behavior Disorders (i.e., attention deficit hyperactivity disorder, conduct disorder, oppositional defiant disorder), Anxiety Disorders (i.e., avoidant disorder, generalized anxiety disorder, overanxious disorder, separation anxiety disorder, simple phobia, and social phobia), and Affective Disorders (i.e., major depression and dysthymia). The total number of diagnoses within each of the three broad categories were the scores used in the analyses. The DISC-2.3 was administered by advanced clinical psychology graduate students who had received instruction by certified trainers. Validity, interrater and test-retest reliability of the Diagnostic Interview Schedule for Children have been extensively documented (Fisher et al., 1993; Schwab-Stone et al., 1993; Shaffer et al., 1993).

Assessing control-related constructs.— Following Weisz and colleagues' two-dimensional model of perceived control, which construes control as a function of outcome contingency and personal competence (Weisz, 1986; Weisz & Stipek, 1982), three approaches to measuring children's control-related beliefs were used. The first was the 24-item Personal Experience Form A of the Multidimensional Measure of Children's Perceptions of Control (MMCPC; Connell, 1980, 1985). This well-standardized measure distinguishes children's attributions to external, internal, and unknown causes regarding their successes and failures. Children's ratings of how true each item is are summed to form six scores: internal success and failure, powerful other success and failure, and unknown success and failure. The validity of the internal and external MMCPC scales is supported by findings linking scores to other control-related constructs, such as perceived and actual competence (Connell, 1985; Connell & Tero, 1982). However, evidence linking the MMCPC Unknown scales to other measures of perceived control is less substantial; thus, these scales were excluded from the analyses. Test-retest reliability of the various scales over periods of 9 and 17 months ranged from means of 0.32 to 0.34 (Connell, 1985). As for internal consistency, averaged coefficient alphas ranged from .59 to .68 across the six scales (see Weisz et al., 1993).

Second, children's contingency beliefs were assessed via the Perceived Contingency Scale for Children (PCSC; Weisz, Proffitt, & Sweeney, 1991). The PCSC includes 30 self-report items, all focused on perceived contingencies for children in general. Children's ratings of how true each item is are summed to form three contingency scores: academic, behavioral, and social contingency. Weisz et al. (1993) report subscale alphas of 0.69, 0.75, and 0.74, and test-retest reliability over a 10-day interval of 0.78, 0.48, and 0.70 for the academic, behavioral, and social subscales, respectively.

Third, children's perceived competence was measured via the Self-Perception Profile for Children (SPPC; Harter, 1985). This 36-item scale is designed to assess children's self-perceptions across five specific domains: academic, social, behavioral, athletic, and physical appearance (the social domain questions emphasize social acceptance by others). Harter (1982) reported test-retest reliabilities for an earlier form of her scale, with 3-month reliabilities for the subscales ranging from 0.70 to 0.87, and 9-month reliabilities ranging from 0.69 to 0.80. For the current version, Harter (1985) has reported internal consistency alphas for the behavioral and social domains ranging from 0.71 to 0.77 and 0.75 to 0.80, respectively. The athletic and physical appearance subscales were excluded from the analyses; they were not included in the assessment battery of the larger project.

Psychosocial Challenge: Parent-Child Interaction Task

A parent-child conflict discussion task was used as the psychosocially challenging event because (*a*) naturally occurring parent-child conflicts often precede increases in children's cortisol levels (Flinn & England, 1992); (*b*) procedures for parent-child interaction tasks are well developed, and studies show that interactions during such tasks are associated with patterns of children's behavior problems (Burge & Hammen, 1991; Cook, Kenny, & Goldstein, 1991); and (*c*) parent-child discord is likely to be a particularly salient social stressor for clinic-referred children since it is often associated with children's referral problems (Weisz & Weiss, 1991).

Prior to their participation in the interaction portion of each interview, children and mothers independently rated 13 topics thought to be potential sources of parent-child problems (e.g., chores, schoolwork, following instructions, curfew). Then interviewers compared the two sets of ratings and choose a topic that both parent and child judged as a source of conflict, and where both people were similarly invested. The topic selected, in each case, was the one on

which parent and child ratings differed by no more than two rating points (on a six-point scale), and for which the sum of the ratings was the largest of all the topics.

Later, mothers and children were united to complete three interaction tasks. The activities were designed to be progressively more interactive and progressively more likely to provoke conflict. The first task was intended to familiarize the dyad with the activity setting and thus placed few demands on the child—mothers were asked to teach the children how to plan a meal for a large family. The next activity provided an opportunity for the children to play a more active role in problem solving and negotiating during a cooperative interaction task—the dyads were asked to work together to generate a plan for a hypothetical weekend vacation. Finally, mother and child were asked to discuss the conflict topic with the goal of reaching some solution. The teaching, planning, and conflict discussion activities continued uninterrupted for 4, 5, and 6 minutes, respectively. Except for explaining task instructions during the transition periods, the interviewers did not intervene in any way. This procedure was identical for both interviews, but the dyads were not allowed to discuss the same conflict topic on the two different occasions.

Assessing task behavior and affect.—Immediately after the social interaction tasks, the children completed brief checklists regarding their behavior and affect during the conflict discussions. Four individual items were summed to form two self-report task behavior scores: Social Inhibition ("I felt afraid to talk," "I tried to avoid talking") and Anxious/Inhibited Affect (e.g., I felt "scared," "nervous").

Cortisol Measurement

Saliva samples were collected from each child immediately before (pretask) and 20 min after (posttask) the parent-child interaction task on both interview occasions. The majority of interviews were conducted in the afternoons and early evenings. Sample collection times were, on average, for pre- and posttask samples 4:25 (SD = 3 hours 13 min) and 5:22 (SD = 3 hours 17 min) P.M. at intake; 2:44 (SD = 3 hours 34 min) and 3:42 (SD = 3 hours 33 min) P.M. for pre- and posttask samples at follow-up.

Sampling procedures were adapted from those used by Gunnar, Mangelsdorf, Larson, and Hertsgaard (1989). Each child tasted sugar-sweetened grape-flavored Kool-Aid crystals and then chewed on a sterile cotton dental roll. After 2–3 min, the cotton was removed from the child's mouth. A syringe was used to compress the saturated cotton and express the saliva into a cryogenic tube. The samples were immediately frozen and then stored at −80°c until packed in dry ice and shipped to the University of Minnesota Hospital Endocrine Lab, where they were assayed in duplicate using a radioimmune assay for cortisol. The duplicate tests for each sample were averaged to represent the cortisol values used in the analyses. The averaged intra- and interassay coefficients of variation were less than 5% and 10%, respectively.

Results

STRATEGY OF THE ANALYSES

First, an ANOVA design was used to assess whether adrenocortical activation, for the children as a group, was related to their participation in the parent-child discussion tasks. These analyses also assessed associations between cortisol levels and age, gender, and sample collection time. Then, the main analyses used regression models to test time-lagged linear relations among cortisol scores and behavior problems, control-belief, and task behavior measures.

EFFECTS OF AGE, GENDER, SAMPLING OCCASION, COLLECTION TIME, AND INITIAL CORTISOL LEVELS

The first analysis used cortisol level as the dependent variable in a sampling occasion (pretask vs. posttask) × assessment time (intake vs. follow-up) × gender × age group (children vs. adolescents) mixed model ANOVA. Sampling occasion and assessment time were the repeated measures. Results revealed a main effect for sampling occasion, $F(1, 60) = 23.29$, $p < .0001$, indicating that, on average, cortisol levels dropped from pretask, mean .24 (μg/dl), SD = .10; SE = .012, to posttask, mean .19 (μg/dl), SD = .13; SE = .016. There were no significant effects observed involving gender, age group, or assessment time. For descriptive purposes "delta scores" were computed (posttask minus pretask cortisol levels). Higher delta scores reflect larger increases in cortisol from pre- to posttask. At intake, delta scores ranged from −.41 to .21 (μg/dl), $M = -.04$ (μg/dl), SD = .11; SE = .014. At follow-up, delta scores ranged from −.32 to .13 (μg/dl), $M = -.05$ (μg/dl), SD = .09; SE = .011. In the absence of empirical guidelines for the expected range of individ-

ual differences in cortisol responses in this context, we used a conservative criterion to define a cortisol increase. A 15% elevation (minimum absolute increase of .03 μg/dl) over pretask levels was used because elevations of this magnitude are unlikely to be attributable to variation in the assay. Using that criterion, 15 of the children at intake and nine at follow-up showed cortisol elevations, and the magnitude of these increases was on average 47.08% and 141.18% at intake and follow-up, respectively. Also, pretask cortisol levels were strongly associated with posttask levels. The correlation between pre- and posttask cortisol levels was .73 at intake, and .81 at follow-up (dfs = 62, $ps < .001$). At intake, posttask, $r(62) = -.29$, $p < .021$, cortisol levels were higher when interviews were conducted earlier than later in the day. A similar pattern was observed at follow-up, pretask, $r(62) = -.42$, $p < .001$, posttask, $r(62) = -.55$, $p < .001$.

Main Analyses: Time-Lagged Effects

Here, time-lagged linear associations were tested to address whether (*a*) individual differences in children's adrenocortical activation to psychosocial challenge predict their subsequent adjustment, (*b*) individual differences in the levels of children's adjustment predict their subsequent adrenocortical activation, or (*c*) both. To control the effects of initial (pretask) cortisol levels and sample collection time, the cortisol scores used in the main analyses were residualized gain scores generated using linear regression as follows. Separately for intake and follow-up, posttask cortisol levels were predicted from their corresponding pretask levels and collection times. The predicted scores were then subtracted from the observed posttask levels. Higher residualized gain scores represented larger task-related increases in salivary cortisol from pre- to posttask.

Are Individual Differences in HPA Axis Activation to Psychosocial Challenge Sensitive Predictors of Children's Psychological Adjustment?

These analyses used a multiple linear regression model, that is, Outcome = a + $b_0\text{Beh}^{t1}$ + $b_1\text{Age}$ + $b_2\text{Gender}$ + $b_3\text{Cort}^{t2}$ + $b_4\text{Cort}^{t1}$ + $b_5(\text{Cort}^{t1} \times \text{Cort}^{t2})$, to predict adjustment at outcome from the HPA axis measures. In the model, "Outcome" represented the level of adjustment measured at the 6-month follow-up; "Beh^{t1}" represented the levels of the outcome variable measured at the intake; "b_3" and "b_4" estimated the main effects of adrenocortical activation measured at follow-up and intake respectively; and "b_5" estimated the interaction between intake and follow-up cortisol gain scores. The interaction term, ($\text{Cort}^{t1} \times \text{Cort}^{t2}$), was included to test the hypothesis that patterns of adrenocortical activation across time (e.g., consistently high, consistently low, or situationally high reactions) may be differentially associated with outcome.

We used a conservative two-step approach to these analyses. First, separate equations, structured as described in the previous paragraph, were computed for each of the behavioral and adjustment measures and (using the standardized betas, b, and incremental change in r^2, Δr^2) the unique contributions of the main and interactive effects over and above those of the control variables to each prediction were interpreted. Then, in specific instances, when more than one behavior and adjustment measure was significantly predicted by a specific adrenocortical activation score (i.e., either cort^{t1}, cort^{t2}, or $\text{cort}^{t1} \times \text{cort}^{t2}$) the respective adrenocortical activation term was regressed (i.e., using stepwise procedures, PIN < .05, and controlling for age, gender, and intake levels of the respective adjustment scores) on the select subset of behavior and adjustment measures. In these follow-up analyses, the equations employed adrenocortical activation terms as the criterion measures, and the behavior and adjustment measures were the predictors. The first step enabled us to estimate the predictive power of the cortisol gain scores independently of the control variables. The second step was needed because the behavior and adjustment measures were not completely orthogonal to each other. Accordingly, step 2 yielded a picture of the relation between the cortisol gain scores and the behavior and adjustment measures, with the relative contribution of the measures reflected by the order in which they entered the equation.

Main effects of intake HPA reactivity ($Cort^{t1}$).—The first set of analyses revealed that lower levels of HPA reactivity at intake predicted higher scores on the cross-informant Externalizing, $\Delta r^2 = .029$, $F(5, 58) = 4.26$, $b = -.17$, $p < .043$, and Aggression scales, $\Delta r^2 = .041$, $F(5, 58) = 6.77$, $b = -.20$, $p < .011$. Higher reactivity scores predicted lower ratings on the cross-informant Internalizing, $\Delta r^2 = .054$, $F(5, 58) = 6.16$, $b = -.24$, $p < .016$, and Anxiety/Depression scales, $\Delta r^2 = .065$, $F(5, 58) = 7.43$, $b = -.26$, $p < .008$, at follow-up. Higher reactiv-

ity scores predicted more DSM-III-R diagnoses of Anxiety Disorders, $\Delta r^2 = .057$, $F(5, 58) = 7.99$, $b = .24$, $p < .006$, and diagnoses of Overanxious Disorder in particular, $\Delta r^2 = .041$, $F(5, 58) = 4.19$, $b = .20$, $p < .043$, at follow-up. Noticeably absent were any main effects of $Cort^{t1}$ on control-related beliefs or task behavior and affect at follow-up. Next, $Cort^{t1}$ was regressed on the subset of outcome measures listed above using stepwise procedures. After the control variables were entered, DSM-III-R diagnoses of Overanxious Disorder entered the equation, $\Delta r^2 = .125$, $F(8, 55) = 8.99$, $b = .55$, $p < .004$, followed by the cross-informant internalizing behavior problem scale, $\Delta r^2 = .121$, $F(9, 54) = 10.10$, $b = -.48$, $p < .002$.

Main effects of follow-up HPA reactivity ($Cort^{t2}$).—The first set of equations also revealed that higher follow-up HPA reactivity levels predicted higher scores on the cross-informant Thought Problems, $\Delta r^2 = .069$, $F(4, 59) = 5.90$, $b = .28$, $p < .018$, and Delinquency, $\Delta r^2 = .047$, $F(4, 59) = 5.98$, $b = .23$, $p < .017$, scales at follow-up. Noticeably absent were any associations between HPA reactivity and control-related beliefs, DSM-III-R diagnoses, or task behavior and affect at follow-up. When the follow-up HPA reactivity scores were regressed (using stepwise procedures) on the two cross-informant scales, both scales contributed uniquely to the prediction. After the control variables were entered, the Thought Problems scale entered the equation first, $\Delta r^2 = .076$, $F(5, 58) = 5.68$, $b = .31$, $p < .020$, followed by the Delinquency scale, $\Delta r^2 = .077$, $F(6, 57) = 6.24$, $b = -.40$, $p < .015$.

Interactive effects of patterns of HPA reactivity across time ($Cort^{t1} \times Cort^{t2}$).—The $Cort^{t1} \times Cort^{t2}$ interaction term predicted the children's self-ratings on the PCSC Social Contingency scale, $\Delta r^2 = .068$, $F(6, 56) = 6.19$, $b = .28$, $p < .016$, and SPPC Social Competence scale, $\Delta r^2 = .037$, $F(6, 55) = 4.18$, $b = -.19$, $p < .046$. The follow-up stepwise regression analysis revealed that only the SPPC Social Competence scale contributed uniquely to the prediction, $\Delta r^2 = .069$, $F(5, 55) = 4.59$, $b = -.36$, $p < .036$, after the control variables were entered.

The source of the interaction effect was determined using median splits to construct a between-subjects factor representing the four factorial combinations of high and low scores on the intake and follow-up residualized HPA reactivity scores. A time (intake vs. follow-up) × HPA reactivity pattern (high-high, low-high, high-low, vs. low-low HPA activation across time) mixed-model ANOVA was computed with the SPPC Social Competence subscale as the dependent variable. Simple effects tests indicated that self-perceived social competence for children who had HPA reactivity scores consistently *above* the medians at both intake and follow-up (high-high reactivity pattern, $N = 14$) decreased over the 6-month interval ($M = -1.93$, SD = 2.23) more than did the self-perceptions of children in either of the two situationally high reactivity groups (i.e., the low-high reactivity pattern, $M = 1.81$, SD = 3.58, $N = 16$, or high-low reactivity pattern, $M = 1.41$, SD = 4.20, $N = 17$, groups), Student Newman-Keuls Tests, $ps < .05$ (the mean was .40, SD = 3.02, for the low-low reactivity pattern group, $N = 15$). A planned contrast revealed that, on average, self-perceived social competence levels decreased for children in the high-high reactivity pattern group ($M = -1.93$, SD = 2.23) but increased for all other children ($M = 1.23$, SD = 3.63), $t(58) = 3.04$, $p < .004$.

Taken together, these results support the hypothesis that children's HPA stress-reactivity may predict behavioral and diagnostic aspects of their short-term psychological adjustment. They also highlight the possibility that considering the consistency across time in social challenge-related adrenocortical activation may help clarify the links between social environmental events and the continuity in children's self-perceived social competence. We now turn to a rival hypothesis.

Are Individual Differences in Children's Problem Behavior Profiles, or Patterns of Control-Related Beliefs, Mediators of Their Subsequent HPA Reactivity to Psychosocial Challenge?

These analyses used a linear regression model, that is, HPA $Reactivity^{t2} = a + b_0Cort^{t1} + b_1Age + b_2Gender + b_3Beh^{t2} + b_4Beh^{t1} + b_5(Beh^{t1} \times Beh^{t2})$, to predict HPA reactivity scores at the 6-month follow-up from the measures of psychological adjustment assessed during the intake and follow-up interviews. In the model, the dependent variable (i.e., "HPA $Reactivity^{t2}$") was the HPA reactivity score measured at follow-up (i.e., $Cort^{t2}$ in the previous analyses); "$Cort^{t1}$" represented the level of HPA reactivity at the intake interview; "b_3" and "b_4" estimated the main effects of follow-up and intake adjustment scores; and "b_5" estimated the interaction between intake and

follow-up adjustment scores. The interaction term "($Beh^{t1} \times Beh^{t2}$)" was included to examine whether patterns of adjustment (e.g., consistently high, consistently low, or situationally high problem levels) were differentially associated with subsequent HPA reactivity.

As in the preceding section, these analyses were conducted using a conservative two-step approach. First, separate equations predicted follow-up HPA reactivity scores from each of the outcome measures, and (using b and the incremental Δr^2) the unique contributions of the main and interactive effects of the psychological measures were interpreted.

When more than one main (i.e., Beh^{t1} or Beh^{t2}) or interaction effect (i.e., $Beh^{t1} \times Beh^{t2}$) predicted follow-up HPA reactivity, the follow-up HPA reactivity scores were regressed on the set of terms representing effects of the different behavior and adjustment measures. Thus, paralleling the analyses described above, HPA reactivity was the criterion measure and the behavioral adjustment variables were the predictors. The first step enabled us to estimate the predictive validity of the adjustment scores independently of HPA reactivity during the initial assessment, as well as age and gender differences that might be associated with levels of, or changes across time in, the behavior and adjustment measures. As above, the second step ensured that the observed associations were not dependent on intercorrelations among the behavior and adjustment measures.

Main effects of initial (Beh^{t1}) and follow-up (Beh^{t2}) levels of adjustment.—Noticeably absent were any significant associations when predictions were made from diagnostic or behavior problem scores, patterns of control-related beliefs, and task behavior and affect ratings assessed at intake to levels of HPA reactivity at follow-up.

Patterns of adjustment across time ($Beh^{t1} \times Beh^{t2}$).—Separate equations revealed that the pattern of children's problem behavior (i.e., the $Beh^{t1} \times Beh^{t2}$ interaction term) over time measured by the cross-informant Internalizing, $\Delta r^2 = .158$, $F(6, 57) = 12.38$, $b = .43$, $p < .001$, Anxiety/Depression, $\Delta r^2 = .134$, $F(6, 57) = 10.34$, $b = .38$, $p < .002$, and Somatic Complaint scales, $\Delta r^2 = .103$, $F(6, 57) = 7.51$, $b = .40$, $p < .008$, as well as the SASC self-report Social Anxiety Scale, $\Delta r^2 = .058$, $F(6, 55) = 3.90$, $b = .76$, $p < .053$, predicted HPA reactivity at follow-up. When the follow-up HPA reactivity measure was regressed (using stepwise procedures after controlling for age, gender, $cort^{t1}$, and the respective Beh^{t1} and Beh^{t2} terms for each of these behavior and adjustment measures) on the four interaction terms, only the cross-informant internalizing behavior problem summary scale contributed uniquely to the prediction, $\Delta r^2 = .22$, $F(12, 49) = 18.41$, $b = .56$, $p < .0001$.

Using a median split procedure parallel to that described above, a time (intake vs. follow-up) × adjustment pattern (high-high, low-high, high-low, vs. low-low levels of internalizing behavior problems across time) mixed-model ANOVA was computed with the residualized cortisol scores as the dependent measure. Simple effects tests revealed, that in contrast to children in the high-low adjustment group (i.e., those who showed improving levels of internalizing problems) who had lower average levels of HPA reactivity at follow-up than at intake ($M = -.08$, SD = .01, $N = 8$), the children in the low-high adjustment group (i.e., those who showed increasing levels of internalizing problems) had higher HPA reactivity scores 6 months later than they did at intake ($M = .06$, SD = .05, $N = 7$), Student Newman-Keuls Tests, $ps < .05$. A planned contrast revealed that in comparison to the other children ($M = -.01$, SD = .11), HPA reactivity levels increased for children in the low-high adjustment group ($M = .06$, SD = .05), $t(60) = 2.02$, $p < .047$.

Thus, in contrast to the equations predicting adjustment from HPA reactivity scores, there were no significant time-lagged associations detected when subsequent HPA reactivity scores were predicted from the initial assessments of psychological adjustment. However, when the *pattern* of psychological adjustment across time was considered, the results supported the hypothesis that increasing levels of internalizing behavior problems may affect children's subsequent HPA responsiveness to ecologically valid, developmentally salient social challenges.

Discussion

When time-lagged associations between adrenocortical reactivity and clinic-referred children's psychological adjustment were explored, a potentially important pattern was observed. First, adrenocortical activation in response to social challenge predicted the children's subsequent internaliz-

ing problem behavior and anxiety disorders 6 months later. Second, the consistency over time of children's adrenocortical reactivity to social challenge had meaningful implications for the stability of their self-perceived social competence. Third, noticeably absent was any evidence that individual differences in children's psychiatric diagnoses or control-related beliefs predicted their subsequent adrenocortical reactivity. Fourth, the short-term continuity (i.e., increases and decreases) in the levels of the children's internalizing behavior problems was linked with their subsequent adrenocortical responsiveness to social challenge. Finally, it is interesting that no measure of behavioral or psychological adjustment at intake contributed to the prediction of subsequent adrenocortical reactivity, independent of that predicted uniquely by, or in combination with, follow-up levels of adjustment.

One particularly significant aspect of these data is that they are among the first to suggest that individual differences in clinic-referred children's adrenocortical reactivity to parent-child conflict may affect psychological developments related to their short-term behavioral and emotional adjustment. Also, they indicate that it may be especially informative to consider *patterns* of adrenocortical reactivity (across time and situations) in determining the role that the psychobiology of the stress response plays in mediating the bidirectional effects among environmental, behavioral, and biological influences on individual development (Gottlieb, 1992). The findings have several additional implications.

Previous experimental studies with adult and animal populations suggest that the controllability of stimulation is linked to adrenocortical activity (Gunnar, Marvinney, et al., 1989; Levine, 1980). Evidence also supports the notion that the HPA stress response is affected more by the perception or expectation of control than by the actual "fact" of control (Weiss, 1971). Moreover, studies reveal that depressed adults may have increased sensitivity to uncontrollable stress and suggest that there may be important interrelations between the cognitive deficits of depression and heightened HPA reactivity in these patients (Breier, 1989). Our previous findings (Granger, Weisz, & Kauneckis, 1994) paralleled this pattern of observations. They supported the notion that perceived control was among the factors determining the impact of social stressors on children's HPA activity and that specific patterns of behavioral and emotional difficulties may be associated with increased psychobiological sensitivity to uncontrollable stress. The present findings extend those observations by suggesting that aspects of the biological systems regulating children's HPA activity may be sensitive predictors of subsequently lower levels of perceived social contingencies and competencies. These findings support the need for additional research aimed at experimentally evaluating the interconnections between children's control-related beliefs and adrenocortical function.

Also noteworthy is that these data support findings of previous studies with nonclinical populations of children that link socially inhibited behavioral styles (Kagan et al., 1987), and overcontrolled problem social behaviors (Granger, Stansbury, & Henker, 1994), to stress-related adrenocortical reactivity in social contexts. These results extend those previous observations by suggesting that the continuity of internalizing behavior problems in clinic-referred populations is closely associated with adrenocortical responsiveness to psychosocial challenge.

In our previous report, we found individual differences in adrenocortical reactivity to be associated with higher levels of social withdrawal, social anxiety, somatic complaints, and social problems. Based on that pattern of concurrent associations, we anticipated that the present findings might be helpful heuristically, particularly for researchers interested in relations among patterns of children's cognitions, behavioral coping responses, and the development of psychopathology. The present findings extend our ability to sort out the etiologic possibilities and assess their relative plausibility by suggesting the following developmental sequence. Perhaps initially the magnitude and duration of individual differences in HPA stress-related activation are closely tied to the intensity and/or frequency of negative environmental events (e.g., parent-child interactions characterized by conflict, hostility, or abuse) or the expression of internalizing behavior problems, or both. Over time the cognitive-behavioral response styles that characterize children with internalizing behavior problems may lead to more frequent or exaggerated adrenocortical responses to the social environment. It is tempting to speculate that adrenocortical activation is a key biological process that potentates the development of internalizing behavior problems into risk factors for more serious or extreme problems (i.e., anxiety

disorders). However, any conclusions at this point are speculative at best and must therefore be offered for heuristic purposes only.

Although the present study yielded some potentially important findings, several limitations qualify the interpretation of the data. Of course, without a nonreferred control group it is not possible to determine whether the observed pattern of correlates is specific to children with adjustment problems. Also without having measured cortisol levels in the absence of the psychosocially challenging task, we cannot be sure that the observed relations are not specific to the children's reactions to the interaction tasks. It is also true that we do not know how other factors that appear in the literature, such as eating disorders, ethnicity, and sexual abuse, may have influenced reactivity in these children. Another limitation concerns the lack of information about the parent partner in the interaction; it is possible that the children's HPA reactivity was moderated by parental behavior (Gunnar, 1993b) and psychopathology. This, in turn, suggests that our speculation, that children's adrenocortical activity is a mechanism by which psychosocial experiences are translated into individual differences in behavior problems, may be askance. That is, adrenocortical activation may only be a surrogate marker or byproduct of the actual mechanism. Finally, it is unclear as to how these findings may have been influenced by the effects of the specific treatments these children experienced during the 6-month period.

Further investigations of the time-lagged associations among clinic-referred children's HPA reactivity, behavior, and cognitions in quasinaturalistic and everyday social contexts are needed to replicate and elaborate the implications of these data. In particular, four avenues of research seem fruitful. First, naturalistic and analog studies that assess the cross-situational consistency of individual differences in children's HPA reactivity seem justified. Second, studies that extend the present study's short-term longitudinal focus are needed. Third, children at high risk for developing behavioral and emotional problems would seem to be an appropriate population to include in future studies. Finally, further study is needed to explore the potential links among individual differences in adrenocortical reactivity, patterns of deviant behavior, and immune activity in children (Granger, Ikeda, Schmeelk, & Block, 1996).

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