Journal of Abnormal Psychology
2006, Vol. 115, No. 1, 174–178

0021-843X/06/$12.00 DOI: 10.1037/0021-843X.115.1.174

BRIEF REPORTS

Autonomic Correlates of Attention-Deficit/Hyperactivity Disorder and Oppositional Defiant Disorder in Preschool Children

Sheila E. Crowell, Theodore P. Beauchaine, Lisa Gatzke-Kopp, Patrick Sylvers, Hilary Mead, and Jane Chipman-Chacon
University of Washington

Numerous studies have revealed autonomic underarousal in conduct-disordered adolescents and antisocial adults. It is unknown, however, whether similar autonomic markers are present in at-risk preschoolers. In this study, the authors compared autonomic profiles of 4- to 6-year-old children with attention-deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD; $n = 18$) with those of age-matched controls ($n = 20$). Children with ADHD and ODD exhibited fewer electrodermal responses and lengthened cardiac preejection periods at baseline and during reward. Although group differences were not found in baseline respiratory sinus arrhythmia, heart rate changes among ADHD and ODD participants were mediated exclusively by parasympathetic withdrawal, with no independent sympathetic contribution. Heart rate changes among controls were mediated by both autonomic branches. These results suggest that at-risk preschoolers are autonomically similar to older externalizing children.

Keywords: ADHD, conduct disorder, autonomic responding

Preschool symptoms of attention-deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD) mark significant risk for more serious externalizing behaviors in middle childhood, including early onset conduct disorder (CD), delinquency, and aggression (Campbell, Shaw, & Gilliom, 2000). In turn, children with early onset CD and ADHD are at risk for persistent criminality and antisocial behavior in adulthood (Lynam, 1996; Moffitt, 1993).

Both CD in childhood and antisocial behavior in adulthood are marked by autonomic underarousal, including reduced electrodermal responding (EDR) and heart rate (HR; see McBurnett & Lahey, 1994; Scarpa & Raine, 1997). Moreover, research with both elementary schoolchildren and adolescents has revealed reduced sympathetic- and parasympathetic-linked cardiac activity among probands with conduct problems (Beauchaine, 2003; Beauchaine, Katkin, Strassberg, & Snarr, 2001; Boyce et al., 2001). Individual contributions of each autonomic branch to HR may have unique implications. Parasympathetic nervous system (PNS)-linked cardiac activity has been associated with emotion regulation capabilities (Porges, 1995), whereas deficiencies in sympathetic nervous system (SNS)-linked cardiac activity have been associated with reward insensitivity (Beauchaine et al., 2001; Beauchaine, Gatzke-Kopp, & Mead, in press).

Sheila E. Crowell, Theodore P. Beauchaine, Lisa Gatzke-Kopp, Patrick Sylvers, Hilary Mead, and Jane Chipman-Chacon, Department of Psychology, University of Washington.

This work was supported in part by Grant MH63699 from the National Institute of Mental Health to Theodore P. Beauchaine. We thank Sharon Brenner for her helpful contributions.

Correspondence concerning this article should be addressed to Theodore P. Beauchaine, Department of Psychology, University of Washington, Box 351525, Seattle, WA 98195-1525. E-mail: tbeaucha@u.washington.edu

Although EDR and cardiac activity and reactivity have been linked to temperament and empathy in normative samples of preschoolers (e.g., Fabes, Eisenberg, Karbon, Troyer, & Switzer, 1994; Fowles, Kochanska, & Murray, 2000), associations between autonomic functioning and externalizing psychopathology have not been explored in preschool children at risk for CD by virtue of an ADHD and ODD diagnostic status. Doing so is important because the preschool years may represent a critical period during which developing noradrenergic, serotonergic, and dopaminergic systems that govern behavioral control are vulnerable to long-term changes in functioning (e.g., Bremner & Vermetten, 2001; Pine et al., 1996). Attenuated EDR, which marks both serotonergic and noradrenergic dysregulation (Beauchaine, 2001), is a consistent marker of antisocial behavior across development. Recently, Raine et al. (2001) reported that an enriched preschool environment that included instruction for teachers in behavioral management and weekly counseling sessions for parents conferred a 61% increase in electrodermal activity on children 6–8 years later compared with controls who were assigned randomly to a no-treatment condition. These data suggest that long-term changes in biological systems implicated in impulsive and aggressive behavior can be effected through early intervention. It remains to be determined whether such effects obtain among children at risk for delinquency. Identifying autonomic markers of ADHD and ODD represents an initial step toward addressing this question.

In the present study, we compare a normal control group of preschoolers with a group at high risk for CD on a number of autonomic measures. The following hypotheses were explored. First, we predicted reduced baseline EDR in children with ADHD and ODD, consistent with data from older antisocial groups. Second, we hypothesized that the children in the ADHD and ODD group would exhibit (a) attenuated SNS-linked cardiac activity at

Table 1
Demographics, Child Behavior Checklist Scores, and Child Symptom Inventory Scores by Group

Variable	ADHD and ODD (n = 18)		Control (n = 20)			
	M	*SD*	*M*	*SD*	$F(1, 36)$	η^2
Age (years)	4.78	.73	4.55	.60	1.1	.03
Annual family income	$48,000	$40,000	$55,000	$37,300	1.1	.03
ADHD symptoms						
CSI Hyperactivity scale score	20.6	3.9	4.4	2.8	218.0	.86
No. of symptoms endorsed	6.2	1.7	0.5	0.8	183.1	.84
CSI Inattention scale score	17.6	5.7	4.0	2.7	90.2	.71
No. of symptoms endorsed	5.8	2.4	0.2	0.7	102.4	.74
CBCL Attention Problems subscale	74.8	7.6	51.3	2.1	174.35	.83
ODD symptoms						
CSI Oppositional Defiant scale score	14.1	3.9	3.4	1.7	123.6	.77
No. of symptoms endorsed	5.4	1.9	0.1	0.3	147.1	.80
CBCL Aggression subscale	78.5	8.1	51.0	1.7	218.1	.86

Note. All group differences are significant at $p < .001$. CSI scale scores represent the sum of dimensionalized symptoms (0 = *never* through 3 = *very often*). CSI symptom counts include the number of scale items endorsed at a level of 2 or higher (Gadow & Sprafkin, 1997). CBCL data are expressed in *T* scores. ADHD = attention-deficit/hyperactivity disorder; ODD = oppositional defiant disorder; CSI = Child Symptom Inventory; CBCL = Child Behavior Checklist.

baseline and (b) less SNS-linked cardiac reactivity during a reward task. Finally, we predicted attenuated PNS-linked cardiac activity among children with ADHD and ODD.

Method

Participants

After receiving institutional review board approval, we recruited 18 children with ADHD and ODD and 20 controls through ads placed in Seattle, Washington, newspapers and fliers posted at local preschools. All participating children were between the ages of 4 and 6 years, with 9 girls and 11 boys in the control group and 7 girls and 11 boys in the ADHD and ODD group, $\chi^2(1, N = 38) = 0.15, p > .70$. The sample included 2 African American, 2 Asian American, 27 Caucasian, and 2 Latino children. The remaining 5 participants were of mixed racial and/or ethnic heritage. Additional descriptive statistics are provided in Table 1.

Parents who responded to our ads (n = 67) completed a computerized structured telephone interview administered by a trained research assistant. This interview lasted for about 20 min and incorporated portions of the Child Symptom Inventory (CSI; Gadow & Sprafkin, 1997) and the Child Behavior Checklist (CBCL; Achenbach, 1991). The CSI provides dimensional scores and diagnostic cutoffs for several *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 2000) syndromes. Each symptom is assessed on a 4-point scale (0 = *never*, 1 = *sometimes*, 2 = *often*, 3 = *very often*), with a rating of 2 or higher considered positive for a diagnostic criterion. Parents completed the ADHD and ODD scales of the CSI. Parents also completed the Aggression, Attention Problems, and Anxious/Depressed subscales of the CBCL. Families were included in the experimental group if their child met *DSM–IV* criteria for ODD and ADHD (hyperactive-impulsive or combined subtype) and scored at or above the 98th percentile on the Attention Problems and Aggression subscales of the CBCL. Control group children were required to score below the 60th percentile on the CBCL scales and to exhibit no more than two symptoms of ADHD or ODD. Participants were excluded from both groups if they scored above the 60th percentile on the CBCL Anxious/Depressed subscale, because children with comorbid depression do not exhibit the same autonomic deficiencies as do children without internalizing symptoms.[1] Interviews were scored by computer, and parents of children who met inclusion criteria for either group were invited for a lab visit. A $75 monetary incentive was provided to the parent, in addition to a $10 toy reward for the participant child (see below). None of the recruited children were receiving stimulants. Scores on the CBCL and the CSI are reported in Table 1. Among the ADHD participants, 4 met criteria for the hyperactive–impulsive subtype, and 14 met criteria for the combined subtype. Two potential ADHD participants were rejected due to elevated anxiety/depression scores, and 4 were rejected because they did not meet ODD criteria and/or did not score above the 98th percentile on the CBCL Aggression subscale.

Procedure

The 30-min protocol was conducted in a sound-attenuated room that we monitored with audio and video equipment. Patterns of cardiac and electrodermal activity were measured during a 5-min baseline, after which children played Perfection, a commercially available game designed for 4- to 7-year-olds that was used to elicit psychophysiological response patterns during reward. Children were required to place a number of shapes (e.g., stars, triangles) into corresponding holes before a spring-loaded platform ejected the pieces after 1 min. To increase the reward value of the game, we showed the children a large container of toys valued at about $10 each and told them that they could choose their favorite toy on successful completion of the game. After the game, all were given their chosen toy for "trying hard." Parents waited in the adjacent room with the experimenter.

Psychophysiological Measures

EDR. Skin conductance was recorded using a Grass 15LT Physiodata Amplifier System and a 15A12 DC amplifier (West Warwick, RI). The

[1] Although few studies have examined relations between autonomic responding and comorbid internalizing symptoms among children with disruptive behavior disorders, we reported greater heart rate variability among 5- to 12-year-old inpatients with both CD and depression compared with their CD-only counterparts (Beauchaine, Gartner, & Hagen, 2000). For this reason, ADHD and ODD children with comorbid internalizing symptoms were excluded from the present study.

Table 2
Descriptive Statistics and Analysis of Variance Results for Psychophysiological Measures Collected at Baseline and During Perfection

	ADHD and ODD		Control		Analysis of variance			
Measure	*M*	*SD*	*M*	*SD*	Effect	F[a]	p	η^2
Baseline								
EDR	2.30	1.91	4.25	2.12	Group	10.27	.003	.23
					Epoch	1.25	.27	.03
					Group × Epoch	0.83	.37	.02
PEP	85.62	22.11	71.50	14.92	Group	5.77	.02	.14
					Epoch	0.77	.39	.02
					Group × Epoch	0.49	.49	.01
RSA	6.68	1.26	6.73	1.36	Group	0.02	.89	.00
					Epoch	1.72	.20	.05
					Group × Epoch	0.16	.70	.00
Perfection								
PEP	89.27	14.12	69.95	14.92	Group	14.93	<.001	.30
					Epoch	0.99	.33	.03
					Group × Epoch	1.05	.31	.03
RSA	5.79	1.28	6.08	1.22	Group	0.53	.47	.01
					Epoch	7.08	.01	.17
					Group × Epoch	0.29	.59	.01

Note. All reported means and standard deviations are averages across the two 30-s baseline and Perfection epochs. EDR = electrodermal responding (nonspecific fluctuations); PEP = pre-ejection period (ms); RSA = respiratory sinus arrhythmia (log[beats/min^2/Hz]); ADHD = attention-deficit/hyperactivity disorder; ODD = oppositional defiant disorder.
[a] The degrees of freedom for the EDR F tests were 1, 35. For all remaining F tests, the degrees of freedom were 1, 36.

EDR signal was collected through two 0.8 cm^2 Ag-AgCl electrodes adhered to the thenar eminence of the child's nondominant hand. Parker Labs Signa Gel (Fairfield, NJ) was used as a medium. Nonspecific fluctuations in skin conductance responses (SCRs) exceeding 0.05 μS were coded by a trained research assistant using Grass PolyVIEW software.

Cardiac preejection period (PEP). Sympathetic-linked cardiac activity was indexed by PEP, or the time elapsed between the electrocardiographic (ECG) Q wave and the impedance cardiographic (ICG) B wave. The validity of PEP as an index of sympathetic-linked cardiac activity has been established via pharmacologic (β-adrenergic) blockade (see Sherwood et al., 1990). ECG and ICG signals were recorded using Bio-Impedance Technology's AIM-8-V3 ambulatory impedance cardiograph (Chapel Hill, NC). The AIM-8 detects gross body movements, which can later be controlled statistically. Waveforms were collected using a spot electrode configuration (Qu, Zhang, Webster, & Tompkins, 1986). PEP data were ensemble averaged in 30-s epochs using COP-WIN 5.05-H software (Bio-Impedance Technology, Inc., n.d.).

Respiratory sinus arrhythmia (RSA). Parasympathetic-linked cardiac activity was assessed via spectral analysis, using a software package developed by Richard Sloan and colleagues at Columbia University. Spectral analysis decomposes the heart rate variability time series into component frequencies using fast-Fourier transformations. High-frequency spectral power (>0.15 Hz) was extracted to measure RSA. The validity of RSA as an index of parasympathetic-linked cardiac activity has been established via pharmacologic (cholinergic) blockade (see Berntson et al., 1997). Spectral densities were calculated in 30-s epochs and normalized using log transformations.[2]

Results

Prior to calculating inferential statistics, we evaluated the distributions of all physiological variables to ensure that (a) analysis of variance (ANOVA) assumptions were met and (b) outliers would not affect analyses disproportionately. All skew (range = −.33 to .34) and kurtosis (range = −1.1 to .01) values were acceptable. Variables were then standardized and z scores were examined. Because the most extreme z score was 2.50, all raw data points were retained for inferential analyses.

Baseline Responding

Mean SCRs, PEPs, and spectral densities were calculated for the final 1 min of the 5-min baseline (two epochs total). Differences between ADHD and ODD participants and controls were assessed using separate 2 (group) × 2 (epoch) repeated-measures ANOVAs for each physiological measure. Analyzing both the baseline and the task data in 30-s epochs allowed us to examine trends in responding within conditions. Analysis of gross body movements revealed no group difference, $F(1, 36) = 0.03$, $p > .86$, $\eta^2 < .01$. Thus, all comparisons at baseline were conducted without controlling for movement.

Descriptive statistics and group main effects for each psychophysiological measure at baseline appear in the top portion of Table 2. Because few epoch effects or Group × Epoch interactions were significant, reported means and standard deviations are av-

[2] Log transformations are typically used to normalize spectral analytic data and were not applied to correct for any distributional anomalies specific to this study. Respiration frequency data were also collected and scored for possible statistical control in calculations of RSA. Because no group difference in respiration rate was found, these data are not reported.

eraged across epochs to conserve space. Because of equipment failure, baseline EDRs were missing for 1 control participant. As shown in Table 2, ADHD and ODD participants exhibited significantly fewer baseline SCRs than controls did. Analyses of PEP data yielded a similar effect, with ADHD and ODD participants exhibiting longer PEPs than controls did. In contrast, no group difference was observed for RSA.

Perfection Task

Mean PEPs and spectral densities during the 1-min reward task were calculated within two 30-s epochs. Group differences were again assessed using a 2 (group) × 2 (epoch) repeated-measures ANOVA. Once again, no difference in body movement was observed across groups, $F(1, 36) = 0.06$, $p > .82$, $\eta^2 < .01$. Thus, all comparisons during play of Perfection were conducted without controlling for movement.

Group main effects during play of Perfection are reported in the bottom portion of Table 2. As with the baseline data, ADHD and ODD participants exhibited longer PEPs than controls did, yet no group effect was observed for RSA. Nevertheless, a significant epoch effect indicated reduced RSA for both groups during the second half of Perfection play compared with the first half, $F(1, 36) = 7.08$, $p = .01$, $\eta^2 = .16$. No other epoch effects or Group × Epoch interactions were significant.

SNS and PNS Contributions to HR Change During Reward

To evaluate the independent contributions of the SNS and PNS to HR change during reward, we first assessed differences in HR between baseline and Perfection play using a 2 (group) × 2 (baseline, Perfection) repeated-measures ANOVA. For this analysis, HR was averaged across the two baseline and the two game epochs. A significant trial effect indicated HR accelerated during reward for both groups, $F(1, 36) = 14.28$, $p < .001$, $\eta^2 = .28$. However, no group difference in HR reactivity was observed, $F(1, 36) = 0.11$, $p > .75$, $\eta^2 < .28$. Averaged across epochs, HRs at baseline and during reward were 100.84 ± 13.48 beats/min and 105.35 ± 10.92 beats/min, respectively, for the ADHD and ODD group and 99.51 ± 9.94 beats/min and 104.25 ± 12.89 beats/min, respectively, for controls. Next, we examined partial correlation coefficients between HR change and both PEP change and RSA change for each group. These analyses enabled us to determine the independent contribution to HR change of the SNS while controlling for PNS influences and of the PNS while controlling for SNS influences. For the control group, both partial correlations were significant, indicating independent contributions to HR change from both the PNS, partial $r = -.46$, $p < .05$, and the SNS, partial $r = -.64$, $p < .01$. For ADHD and ODD participants, only the PNS made an independent contribution to HR change, partial $r = -.64$, $p < .01$; the SNS did not, partial $r = .08$, *ns*. The group difference in partial correlations describing SNS influences while controlling for PNS influences was significant, $p = .02$, two-tailed.

Discussion

In this study, we tested the general hypothesis that preschool children with ADHD and ODD would show patterns of autonomic responding similar to those observed in older antisocial groups. Three specific hypotheses were explored. The first, that EDR would be attenuated in the ADHD and ODD group, was supported. Reduced EDR therefore appears to mark impulsive and oppositional traits among preschoolers in the same manner it does among older children. In adolescent samples, we have shown that attenuated EDR accompanies hyperactive–impulsive ADHD even in the absence of comorbid oppositionality and conduct problems (Beauchaine et al., 2001). Thus, EDR may be a more specific marker of disinhibition than of oppositionality or aggression. Future studies of preschool children with pure ADHD will be required to confirm this in younger samples.

The second hypothesis, that SNS-linked cardiac activity would be attenuated in the ADHD and ODD group, was also supported. Children with ADHD and ODD exhibited lengthened PEPs at baseline and showed no independent SNS contribution to HR change during reward. We have argued elsewhere that during incentive conditions, SNS-linked cardiac activity serves as a peripheral marker of central nervous system reward sensitivity (Beauchaine, 2001; Beauchaine et al., 2001). The present findings are consistent with a reward insensitivity interpretation of ADHD and ODD, as are recent neuroimaging studies that have revealed underactivity in the striatum and its frontal projections among ADHD children with and without CD (e.g., Bush et al., 1999). These brain regions are rich in dopaminergic neurons and have long been implicated in reward processing. Children with hypoactive reward systems may engage in reward-seeking behavior to up-regulate chronically suppressed dopaminergic neurotransmission. It is interesting to note that reduced striatal activity among ADHD probands is largely normalized by methylphenidate administration (Vaidya et al., 1998).

The final hypothesis, that PNS-linked cardiac activity would be attenuated in the ADHD and ODD group, was not supported. Thus, an autonomic deficiency that has been observed in several studies of delinquent adolescents (e.g., Beauchaine et al., 2001; Mezzacappa et al., 1997) and has been linked to emotion dysregulation and lability was not observed in at-risk preschoolers. This may provide indirect support of theories suggesting that emotional lability is socialized within families of delinquent children and adults through repeated negative reinforcement of conflict (Snyder, Schrepferman, & St. Peter, 1997). If future longitudinal studies support this conjecture, it will confirm that autonomic nervous system response patterns are malleable in young children.

Overall, these results (a) support the general hypothesis that children with ADHD and ODD are autonomically similar to older antisocial groups and (b) partially replicate findings from our previous research with adolescents with ADHD and CD. Although these findings are intriguing, additional research addressing the autonomic substrates of impulsivity among children at risk for antisocial behavior is needed. Although the present study is consistent with the reward insensitivity hypothesis, alternative interpretations cannot be ruled out. Because most of the ADHD group met criteria for the inattentive subtype, inattention could also be responsible for the group differences observed. Moreover, although both theory and empirical findings suggest that SNS-linked cardiac activity serves as a peripheral marker of reward sensitivity (e.g., Brenner, Beauchaine, & Sylvers, 2005), the SNS differences observed between these two groups could have resulted from aspects of the task other than incentive. The Perfection task was

also novel and potentially frustrating, which could have contributed to the differential response patterns found. Although analogue tasks used with older children implicate reward conditions more specifically (e.g., Beauchaine et al., 2001), future studies should rule out the possibility that other task characteristics were responsible for the pattern of results found. One way to do this is to vary the size of rewards while holding other stimulus properties constant. The present study also excluded children with internalizing symptoms. Thus, our results only apply to preschool children without symptoms of depression and cannot be generalized to all children at risk for CD.

Finally, future research addressing endophenotypic risk for CD should also assess environmental risk factors and Biology × Environment interactions across time. It is known that impulsive children fare worse than nonimpulsive children in high-risk contexts (e.g., Lynam et al., 2000). Elucidating the specific mechanisms of such effects, both biological and social, will represent major scientific advancements.

References

Achenbach, T. M. (1991). *Manual for the Child Behavior Checklist/4–18 and 1991 profile*. Burlington: University of Vermont, Department of Psychiatry.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Beauchaine, T. P. (2001). Vagal tone, development, and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology, 13,* 183–214.

Beauchaine, T. P. (2003). Autonomic substrates of heart rate reactivity in adolescent males with conduct disorder and/or attention-deficit/hyperactivity disorder. In T. A. Prester (Ed.), *Psychology of adolescents* (pp. 133–145). New York: Nova Science.

Beauchaine, T. P., Gartner, J. G., & Hagen, B. (2000). Comorbid depression and heart rate variability as predictors of aggressive and hyperactive symptom responsiveness during inpatient treatment of conduct-disordered, ADHD boys. *Aggressive Behavior, 26,* 425–441.

Beauchaine, T. P., Gatzke-Kopp, L., & Mead, H. K. (in press). Polyvagal theory and developmental psychopathology: Emotion dysregulation and conduct problems from preschool to adolescence. *Biological Psychology.*

Beauchaine, T. P., Katkin, E. S., Strassberg, Z., & Snarr, J. (2001). Disinhibitory psychopathology in male adolescents: Discriminating conduct disorder from attention-deficit/hyperactivity disorder through concurrent assessment of multiple autonomic states. *Journal of Abnormal Psychology, 110,* 610–624.

Berntson, G. G., Bigger, T., Eckberg, D. L., Grossman, P., Kaufman, P. G., Malik, M., et al. (1997). Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiology, 34,* 623–648.

Bio-Impedance Technology, Inc. (n.d.). COP-WIN (Version 5.05-H) [Computer software]. Chapel Hill, NC: Author.

Boyce, W. T., Quas, J., Alkon, A., Smider, N. A., Essex, M. J., & Kupfer, D. J. (2001). Autonomic reactivity and psychopathology in middle childhood. *British Journal of Psychiatry, 179,* 144–150.

Bremner, J. D., & Vermetten, E. (2001). Stress and development: Behavioral and biological consequences. *Development and Psychopathology, 13,* 473–489.

Brenner, S. L., Beauchaine, T. P., & Sylvers, P. D. (2005). A comparison of psychophysiological and self-report measures of BAS and BIS activation. *Psychophysiology, 42,* 108–115.

Bush, G., Frazier, J. A., Rauch, S. L., Seidman, L. J., Whalen, P. J., Jenike, M. A., et al. (1999). Anterior cingulate cortex dysfunction in attention-deficit/hyperactivity disorder revealed by fMRI and the counting Stroop. *Biological Psychiatry, 45,* 1542–1552.

Campbell, S. B., Shaw, D. S., & Gilliom, M. (2000). Early externalizing behavior problems: Toddlers and preschoolers at risk for later maladjustment. *Development and Psychopathology, 12,* 467–488.

Fabes, R. A., Eisenberg, N., Karbon, M., Troyer, D., & Switzer, G. (1994). The relations of children's emotion regulation to their vicarious emotional responses and comforting behaviors. *Child Development, 65,* 1678–1693.

Fowles, D. C., Kochanska, G., & Murray, K. (2000). Electrodermal activity and temperament in preschool children. *Psychophysiology, 37,* 777–787.

Gadow, K. D., & Sprafkin, J. (1997). *Child Symptom Inventory–4 norms manual.* Stony Brook, New York: Checkmate Plus.

Lynam, D. R. (1996). The early identification of chronic offenders: Who is the fledgling psychopath? *Psychological Bulletin, 120,* 209–234.

Lynam, D. R., Caspi, A., Moffitt, T. E., Wikström, P. H., Loeber, R., & Novak, S. (2000). The interaction between impulsivity and neighborhood context on offending: The effects of impulsivity are stronger in poorer neighborhoods. *Journal of Abnormal Psychology, 109,* 563–574.

McBurnett, K., & Lahey, B. B. (1994). Psychophysiological and neuroendocrine correlates of conduct disorder and antisocial behavior in children and adolescents. In D. C. Fowles, P. Sutker, & S. H. Goodman (Eds.), *Progress in experimental personality and psychopathology research* (pp. 199–231). New York: Springer.

Mezzacappa, E., Tremblay, R. E., Kindlon, D., Saul, J. P., Arseneault, L., Seguin, J., et al. (1997). Anxiety, antisocial behavior, and heart rate regulation in adolescent males. *Journal of Child Psychology and Psychiatry, 38,* 457–469.

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

Pine, D. S., Wasserman, G., Coplan, J., Fried, J., Huang, Y., Kassir, S., et al. (1996). Platelet serotonin 2A(5-HT_{2A}) receptor characteristics and parenting factors for boys at risk for delinquency: A preliminary report. *American Journal of Psychiatry, 153,* 538–544.

Porges, S. W. (1995). Orienting in a defensive world: Mammalian modifications of our evolutionary heritage: A polyvagal theory. *Psychophysiology, 32,* 301–318.

Qu, M., Zhang, Y., Webster, J. G., & Tompkins, W. J. (1986). Motion artifact from spot and band electrodes during impedance cardiography. *Transactions on Biomedical Engineering, 11,* 1029–1036.

Raine, A., Venables, P. H., Dalais, C., Mellingen, K., Reynolds, C., & Mednick, S. A. (2001). Early educational and health enrichment at age 3–5 years is associated with increased autonomic and central nervous system arousal and orienting at age 11 years: Evidence from the Mauritius Child Health Project. *Psychophysiology, 38,* 254–266.

Scarpa, A., & Raine, A. (1997). Psychophysiology of anger and violent behavior. *Psychiatric Clinics of North America, 20,* 375–394.

Sherwood, A., Allen, M. T., Fahrenberg, J., Kelsey, R. M., Lovallo, W. R., & van Doornen, L. J. P. (1990). Committee report: Methodological guidelines for impedance cardiography. *Psychophysiology, 27,* 1–23.

Snyder, J., Schrepferman, L., & St. Peter, C. (1997). Origins of antisocial behavior: Negative reinforcement and affect dysregulation of behavior as socialization mechanisms in family interaction. *Behavior Modification, 21,* 187–215.

Vaidya, C., Austin, G., Kirkorian, G., Ridlehuber, H., Desmond, J., Glover, G., & Gabrieli, J. (1998, November 24). Selective effects of methylphenidate in attention deficit hyperactivity disorders: A functional magnetic resonance study. *Proceedings of the National Academy of Sciences, USA, 95,* 14494–14499.

Received January 14, 2004
Revision received October 17, 2004
Accepted January 24, 2005 ■