

Aggression at Age 5 as a Function of Prenatal Exposure to Cocaine, Gender, and Environmental Risk

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Objective To examine childhood aggression at age 5 in a multiple risk model that includes cocaine exposure, environmental risk, and gender as predictors. **Methods** Aggression was assessed in 206 children by using multiple methods including teacher report, parent report, child's response to hypothetical provocations, and child's observed behavior. Also examined was a composite score that reflected high aggression across contexts. **Results** Multiple regression analyses indicated that a significant amount of variance in each of the aggression measures and the composite was explained by the predictors. The variables that were independently related differed depending on the outcome. Cocaine exposure, gender, and environmental risk were all related to the composite aggression score. **Conclusions** Cocaine exposure, being male, and a high-risk environment were all predictive of aggressive behavior at 5 years. It is this group of exposed boys at high environmental risk that is most likely to show continued aggression over time.

Key words aggression; cocaine exposure; environmental risk.

Childhood aggression can be a serious problem particularly among children who exhibit aggression across time and contexts (Campbell, Shaw, & Gilliom, 2000) as they are more likely to exhibit future juvenile delinquency (Kupersmidt & Coie, 1990; Roff, Sells, & Golden, 1972) and violent offending (Broidy et al., 2003; Farrington, 1994; Stattin & Magnusson, 1989). Studies of individual trajectories of physical aggression typically identify three or four groups of children, of which one shows consistently high aggression from early childhood to adolescence (Broidy et al.; Nagin & Tremblay, 1999). Researchers are seeking to understand the factors that contribute to such persistent high aggression.

In general, children who remain aggressive throughout childhood are more likely to experience multiple levels of risk that include a difficult temperament, poor parenting, a stressed family situation, and dangerous neighborhoods (Attar, Guerra, & Tolan, 1994; Cairns & Cairns, 2000; Campbell et al., 2000;

Dodge & Pettit, 2003; Kupersmidt, Griesler, DeRosier, Patterson, & Davis, 1995; Loeber & Wikström, 1993). They tend to be from large, single-parent families in which mothers have low education and occupational status, and poor social support (Dodge, Pettit, & Bates, 1994; Fox, Platz, & Bentley, 1995; Gagnon, Craig, Tremblay, Zhou, & Vitaro, 1995; Kupersmidt et al., 1995; Loeber, Tremblay, Gagnon, & Charlebois, 1989; Moffitt, 1990; Tremblay, Masse, Kurtz, & Vitaro, 1997). Specific parenting behaviors that have been implicated in contributing to aggression include poor monitoring, inadequate limit setting, and lack of warmth (Belsky, Woodworth, & Crnic, 1996; Campbell, Pierce, Moore, Marakovitz, & Newby, 1996; Patterson & Yoerger, 1997; Webster-Stratton & Hammond, 1998), although child characteristics (e.g., gender; hyperactivity-impulsivity) also have been implicated (Anderson & Bushman, 2002; Cairns & Cairns, 2000; Campbell, Pierce, March, Ewing, & Szumowski, 1994; Coie & Dodge,

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1997; Dodge & Pettit; Knight, Guthrie, Page, & Fabes, 2002; Loeber, 1988; Moffitt, Caspi, Dickson, Silva, & Stanton, 1996).

Prenatal exposure to cocaine is a candidate for a child level variable that may contribute to an aggression problem. Animal studies that control environmental factors suggest that cocaine exposure is related to higher aggression (Johns & Noonan, 1995; Johns et al., 1994; Wood & Spear, 1998). For example, gestationally exposed male rats that were cross-fostered at birth to untreated dams showed increased aggression to intruders as adults (Johns & Noonan). Several human studies have found increased externalizing problems in children who were prenatally exposed to cocaine (Delaney-Black et al., 2000; Griffith, Azuma, & Chasnoff, 1994; Hawley, Halle, Drasin, & Thomas, 1995; Yolton & Bolig, 1994). However research in children on the relations between biological factors such as autonomic reactivity or brain activity and externalizing problems suggests that the association is not direct, but rather mediated by underlying processes such as emotion regulation and impulse control (Campbell et al., 2000; Eisenberg & Fabes, 1992; Fox, Schmidt, Calkins, Rubin, & Coplan, 1996; Giancola, Martin, Tarter, Pelham, & Moss, 1996; Pennington & Bennetto, 1993; Séguin et al., 1995). This may explain in part the higher levels of aggression reported in cocaine-exposed children as several studies have found them to exhibit poorer emotional modulation (Bendersky & Lewis, 1998b, 2001; Mayes, Bornstein, Chawarska, Haynes, & Granger, 1996), impulse control (Bendersky & Lewis, 1998a; Bendersky, Gambini, La Stella, Bennett, & Lewis, 2003; Espy, Kaufmann, & Glisky, 1999), and attention abilities (Bandstra, Morrow, Anthony, Accornero, & Fried, 2001; Heffelfinger, Craft, White, & Shyken, 2002; Leech, Richardson, Goldschmidt, & Day, 1999; Mayes, Grillon, Granger, & Schottenfeld, 1998).

Additional evidence supporting the hypothesis that prenatal cocaine exposure is related to an aggressive behavioral tendency comes from neurophysiological studies. Aggressive behavior is associated with functional deficits in the basal-orbital prefrontal region (Blumer & Benson, 1975; Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994). This dopaminergic neuronal circuit underlies reactivity, arousal modulation, and attentional regulation. Because cocaine blocks the reuptake of monoaminergic neurotransmitters, it has been suggested that it disrupts the development of this pathway (Anderson-Brown, Slotkin, & Seidler, 1990; Dow-Edwards, 1991; Lidow, 1995; Mayes, 1999).

In addition to the possible biological effect of prenatal cocaine exposure on aggression, either directly, or

through its impact on emotional regulation and impulse control, many exposed children are also at increased environmental risk. Women who use cocaine have higher rates of depression (Woods, Behnke, Eyler, Conlon, & Wobie, 1995) and have been found to be less responsive in interactions with their children (Mayes et al., 1997). Many families in which cocaine, especially crack, is used are characterized by being headed by single, poorly educated women with high stress and little support living in impoverished neighborhoods (Bendersky, Alessandri, Gilbert, & Lewis, 1996; Woods et al., 1995). These conditions create a chaotic home environment that increases the risk of aggression through parenting, family and neighborhood level variables.

Being male also has been associated with increased physical aggression, especially the most violent kind (Anderson & Bushman, 2002; Cairns & Cairns, 2000; Coie & Dodge, 1997; Hoving, Wallace, & LaForme, 1979; Knight, Guthrie, Page, & Fabes, 2002). The male fetus may be more susceptible to intrauterine factors affecting the CNS (central nervous system) (Flannery & Liederman, 1994; Hynd & Semrud-Clikeman, 1989; Mathura, 1979; Montague, 1962; Weinberg, Zimmerberg, & Sonderegger, 1992). Evidence from animal research has suggested that particular mechanisms of effect of cocaine on the developing CNS may be more susceptible to damage in males (Glatt, Bolaños, Trksak, & Jackson, 2000; Markowski, Cox, & Weiss, 1998; Spear, 1995). One study found prenatally cocaine exposed adolescent male rats to show more aggression than females during a stressful competitive task (Wood & Spear, 1998). In humans there is evidence that boys living in stressful conditions, such as low income and single-parent households, have more social and behavioral problems than girls in the same environments (Elder, 1979; Hetherington, Camara, & Featherman, 1981). Therefore, there is a potential moderating effect of gender in the relation between cocaine and aggression. In fact, several studies have found boys to be more vulnerable to deficits related to cocaine exposure (Bendersky et al., 2003; Bennett, Bendersky, & Lewis, 2002; Delaney-Black et al., 2000), including externalizing problems. Specifically, Delaney-Black and colleagues found that boys who were prenatally exposed to cocaine were almost twice as likely as unexposed boys to have clinically significant levels of externalizing problems whereas, no effects of exposure were found for girls.

The measurement of aggression presents a number of problems. One problem is the poor agreement between different raters (Achenbach, McConaughy, & Howell, 1987; Hughes et al., 2002; Stanger & Lewis,

1993). Studies that have used parent report, teacher report, self-report, and observations of aggressive behavior have generally found only modest agreement across informants. For example, Dodge and Coie (1987) reported a correlation of .27 between teacher ratings of aggression and behavior observed during a peer interaction procedure. Low inter-rater agreement is likely in part due to the fact that children behave differently in different contexts (Achenbach et al.; Cairns & Cairns, 2000; Hoving et al., 1979; Lewis, 2002). Evidence suggests that it is the children who show increased aggression across different settings and observers that are at highest risk for continuing problems in adolescence (Campbell et al., 2000). These studies suggest that it is important to assess aggression in multiple contexts using multiple sources.

Given these prior findings, especially those implicating prenatal cocaine exposure, this study examined the effects of prenatal cocaine exposure and high environmental risk on aggression of boys and girls at 5 years of age. It was predicted that the impact of the three risk factors (cocaine exposure, being male, and living in high risk environments) would all contribute to high aggression consistent with a multirisk model. Multiple measures of aggression were used in this study including teacher report, parent report, child response to hypothetical provocations, and observation, to permit examination of factors associated with aggression in different contexts and to determine the profile of children exhibiting high aggression across contexts.

Method

Participants

The sample consisted of 206 children recruited for a longitudinal study of the effects of prenatal drug exposure and environmental risk on child development. Eighty three (40%) were exposed to cocaine in utero. Ninety nine (48%) were girls. Children were 5 years old ($M = 5.1$ years, $SD = 0.1$) at the time of the study. Mothers were predominantly African American (87%), with 10% Caucasian, 2% Hispanic, and 1% Asian. Pregnant women attending hospital-based prenatal clinics or who had just delivered at hospitals in low socioeconomic status (SES) areas of Trenton and Philadelphia were recruited. Eighty-two percent of those asked agreed to participate. Children were excluded from the study if they were born before 32 weeks of gestation, required special care or oxygen therapy for more than 24 h, exhibited congenital anomalies, were exposed to opiates or phencyclidine (PCP) in utero, or were born to mothers infected with HIV.

The sample represents 80% of 258 children who participated in the first lab visit at 4 months of age. Of the 52 families not seen at 5 years, 15 moved out of the area, 10 declined to participate, 18 could not be contacted, 1 child and 2 mothers died, and 6 children went to foster parents who declined to participate. There were no significant differences in the distributions of cocaine exposure, gender, perinatal medical risk, or environmental risk between children who participated and those who refused to continue or were lost to the study from the neonatal period through 5 years of age. Participation was voluntary, and incentives were provided in the form of vouchers for use at local stores. The study was approved by the Institutional Review Boards of the participating hospitals, as well as the medical school. Informed consent was obtained at the time of the study from the child's legal guardian.

Procedure

Children were brought to the laboratories where they were shown into a playroom by an examiner. The procedures were videotaped through a one-way mirror. Two child aggression procedures were used, the Reinisch Revision of the Leifer-Roberts Response Hierarchy (LRRH, Reinisch, 1981), and an observation of behavior with an inflated punching doll. Other tasks at this age assessed inhibitory motor control and executive functioning. The total session lasted about 1 hour. Caregivers remained in another room where they completed interviews and questionnaires. Questionnaires were mailed to kindergarten teachers in the early spring of the school year.

Measures

Prenatal Substance Exposure Risk

Prenatal substance exposure information was obtained through a semi-structured interview administered to the mother by trained interviewers within 2 weeks of the infant's birth. Interviews were conducted in an examination room at the prenatal clinic, in the mother's room on the maternity ward if she had just delivered, in our laboratories near the hospital, or in the woman's home. The drug use interview contained questions about the frequency, amount, and trimester of the mother's use of cocaine, alcohol, cigarettes, marijuana, opiates, PCP, tranquilizers, amphetamines, and barbiturates; the form of cocaine used; how disruptive substance abuse was to her life; and the history of her substance abuse. Substance interview information was confirmed by analysis of the newborn's meconium for the presence of benzoyl ecgonine (cocaine metabolite),

cannabinoids, opiates, amphetamines, and PCP using radioimmunoassay followed by confirmatory gas chromatography/mass spectrometry (Bendersky & Lewis, 1998b). Infants were considered cocaine exposed if they were positive by maternal report or radioimmunoassay. Infants classified as unexposed were negative by radioimmunoassay. There were no discrepancies in this sample between the results of the biological assay and maternal report of cocaine use. Maternal report of substance use was missing for 16 children because the biological mother was unavailable for interview but subjects were retained based on meconium analysis.

Environmental Risk Score

Demographic and lifestyle information was obtained through structured interviews administered to the mother when the children were 4½ years of age. These interviews included questions about the mother's race (non-European American = higher risk), educational achievement (number of years completed, reverse coded), single parenthood (living alone with children = higher risk), and public assistance status (public assistance as main source of income = higher risk). The number of caregivers (greater number = higher risk), irregularity of the child's schedule (15 items rated on a 4-point scale that assessed variability in when the child woke up, ate, went to sleep, etc., during the past week), and the instability of the child's surroundings (9 items assessing the number of changes in the room in which the child slept, who lived in the house, etc., during the past 6 months) were measured using the Family Chaos Scale (A. Sameroff, personal communication, September 1993). Maternal social support (the number of important or significant people in the mother's network, reverse scored) was measured with the Norbeck Social Support Questionnaire (Norbeck, Lindsey, & Carrieri, 1981). Maternal life stress was based on the Social Environment Inventory (Orr, James, & Casper, 1992) which asks about stressful events during the past 6 months (e.g., "you lost your job"). The variables were converted to standard scores, and summed to produce a cumulative risk score that was scaled to have a mean of 50 and a standard deviation of 10 (Bendersky & Lewis, 1998b). Such aggregate variables are more stable than any individual measure, and there is increased power to detect effects of the environment because errors of measurement decrease as scores are summed and degrees of freedom are preserved (Burchinal, Roberts, Hooper, & Zeisel, 2000; Wachs, 1991). This, and similar cumulative environmental risk measures have been found to explain more variance in children's outcomes including externalizing problems than single factors (Atzaba-Poria, Pike, &

Deater-Decker, 2004; Bendersky & Lewis, 1994, 1998b; Deater-Decker, Dodge, Bates, & Pettit, 1998; Sameroff, Seifer, Baldwin, & Baldwin, 1993; Sameroff, Seifer, Barocas, Zax, & Greenspan, 1987; Sameroff, Seifer, Zax, & Barocas, 1987; Stanton, McGee, & Silva, 1991).

Aggression Measures

Observed Aggressive Behavior. It is difficult to measure children's aggressive behavior directly because it does not occur very frequently. To obtain a reasonable sample the children were left in a room with a punching doll that invited aggressive behavior. A 4-foot tall, air-filled punching doll with a clown face and a large red nose (Bobo doll) was in the playroom along with a basket of age-appropriate toys. The examiner said to the child: "I will be back in 5 min, you can play with the toys while I'm gone." On the way out of the room she gently tapped the Bobo doll while the child watched (Homatidis & Konstantareas, 1981; Johnston, DeLuca, Murtaugh, & Drener, 1977). In the more standard "Bobo" doll procedure (Bandura, Ross, & Ross, 1961), the experimenter models aggressive behavior toward the doll. This has been criticized on the basis that the children are simply imitating the experimenter with no intent to harm (Tedeschi & Quigley, 1996). The adaptation of Homatidis and Konstantareas used in this study serves to draw the child's attention to the doll, but does not demonstrate intense aggressive behavior. The child's behavior was videotaped for 5 min after which the examiner returned to the room.

Videotapes of the punching doll procedure were coded for a variety of types of contact with the doll such as fistfisted hits to the face or body and kicks. Two coders independently scored 15 tapes. Interrater reliability was high. The intraclass correlations (ICC; Shrout & Fleiss, 1979) ranged from .73 to .99. The ICC reflects agreement between coders on both rank order and magnitude of the variable. Fistfisted and open hits to the face were behaviors of particular interest and had ICCs of .99 and .95, respectively. The coders were blind to the drug exposure status of the children.

There were two dimensions that characterized most contacts with the Bobo doll. One was the location of the contact, whether to the face or to the body. We considered hitting the face a more severe attack than hitting the body. The other dimension was type of hit, whether fistfisted or openhanded. We considered fistfisted hits more aggressive than openhanded hits. Four types of contacts can be generated by the combination of these dimensions: fistfisted hits to the face; fistfisted hits to the body; open hits to the face; and open hits to the body. These four categories constituted 83% of the contacts with the

punching doll. Behaviors such as kicking and jumping on the doll were of extremely low frequency. Of the four common behaviors, we considered fist hits to the face most indicative of aggressive intent. Children who were very active were more likely to punch and play with the Bobo doll than less physically active children. To reduce the potential confounding effect of activity level, the frequency of fist hits to the face was divided by the frequency of fist and open hits to the face. This proportion of hits to the face that were fist was the measure of the child's observed aggressive behavior. Children who did not hit the Bobo's face at all were assigned a value of 0.

Children's Own Aggression Rating. The LRRH, Reinisch Revision (Reinisch, 1981; Reinisch & Sanders, 1986), is a self-report measure of reactions to hypothetical interpersonal conflict situations. Four provocation scenarios were read to the child. These were common peer interaction situations that a child might encounter. They had been found in interviews to be irritating and moderately likely to elicit aggression in children between the ages of 4 and 16 (e.g., "You're standing in line for a drink of water. A kid comes along and just pushes you out of line. What do you do?"). The scenarios have both boy and girl protagonists to match the gender of the child. Following the reading of the scenario, the child was shown illustrations of two response choices and was asked to endorse one of the alternatives. Possible responses indicate physical aggression (e.g., push them), verbal aggression (e.g., yell at them), withdrawal (e.g., just go away), or nonaggressive coping (e.g., say 'That's all right'). The forced choice was repeated a total of 6 times so that every combination of response types was presented once and each response type was presented three times. The number of the child's responses of each type was summed across scenarios to yield 4 subscale scores ranging from 0 to 12. The instrument has been found to be valid and sensitive (Collins, 1973; Leifer & Roberts, 1972; Reinisch, 1981). As in previous research using this instrument, the physical aggression subscale was used as the measure of the aggressive response tendency for each child.

Mother's Aggression Rating. The mother or child's current guardian rated the child on 20 items that comprise the Aggression subscale of the Child Behavior Checklist for ages 4–18 (CBCL; Achenbach, 1991). Items are rated on 3-point scales from not true to very true. Examples of aggression items are: cruelty; bullying or meanness to others; disobedient; demands a lot of attention; screams; and gets into many fights. The sum of the ratings of these items constitutes the subscale score. The CBCL is a

widely used instrument with adequate psychometric properties.

Teacher's Aggression Rating. The kindergarten teacher rated the child on 6 questions comprising the Teacher Rating of Aggression (TRA; Dodge & Coie, 1987). Examples of questions are "The child threatens or bullies to get his own way" and "When a peer accidentally hurts the child, the child assumes the peer meant to do it then overreacts with anger/fighting." Items were rated on a 5-point scale from never true to always true. The sum of the ratings constitutes the aggression measure. The scale has been shown to have high validity and internal consistency (Dodge & Coie, 1987).

Composite Measure of Aggression

The correlations among the four measures of aggression were examined. The maternal report from the CBCL and the teacher report from the TRA were only modestly correlated ($r = .19, p < .05$). The proportion of facial hits that were fist in the Bobo doll observation was positively, but not significantly related to the teacher report of aggression ($r = .16, p < .10$), and unrelated to mother ($r = -.02$) and child ($r = -.06$) reports. The child's physical aggression score from the LRRH aggression scripts was not significantly related to the other aggression ratings ($r = .08, .13, \text{ and } -.06$ with CBCL, TRA and observation, respectively). To ensure that there were not nonlinear relations among the aggression variables each of the four measures of aggression was dichotomized into a high aggression group (top 25% of the distribution) and assigned a value of 1 for that measure, and a low aggression group (low 75% of the distribution) and assigned a value of 0 for that measure. It was possible that there was more consistency in the children observed to be highly aggressive. Chi-square analyses examined the correspondence between the proportion of children in the high and low aggression groups for each pair of aggression measures. Consistent with the correlational analyses, those rated high on the TRA by the teacher were also rated high on the CBCL by the mother. Out of 62 children rated high on aggression by either teacher or mother, 20 (32%) were considered high by both ($\chi^2(1 \text{ df}) = 12.36, p < .001$). Similarly, high teacher ratings were related to a greater proportion of fist hits in the observation with 27% (19/71) agreement on high aggression ($\chi^2(1 \text{ df}) = 3.63, p < .05$ 1-sided Fisher's Exact Test). These cross-tabulations indicate somewhat higher correspondence in the identification of highly aggressive children especially by mothers and teachers than the correlations suggest. There was no other

consistency in children rated high on aggression by different observers.

Because children who receive high rankings across a number of situations and observers are more likely to be at risk a composite aggression variable was computed. The dichotomized values (top 25% and low 75% on each measure) were summed for each child resulting in a variable that ranged from 0 to 4 indicating the number of different contexts in which the child was ranked relatively high on aggression.

Results

Perinatal and Demographic Characteristics

Table I presents perinatal and demographic characteristics by cocaine exposure group.¹ Children who were prenatally exposed to cocaine had a greater number of neonatal medical complications $t(204) = -3.65, p < .001$, as well as mothers who also used more alcohol $t(204) = 4.33, p < .001$, and smoked more cigarettes $t(204) = -6.69, p < .001$. The frequency of maternal use of substances other than these was extremely low.

Aggression as a Function of Exposure and Gender

Table II presents the means, standard deviations and cell sizes for each aggression measure for the total sample and broken down by gender, cocaine exposure, and their interaction. For all individual measures except the observed proportion of fisted hits to the punching doll's face, cocaine-exposed children were rated more aggressive than those who were unexposed. In addition, boys were rated more aggressive on every measure. Except in the punching doll situation, cocaine-exposed boys had the highest ratings. Similarly, for the composite measure, both cocaine exposure and being a boy resulted in being rated higher, with cocaine-exposed boys having the highest composite aggression score. The significance of these differences was examined using a cocaine exposure \times gender analysis of variance². Cocaine-exposed children chose significantly more physical aggression

¹Children exposed to cocaine were further classified into those whose mothers used cocaine less than twice per week on average (light exposure, $n = 26$) and those whose mothers used at least twice per week (heavy exposure, $n = 41$), definitions of light and heavy exposure that have been used in other studies (Bendersky & Lewis, 1998b; Jacobson & Jacobson, 1996). Use of the three-level variable instead of the dichotomous exposure variable (exposed vs. unexposed) in multiple regression analyses resulted in less significant findings for every outcome. In addition, there were no significant mean differences in these outcomes between the lightly and heavily exposed participants. Therefore the dichotomous variable was used in subsequent analyses.

responses to hypothetical provocations $F(1,186) = 5.37, p < .05$. Teachers rated boys significantly higher in aggression $F(1,164) = 6.37, p < .05$, and boys exhibited a higher proportion of fisted hits to the face of the punching doll $F(1,181) = 16.06, p < .001$. For the CBCL maternal aggression rating the differences were not significant. Examining the composite, both cocaine exposure $F(1,138) = 7.66, p < .01$ and gender $F(1,138) = 16.46, p < .001$ were related to the sum of high aggression ratings. The cocaine \times gender interaction was not significant for any of the individual measures or the composite score.

Aggression as a Function of Cocaine Exposure, Gender, Environmental Risk and Other Covariates

A series of multiple regression analyses was conducted to examine the independent and joint contributions of cocaine exposure, gender, and environmental risk to the variance of each of the aggression outcomes, while controlling for the possible effects of neonatal medical condition, and prenatal exposures to alcohol, cigarettes, and marijuana which differed for the different groups. Each aggression outcome was examined separately. Cocaine exposure, gender, environmental risk, neonatal medical condition, and other substance exposures were entered simultaneously in the first step of the analyses. Two- and three-way interaction terms between cocaine exposure, gender, and environmental risk were computed and added in a second step of the analyses. In no case did the second step contribute significant variance to the model and so the interactions are not considered further. Table III presents the standardized beta weights for each predictor and the overall model R^2 for each aggression outcome.³

The multiple regression analyses indicated that a significant amount of variance in each outcome was explained by this set of predictor variables, ranging from approximately 8–12.5% for the individual aggression outcomes, and about 22% for the composite sum of high aggression ratings. Examination of the independent contributions of the predictors indicated that the environmental risk score was related to all of the aggression measures except the proportion of fisted

²Mann-Whitney U non-parametric analyses for cocaine exposure and gender separately confirmed the parametric findings, that is both were significantly related to the sum of high aggression composite score ($U = 1803, p = .05$; $U = 1636, p = .001$, respectively).

³Results of the analysis of the sum of high aggression composite score using the Polytomous Logit Universal Models procedure in Statistical Package for the Social Services (SPSS) for ordinal level outcomes were substantively identical to those obtained using multiple linear regression.

Table I. Perinatal and Demographic Characteristics by Cocaine Exposure

Variable	Exposed (n = 83)	Unexposed (n = 123)	p
Medical complications score ^a (mean ± SD)	1.47 ± 2.56	0.58 ± 1.39	<.001
Prenatal exposure to alcohol (average number of drinks/day ^b) (mean ± SD)	1.42 ± 2.93	0.02 ± 0.12	<.001
Prenatal exposure to cigarettes (average number of cigarettes/day) (mean ± SD)	9.16 ± 10.11	1.33 ± 4.17	<.001
Prenatal exposure to marijuana (average number of joints/day) (mean ± SD)	0.27 ± 1.34	0.02 ± 0.18	<.10
Environmental risk score (mean ± SD)	50.56 ± 9.86	48.64 ± 9.41	ns
Percentage of boys (n)	47 (20)	55 (49)	ns

^aThe Neonatal Medical Complications Score is the sum of 35 possible complications (Hobel, Hyvarinen, Okada, & Oh, 1973) abstracted from hospital records.

^bA drink was defined as 1 oz. liquor, 4 oz. wine, or 12 oz. beer.

Table II. Aggression Outcomes by Gender and Cocaine Exposure

	Total sample			Unexposed			Exposed		
	Males	Females	Total	Males	Females	Total	Males	Females	Total
Child physical aggression responses (LRRH)	4.59 ± 3.01 (n = 94)	4.19 ± 2.88 (n = 93)	4.39 ± 2.94 (n = 187)	4.08 ± 3.15 (n = 61)	3.92 ± 3.03 (n = 50)	4.01 ± 3.09 (n = 111)	5.52 ± 2.51 (n = 33)	4.51 ± 2.69 (n = 43)	4.95 ± 2.65 (n = 76)
Teacher aggression rating (TRA)	7.36 ± 6.20 (n = 85)	5.29 ± 4.90 (n = 80)	6.36 ± 5.69 (n = 165)	6.86 ± 6.08 (n = 58)	5.12 ± 4.90 (n = 49)	6.07 ± 5.61 (n = 107)	8.44 ± 6.45 (n = 27)	5.55 ± 4.97 (n = 31)	6.90 ± 5.84 (n = 58)
Behavioral observation prop fist face hits	0.55 ± 0.47 (n = 92)	0.27 ± 0.42 (n = 90)	0.41 ± 0.47 (n = 182)	0.57 ± 0.47 (n = 59)	0.29 ± 0.43 (n = 48)	0.44 ± 0.47 (n = 107)	0.51 ± 0.49 (n = 33)	0.24 ± 0.42 (n = 42)	0.36 ± 0.47 (n = 75)
Maternal aggression rating (CBCL)	9.90 ± 8.11 (n = 94)	9.24 ± 6.79 (n = 93)	9.57 ± 7.47 (n = 187)	9.50 ± 8.00 (n = 62)	8.04 ± 5.70 (n = 50)	8.85 ± 7.07 (n = 112)	10.69 ± 8.39 (n = 32)	10.63 ± 7.71 (n = 43)	10.65 ± 7.95 (n = 75)
Composite aggression rating	1.49 ± 1.09 (n = 69)	0.87 ± 0.90 (n = 70)	1.18 ± 1.04 (n = 139)	1.33 ± 1.01 (n = 49)	0.70 ± 0.82 (n = 40)	1.04 ± 0.98 (n = 89)	1.90 ± 1.21 (n = 20)	1.10 ± 0.96 (n = 30)	1.42 ± 1.13 (n = 50)

Table III. Multiple Regressions Predicting Aggression Outcomes

	Child responses (LRRH)	Teacher rating (TRA)	Observation of facial hits	Maternal rating (CBCL)	Sum of high aggression ratings
Cocaine	.220**	.121	-.078	.046	.273***
Gender ^d	-.097	-.199***	-.294****	-.093	-.367****
Environmental risk	.199***	.142*	-.055	.219***	.216***
Alcohol exposure	-.062	.008	.115	.004	-.128
Cigarette exposure	-.070	-.124	.068	.088	-.055
Marijuana exposure	.052	.104	-.024	.128*	-.098
Neonatal medical risk	-.072	-.108	-.131*	.024	-.073
Model R ²	.083**	.086**	.126****	.094**	.219****

Values are standardized beta weights.

LRRH, Leifer-Roberts Response Hierarchy; TRA, Teacher Rating of Aggression; CBCL, Child Behavior Checklist.

^dGender was coded as 1 = boys, 2 = girls.

*p < .10. **p < .05. ***p < .01. ****p < .001.

hits to the face observed in the punching doll situation, such that the greater the environmental risk, the higher the aggression ratings. Gender was an independent predictor of teacher ratings, the observational measure, and the composite score with boys consistently ranked more aggressive than girls. Cocaine exposure was independently related to the child's aggressive responses to hypothetical provocations and the composite measure, with children who were exposed to cocaine having higher aggression scores.

None of the control variables was related significantly to the aggression outcomes.

Discussion

This study provides support for a multirisk model of aggression. Prenatal cocaine exposure, being a male, and high environmental risk were all related to higher aggression at age 5. Of note is that different variables emerged as being predictive depending upon how

aggression was measured. For the composite measure of high aggression across raters and situations all three factors were predictive.

A strength of this study is that aggression was measured using four different methods that included teacher and parent questionnaires, a child measure of response to hypothetical provocations, and an actual behavioral observation. Consistent with other reports in the literature (Achenbach et al., 1987; Hughes et al., 2002), there was only modest consistency among these different measures. However, our data indicate that for the most highly aggressive children there may be greater agreement as indicated by 32% overlap in children identified as higher in aggression by both mothers and teachers, and 27% overlap in those rated higher by teachers and observed to punch the doll more aggressively. Several possible reasons for low interobserver agreement can be offered. These include the context specific nature of behavior. For example, a child who exhibits a lot of fighting with siblings in the home may be much less aggressive at school with peers under a teacher's supervision. Another possible reason for inconsistency is that informants may have different standards for aggressive behavior, for example a teacher may be less tolerant of a child's tendency to hit and grab others than a mother, who may consider such behavior around the house just normal rambunctiousness. Furthermore, the aggression assessments used in this study each reflected somewhat different types of aggression. The teacher report asked about the child's bullying as well as the child's tendency to "fly off the handle," whereas, the mother was queried about behaviors indicating disobedience and attention seeking as well as physical aggression. The child report measured physical aggression reactions to hypothetical provocations, and the observation measured physical aggression directly but in a low provocation situation with a doll, and not a peer. However, despite these differences each measures some aspect of the construct "aggression," and there were children who were rated and/or observed to be relatively highly aggressive across the different methods. In light of the evidence that children who show aggression across settings are more likely to continue to have problems (Campbell et al., 2000), the aggregate measure of high aggression used in this study is more likely than any individual indicator to reveal children at risk for continued externalizing behavior problems into middle childhood and adolescence.

This study provides further support that prenatal exposure to cocaine constitutes a risk factor for aggressive behavior problems at school age (Delaney-Black et al., 2000). Moreover, the group at greatest risk appears

to be exposed boys who live in difficult environmental circumstances. While many theorists accept the multifactorial origins of aggressive behavior (Cairns & Cairns, 2000; Kupersmidt et al., 1995; Raine, 1993), this study highlights the additive nature of organismic and environmental risks.

There is theoretical and empirical support for the contributions of each of these risk factors to child aggression. Prenatal exposure to cocaine may be related because of its potential effects on the developing brain and the functions most likely affected, especially inhibitory control and emotional regulation (Anderson-Brown et al., 1990; Dow-Edwards, 1991; Lidow, 1995; Mayes, 1999). There are several other reports in the literature of an association between prenatal cocaine exposure and aggression (Chasnoff et al., 1998; Griffith et al., 1994; Hawley et al., 1995; Yolton & Bolig, 1994) or, more generally, externalizing behavior that includes delinquent acts along with aggressive behavior (Delaney-Black et al., 2000). However, not all studies have found such a relationship (Accornero, Morrow, Bandstra, Johnson, & Anthony, 2002; Azuma & Chasnoff, 1993; Bennett et al., 2002; Phelps, Wallace, & Bontrager, 1997; Richardson, 1998; Richardson, Conroy, & Day, 1996). One reason for such inconsistency may be that the samples have been recruited from different populations. For example, several of the samples in which positive results were found were recruited from drug treatment programs (Hawley et al.) or used children in foster care (Yolton & Bolig), both possible indicators of high environmental risk. This is opposed to studies with only light to moderate users (Richardson et al.) or a broader spectrum of exposure (Accornero et al.; Bennett et al.; Richardson) that suggests more variability in environmental risks such as stability of the home that characterized several of the studies that reported negative findings. Another possibility is that the effect of cocaine exposure on aggression appears to be modest, therefore studies that examine aggression in a single context (by using a single measure) are less likely to show significant group differences than those examining multiple measures across contexts. For example, we did not find a relation between cocaine exposure and externalizing behavior in the same cohort at 4 years of age (Bennett et al.) when the CBCL maternal report was the only measure of externalizing behavior examined despite high cross-age stability ($r = .67$ between 4 and 5 year CBCL aggression subscales).

This study, consistent with many others, also supports the contribution of male gender to the display of aggressive behavior (Anderson & Bushman, 2002;

Cairns & Cairns, 2000; Coie & Dodge, 1997; Hoving et al., 1979). The current study cannot determine whether effects are due to biology or socialization, and both may play a role. While the male fetus may be more susceptible to substances that affect CNS development than the female (Flannery & Liederman, 1994; Hynd & Semrud-Clikeman, 1989; Mathura, 1979; Montague, 1962; Weinberg et al., 1992), the evidence in this study does not suggest that cocaine affected the developing male CNS exclusively. More females who were exposed showed high aggression than unexposed females.

Consistent with the literature (Atzaba-Poria et al., 2004; Coie & Dodge, 1997; Dodge et al., 1994; Fox et al., 1995; Gagnon et al., 1995; Kupersmidt et al., 1995; Lindner, Hagan, & Brown, 1992; Loeber et al., 1989; Miller & Schouten, 1989; Tremblay et al., 1997; Wahler, 1980), environmental risk emerged as a predictor of several of the individual as well as the composite measure of aggression. Because the environmental risk score included a range of proximal and distal variables there are a number of ways in which it might impact aggressive behavior⁴. Mothers with high life stress, low social support, and few material resources may provide a parenting experience characterized by inconsistency of rule setting, harsh discipline, and low supervision, all found to relate to aggressive behavior in children (Belsky et al., 1996; Campbell et al., 1996; Patterson & Younger, 1997). There also may be variations in emotional availability of the parents that impact aggressive behavior. Increased externalizing behavior may be the child's bids for attention from a relatively emotionally remote parent (Shaw, Keenan, & Vondra, 1994). Such mechanisms were not explored explicitly in this study, but might be candidates for future research.

In conclusion, this study supports a multirisk model of early aggression. In particular, children who were rated, observed, or endorsed relatively high aggression, measured in different contexts by different reporters, were more likely to be cocaine-exposed, male, and living in high environmental risk environments. While consistent with the literature on the correlates of early aggression, this study underlines the unique additional risk conferred by in utero cocaine exposure. Intervention

⁴The individual components of the environmental risk score were not examined individually in relation to all of the aggression outcomes primarily because that would have entailed a large number of statistical comparisons. We were concerned about the need to conserve statistical power and limit chance findings. That is why we chose to use a cumulative risk score which, as discussed by others accomplishes this goal (Burchinal et al., 2000; Sameroff et al., 1993; Sameroff et al., 1987; Sameroff et al., 1987; Stanton et al., 1991; Wachs, 1991; and see page 20).

efforts might be most effectively aimed at this group of children in an effort to divert them from a trajectory of continued aggression and delinquency in later childhood and adolescence.

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