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Childhood Maltreatment and Prospectively Observed Quality of Early Care as Predictors of Antisocial Personality Disorder Features

Zhenyu Shi[1,2], **Jean-Francois Bureau**[1,3], **M. Ann Easterbrooks**[4], **Xudong Zhao**[2], and **Karlen Lyons-Ruth**[1]

[1] Department of Psychiatry, Harvard Medical School, Cambridge Hospital, USA

[2] Medical School, Tongji University, China

[3] School of Psychology, University of Ottawa, Canada

[4] Eliot-Pearson Department of Child Development, Tufts University, USA

Abstract

Few studies have evaluated the separate contributions of maltreatment and ongoing quality of parent-child interaction to the etiology of antisocial personality features using a prospective longitudinal design. 120 low-income young adults (aged 18-23) were assessed for extent of ASPD features on the Structured Clinical Interview for Diagnosis-Axis II, for presence of maltreatment on the Conflict Tactics Scale, Traumatic Experiences Scale, and Adult Attachment Interview, and for referral in infancy to parent-infant clinical services. Fifty-six of these families had been studied longitudinally since the first year of life. In infancy, attachment disorganization and disrupted mother-infant interaction were assessed; in middle childhood, disorganized-controlling attachment behaviors were reliably rated. In kindergarten and second grade, behavior problems were assessed by teacher report. In cross-sectional analyses, maltreatment was significantly associated with ASPD features but did not account for the independent effect of early referral to parent-infant services on ASPD features. In longitudinal analyses, maternal withdrawal in infancy predicted the extent of ASPD features twenty years later, independently of childhood abuse. In middle childhood, disorganized attachment behavior and maladaptive behavior at school added to prediction of later ASPD features. Antisocial features in young adulthood have precursors in the minute-to-minute process of parent-child interaction beginning in infancy.

Keywords

antisocial personality disorder; parent-child interaction; longitudinal studies; attachment; child abuse

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Antisocial personality disorder (ASPD), a pervasive pattern of disregard for the rights of others, has antecedents in childhood and adolescence and by adulthood is associated with an

Direct correspondence to: Karlen Lyons-Ruth, Department of Psychiatry, Harvard Medical School, Cambridge Hospital, 1493 Cambridge Street, Cambridge, MA 02139; Tel: (617)-547-3116; Fax: (617)-503-8470; klruth@hms.harvard.edu..

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array of negative life outcomes (American Psychiatric Association, 2000; Paris, 2003). A diagnosis of ASPD is defined by the presence of at least three of seven features, two of which refer to delinquent acts and physical assaults and five of which index deceitfulness, impulsivity, irresponsibility, and indifference to others. Thus, ASPD is not limited to aggressive or delinquent individuals but could also be displayed by irresponsible but non-aggressive individuals. ASPD is consistently found to be more frequent among males than among females (Moffitt, Caspi, Rutter, & Silva, 2001).

Many have argued for a dimensional rather than categorical approach to assessing personality disorders, due to increasing concern in personality disorder research that "using diagnosis as the measure can limit the information available from traits that are continuous in nature (p 189)" (Horwitz et al., 2001). Hence, both genetic and behavioral studies have used the number of antisocial features as an outcome variable, in addition to diagnostic classification (Kendler et al., 2008; Burt, McGue, Carter, & Iacono, 2007).

The developmental environment has been theoretically proposed to be relevant to the etiology of ASPD (Cohen, 2008). Large twin studies confirm the role of the environment in the development of ASPD, as well as the role of genetic factors. Using a dimensional measure of ASPD, Kendler et al. (2008) found that the environmental contribution to ASPD was 59.1%, while heritability was 40.1%. Another study, focusing on Cluster B personality disorders, which includes ASPD, demonstrated a similar but smaller environmental contribution (Torgersen et al., 2008). Burt, McGue, Carter, and Iacono (2007) also found that the environment played a role in the development of ASPD features. These studies document a general non-shared environmental contribution, but the specific components of the environment contributing to ASPD remain unclear. Identifying those factors comprises one key task of personality disorder research (Johnson, Cohen, Chen, Kasen, & Brook, 2006).

In contrast to the literature on antisocial behavior and delinquency, developmental models of the effects of the caregiving environment on the development of *antisocial personality disorder* have not been well-delineated. Longitudinal studies of ASPD features have focussed on identifying the sequence of symptomatic behaviors unfolding over time (e.g. Diamantopoulou, Verhulst, van der Ende, 2010) and have established that features of ASPD are preceded by symptoms of attention deficit hyperacitivity disorder and conduct problems. While early comorbid internalizing problems have been proposed to add to prediction of ASPD (Loeber et al., 2000), the additional contribution of internalizing problems was not supported in a large recent longitudinal study from ages 6 to 22 years, nor did earlier substance use increase prediction once earlier conduct problems were accounted for (Diamantopoulou et al., 2010).

In contrast to models of ASPD specifically, models of developmental processes relevant to early aggression, conduct disorder, and later delinquency have received a lot of attention (e.g. Loeber, Green, & Lahey, 2003; Lyons-Ruth, 1996; Moffitt, 1993; Patterson, DeBaryshe, & Ramsey, 1989; Shaw & Bell, 1993). In an integrative review of these studies of risk factors for early aggression and conduct disorder, DeKlyen and Greenberg (2008) propose that four risk domains have been shown to be relevant to the development of aggressive behavior problems: 1) child biological factors (reactive temperament, mild neurocognitive impairment), 2) quality of early attachment relationships (insecure or disorganized attachment patterns), 3) parental management strategies (harsh and ineffective discipline, lack of warmth), and 4) family ecology (low resources, low social support, high stress). Furthermore, these factors are likely to be additive or interactive with one another in producing antisocial disorders.We will first review the existing literature on developmental

antecedents of ASPD and then outline a potential model of the contributions of attachment relationships to ASPD.

Maltreatment in childhood has been repeatedly associated with antisocial behavior (Cohen, 2008). Studies have also confirmed this relation using diagnostic criteria for ASPD (Ducci et al., 2008; Horwitz, Widom, McLaughlin, & White, 2001). However, beyond the occurrence of maltreatment, few studies have evaluated the timing and range of caregiving disturbance associated with later ASPD, using observed interactions and prospective assessments. The few longitudinal studies including more subtle caregiving variables have covered a limited span of years from adolescence to young adulthood (Bullock and Dishion, 2007). In addition, antisocial behaviors are typically measured by non-diagnostic criteria (Cohen, 2008).

Two longer-term prospective studies from infancy have examined predictors of child or adolescent aggressive behavior. Aguilar, Sroufe, Egeland, and Carlson (2000) reported that maltreatment in early life predicted antisocial behavior at age 16 (assessed by the Child Behavior Check List, Achenbach, 1991), but mother-child interaction observed at age 13 did not predict antisocial behavior. Trentacosta and Shaw (2008) found that maternal hostile and controlling responses to toddler non-compliance predicted self-reported delinquency at 11 years. In one of the few studies using diagnostic criteria for ASPD, Horwitz et al. (2001) reported that low parental affection/nurturing, assessed by self-report at age 16, was associated with ASPD at age 22.

Thus, maltreatment and disrupted parent-child interaction reflect important facets of severely dysfunctional caregiving environments. However, previous work does not disentangle the effects associated with maltreatment from the effects of the disturbed day-to-day interactions that can occur in both abusive and non-abusive environments. The work reviewed above highlights the need to explore the contribution of the parent-child attachment relationship to the development of ASPD in adulthood, beginning early in life and, preferably, using prospective observational assessments (Hoffman, 2004).

DeKlyen and Greenberg (2008) suggest several processes through which insecure parent-child attachment relationships may influence the development of impulsive, aggressive behavior. A large literature identifies the child's primary caregiver as the person to whom the child turns for comfort and regulation of stressful arousal (Sroufe, Carlson, Levy, & Egeland, 1999; van IJzendoorn, Schuengel, & Bakermans-Kranenburg, 1999). The positive quality of this relationship is associated with reduced cortisol response after stressors such as inoculation while disorganized attachment relationships are associated with elevated cortisol (Hertsgaard, Gunnar, Erickson, & Nachmias, 1995). Randomized animal models have further established that quality of early care moderates the expression of genes related to stress reactivity (Francis, Diorio, Liu, & Meaney, 1999; Barr et al., 2004), and that stress in early life can alter noradrenalin, serotonin, and dopamine neurotransmitter systems and suppress hippocampal neurogenesis in ways that can persist into adulthood and may influence aggressive behaviors (Bremner & Narayan, 1998; Bennett et al., 2002). Thus, DeKlyen and Greenberg (2008) suggest that insecure early attachment relationships are likely to be associated with impaired ability to regulate stress and associated negative affect.

Secondly, disorganized attachment strategies in infancy have been associated with forms of controlling behavior toward the parent by school entry (e.g. Main & Cassidy, 1988). Controlling behaviors by the child are thought to represent compensatory attempts to elicit caregiver attention and involvement when more adaptive efforts have failed. Controlling behaviors associated with earlier disorganization have taken two forms: controlling caregiving behavior, characterized by the child's organizing, guiding, and providing positive

support to the parent, and controlling punitive behavior, characterized by directive and demeaning behavior toward the parent. In addition, some previously disorganized children seem unable to organize a controlling strategy and continue to show disorganized, out of context behavior in the presence of the parent into the school years (Bureau et al., 2009; Moss et al., 2004a,b).

Third, the representational models of the parent as unavailable and untrustworthy that develop as the child becomes more cognitively advanced (e.g. Main, Kaplan, & Cassidy, 1985) are thought to generalize over time to relations with others and contribute to the negative attributions found to characterize aggressive children's interpersonal perceptions (Dodge, Pettit, McClaskey, & Brown (1986). Finally, insecure attachment relationships have been related to increased non-compliance with the parent by preschool age (Greenberg, Speltz, & DeKlyen, 1993; Matas, Arend, & Sroufe, 1978), thus potentially contributing further toward a pathway in which aggressive behavior becomes more frequent and in which societal standards, as represented by the parent, are not identified with and internalized as components of one's own behavior.

To date, the contribution of security of attachment relationships has been tested in relation to aggressive behavior in early and middle childhood but not in relation to adult ASPD (Lyons-Ruth, Alpern, & Repacholi, 1993; Shaw, Owens, Vondra, Keenan, & Winslow, 1996). In the current work, we assess three of the four risk domains in the DeKlyen and Greenberg model (child biological factors were not assessed), which posits contributions of attachment security, parent-child interaction, and family socioeconomic stress to the prediction of ASPD features. To date, no model has attempted to assess separately the infancy and childhood components of the attachment relationship, nor to separate those from the effects of frank maltreatment, in the longitudinal prediction of ASPD features in adulthood.

The study to be reported evaluated the relation of ASPD features in young adulthood to the presence of childhood abuse and to the observed quality of parent-child interaction. Observed parent-child interaction was assessed prospectively in infancy and middle childhood, using measures grounded in the literature on disorganized and controlling attachment relationships. The young adult component of the study involved 120 young adults in the community who were evaluated retrospectively both for the presence of self-reported childhood maltreatment and for the presence of clinician concern about the quality of care in infancy. In addition, fifty-six of the 120 families seen in young adulthood had been participating in a longitudinal study since infancy. In this longitudinal subsample prospective measures of observed parent-child interaction were available in infancy and middle childhood, including security of infant attachment, quality of mother-infant communication, and disorganized/controlling attachment behaviors in middle childhood (age 8). Thus, the cross-sectional analyses were followed by longitudinal analyses of prospectively assessed predictors of ASPD features among the 56 families with longitudinal data.

Hypotheses

1. Childhood abuse and clinician concern about the quality of parent-child interaction in infancy will make separable contributions to the prediction of ASPD features in young adulthood.
2. These results will be specific to ASPD features and will not be accounted for by comorbid substance abuse or depression. Because ASPD is defined by difficulties in interpersonal relations to a greater extent than depression or substance abuse, we expected that childhood interaction patterns would be particularly relevant to ASPD.

3. In the longitudinal cohort, forms of disrupted maternal communication in infancy will predict adult ASPD features, independent of prediction from childhood abuse. Because a spectrum of disturbed parenting, including harsh discipline and lack of warmth and nurturance, has been associated with aggressive behavior in childhood, we did not have specific predictions as to which aspects of disrupted maternal communication would be most strongly associated with ASPD features.

4. Disorganized/controlling attachment behavior in middle childhood will also predict ASPD features in young adulthood. The attachment measure yields three scaled scores for 1) caregiving behavior, 2) punitive behavior, and 3) disorganized behavior. Both punitive behavior and disorganized behavior have been associated with child externalizing behavior (Moss et al., 2004a, b; O'Connor et al., in press). Hence, we predicted that punitive and disorganized behavior would be most strongly associated with later ASPD features.

5. Disorganized/controlling behavior in middle childhood will add to the prediction of later ASPD features, beyond that available from assessments in infancy. According to this hypothesis, the parent-child relationship is a dynamic system that continues to change from infancy to childhood in response to a variety of changes in stresses and protective factors (DeKlyen & Greenberg, 2008). Children in relationships that change toward more disorganized/controlling attachment interactions will incur additional risk for later antisocial personality features.

6. The early onset of child externalizing behavior will mediate any observed relations between the quality of the parent-child relationship and later ASPD features. Internalizing symptoms were not examined as they have not been found to add variance to the prediction of antisocial behavior once externalizing behavior is controlled (Diamentopolou et al., 2010).

Methods

Participants

Participants were 120 young adults (70 females) aged 18-23 years (M = 19.9; SD =1.5) and their mothers. Fifty-six of these families had been participating in a longitudinal study since infancy. These 56 families were part of a cohort of 76 families recruited during the first 18 months of the child's life and were at or below federal poverty level at study entry. In young adulthood, 14% (11) could not be relocated; 11% (7) refused; and 3% (2) lived overseas, yielding a retention rate of 74 % (56). Attrition was not related to any of the infancy and childhood assessments in this report (effect sizes (φ or η) = -.14 through .13) and was associated with only one of eight socioeconomic indices, single parenthood: X^2 (1, N = 76) = 8.66, φ = .34, p = .01; 68% of those followed up had a partner in the home in infancy, compared to 30% of those lost to follow-up. Families seen longitudinally did not differ on mother's educational level, family income, government aid recipient; mother's age at birth of first child, number of young children under age six, child gender, or ethnic minority status, all p n.s.

Sixty-four additional families were recruited in young adulthood as part of a larger study of adaptation in young adulthood that included both cross-sectional and longitudinal groups. Cross-sectionally recruited families were matched to longitudinal families on young adult age, ethnicity, and mother's single parenthood. Family income was slightly higher among the cross-sectional families than the longitudinal families (family income mean range: longitudinal $20,000- $30,000/yr.; cross-sectional $30,000 -$40,000/yr.(F(1, 118)=9.63, η=.28, p=.01)). All procedures were approved by the Hospital Institutional Review Board. Written informed consent was obtained from both parent and young adult.

Measures: All Participants

Antisocial personality disorder features—Extent of ASPD features were assessed for participants using the Structured Clinical Interview for Diagnosis (SCID) for DSM-IV Axis II, administered by trained clinicians (First, Gibbon, Spitzer, Williams, & Benjamin, 1997). Presence of substance abuse and major depressive episodes were assessed on the SCID-I (First, Spitzer, Gibbon, & Williams, 1997). Twelve participants did not have complete SCID data.

Presence of childhood abuse—An overall 7-point rating for severity of abuse from birth to age 18 was assigned by reviewing scores from one self-report and two interview measures that assessed abusive experiences: the Conflict Tactics Scale-2nd version (self-report: CTS-2: Straus, Hamby, Finkelhor, Moore, & Runyan, 1998), the Traumatic Stress Schedule (Interview: TSS: Norris, l990), and the Childhood Traumatic Experiences Scales-Revised (Interview: CTES-R, Dutra, Bureau, Holmes, Lyubchik, & Lyons-Ruth, 2009) coded from the Adult Attachment Interview. Whether the family had been involved with state protective services around the care of the young adult was also assessed from interviews, as was information on whether the young adult had ever been placed in foster care between 0-18 years. Each individual's overall severity of abuse was classified into one of 7 levels as follows: 1-no occurrence of abuse; 2- harsh punishment only; 3- witnessed violence only; 4- verbal abuse only; 5- physical abuse (using state guidelines for abuse), sexual abuse (using state guidelines for abuse), or protective services/foster care involvement; 6- two under level 5; 7- all those under level 5. Reliability of the overall severity of abuse scale was ICC = .99, with 37 participants (30% of sample) coded for reliability. For purposes of the present study this variable was dichotomized to code presence or absence of abuse as defined as the presence of verbal, physical, or sexual abuse (scores of 4 or above on the severity scale). The component multimethod measures contributing to the final rating are described below.

Severity of abusive experiences: Conflict Tactics Scale (2nd version)—The CTS-2 (Straus, Hamby, Finkelhor, Moore, & Runyan, 1998) is a widely used 78-item participant report measure of the type and frequency of strategies used to resolve conflict between family members. The participant rates each item on an 8-point scale indicating the frequency with which a particular conflict tactic occurred during the past year or during another rating period of interest. The rating period specified in the present study was “the year that things were worst between you and your parent.” Tactics include physically abusive behavior and emotionally abusive behavior, e.g. “My parent hit or kicked me; my parent insulted or swore at me.” The overall scale has a stable factor structure, moderate test-retest reliability, and demonstrated validity (Straus, 1990; Straus, Hamby, Boney-McCoy, & Sugarman, 1996).

Severity of abusive experiences: Traumatic Stress Schedule-Short Version—(TSS: Norris, 1990) is an eight-question survey asking about traumatic experiences, including exposure to hazards, natural disasters, accidents, and assaults. In assigning the rating for severity of overall abuse above, only the first three questions of the TSS were relevant because they focus on experiences of sexual or physical assault. In case of a positive answer to any of the items, participants are asked who the assailant was, a brief description of the event, and why the participant believed this had happened to them.

Severity of abusive experiences: Childhood Traumatic Experiences Scales-Revised—The CTES-R ((Dutra et al., 2009) rates the severity of abuse revealed during the Adult Attachment Interview (AAI). The AAI (George, Kaplan, & Main, 1984) is a semi-structured, transcribed one hour interview that asks participants in detail about attachment-

related experiences with primary caretakers. For this study, additional questions probing sexual and physical abuse experiences were added to the standard AAI protocol in order to gain fuller information on traumatic childhood experiences. The AAI was administered to participants in young adulthood. Interviews were transcribed and coded for severity of physical abuse, sexual abuse, verbal abuse and witnessing interpersonal violence, on four 5-point scales for severity of physical abuse, sexual abuse, witnessed violence, and verbal abuse. Inter-rater reliabilities ranged from $r_i = .89$ (verbal abuse) to $r_i = .98$ (sexual abuse). Convergent validity with other measures of childhood abuse has been demonstrated with the CTES-R (Dutra et al., 2009).

Early clinical risk—Twenty-four percent of families, all from the longitudinal group (see Table 1), had been referred to the study by clinical service providers for parent-infant clinical services during the young adult's infancy due to concerns about the quality of parental care. Mothers and infants were referred for parent-infant services by calls from staff members of health, educational, and social service agencies serving low-income families because of concerns about the quality of the caregiving environment for the infant. Program publicity stressed that parent-infant services were available for families showing a range of caregiving disturbances. Infants were required to be no older than 9 months of age for service eligibility. Referral calls were followed up by staff screening to verify caregiving risk. Eight families in the referred group had engaged in state-documented maltreatment in the first nine months of the child's life, but caregiving risk in the remainder of the referred families did not reach criteria for abuse or neglect.

The remainder of the longitudinal cohort (22.5% of the overall sample, see Table 1) were socioeconomically matched families from the same communities who were recruited through advertisements in community locations. These families were matched to referred families on per-person family income; mother's education, age, and ethnicity; and infant's age, sex, and birth order. Mothers in the community group had never been reported to state protective services, had never been referred to clinical services oriented toward parenting, and were not observed to display problematic parenting behavior during a one-hour home observation by study clinicians. Further details are available in Lyons-Ruth, Connell, Grunebaum, and Botein (1990).

The remaining 53.3% of families were recruited in young adulthood and mothers were interviewed about participation in clinical or parenting services during their child's infancy. None of the families in the cross-sectional young adult cohort had received clinical infant services. This is not surprising given the general unavailability of such services in the area in which the study was conducted.

Measures: Longitudinal Subsample

Infant attachment security (18 months)—Mothers and infants were videotaped in the Strange Situation Procedure (Ainsworth et al., 1978) at 18 months. In this procedure the infant is videotaped in a playroom during a series of eight structured 3-min episodes involving the baby, the mother, and a female stranger. During the observation the mother leaves and rejoins the infant twice, first leaving the infant with the female stranger, then leaving the infant alone to be rejoined by the stranger. The procedure is designed to be mildly stressful in order to increase the intensity of activation of the infant's attachment behavior. Videotapes were coded for traditional organized and disorganized attachment behavior (Ainsworth et al., 1978; Main & Solomon, 1990). The three original attachment classifications (secure, avoidant, ambivalent) were assigned both by a coder trained by M. Main and by a computerized multivariate classification procedure developed on the original Ainsworth data (Connell, 1976; Lyons-Ruth, Connell, & Zoll, 1987; see also reference in

Richters, Waters, & Vaughn, 1988). Agreement between the two sets of classifications was 86%. Agreement on the disorganized classification between M. Main and a second coder for 32 randomly selected 12- and 18-months tapes was 83% (k = .73). Additional information is available in Lyons-Ruth et al. (1990).

There were no infants who displayed organized ambivalent attachment patterns in this sample: all four infants classified as ambivalent also met criteria for the disorganized category and were classified as disorganized. Attachment distribution for those seen longitudinally (n = 56) were Secure 27%; Avoidant 16%; Disorganized 46%. In the present analyses, security of attachment was indexed by a three-level variable (1=secure; 2=insecure-organized;3=insecure-disorganized).

Disrupted mother-infant communication (18 months)—Disrupted maternal communication was coded in the Strange Situation Procedure by separate coders naïve to all other data using the Atypical Maternal Behavior Instrument for Assessment and Classification (AMBIANCE) (Lyons-Ruth, Bronfman, & Parsons, 1999). Frequencies of five classes of disrupted maternal communication are coded: a) affective communication errors, b) role confusion, c) negative-intrusive behavior, d) disorientation, and e) withdrawal. Fifteen randomly selected tapes from the current sample were coded by two coders to assess reliability. The reliabilities were as follows: Affective Communication Errors, r_i = .75; Role Confusion, r_i = .76; Negative-Intrusive Behavior, r_i = .84; Disorientation, r_i = .73; Withdrawal, r_i = .73. Validity of the AMBIANCE, as well as its stability over a 5-year period, have been established by meta-analysis (N's of 384 and 203) (Madigan et al., 2006).

Middle Childhood Disorganization and Control scales (MCDC) (Age 8)—were used to rate three dimensions of children's attachment behavior toward their parents on 9-point scales: controlling-punitive behavior, controlling-caregiving behavior, and behavioral disorganization (Bureau, Easterbrooks, & Lyons-Ruth, 2009). Scales were developed based on an extensive body of work identifying controlling and disorganized behavioral presentations among children aged 3 to 6 years of age (Main & Cassidy, 1988; Moss, Rousseau, Parent, St-Laurent, & Saintonge, J., 1998; O'Connor, Bureau, McCartney, & Lyons-Ruth, in press). Following established procedures for schoolaged children (Main & Cassidy, 1988), parent and child were videotaped in a 5-minute reunion following a one-hour separation during which both child and mother completed other interview measures. Behavior in the high range of the *controlling-punitive* scale is characterized by episodes of hostility toward the parent that are marked by a challenging, humiliating, cruel or defying quality. Behavior in the high range of the *controlling-caregiving* scale is marked by actively organizing or taking charge of the interaction or by the child subordinating his/her own desires and prioritizing the parent's needs. The *disorganized behavior* scale codes fearful, unpredictable, confused, inappropriate or disoriented sequences of interaction with the parent. Intraclass reliabilities between two coders on 22 participants in the current sample: Controlling-Punitive = .97; Controlling-Caregiving = .93; and Disorganized= .83. The MCDC scales have been validated in relation to early caregiving risk, elevated levels of behavior problems, and disorganization in projective story narratives (Bureau et al., 2009).

Teacher ratings of child behavior problems (five years)—The Preschool Behavior Questionnaire (PBQ) (Behar & Stringfield, 1974a) was completed by teachers at age 5. The PBQ represents a modified version of Rutter's (1967) Children's Behavior Questionnaire, developed for children in the 3 to 6-year-old age range. Total score ranges from 0 to 60. Test-retest values, interrater reliabilities, and standardization sample characteristics are all acceptable (see Behar & Stringfield, 1974b). Factor analytic studies extracted three factors: hostile, anxious, and hyperactive (Behar, 1977; Behar & Stringfield, 1974b). Because our

interest was in clinically significant behavior, clinical cutoff scores were used in the current analyses. Clinical cutoff scores which maximally discriminated normal and disturbed groups in standardization samples were 8 for hostile behavior, 5 for anxious behavior, and 5 for hyperactive behavior (Behar & Stringfield, 1974b). These cutoff values indicated the upper 10% of scores among children in the normal validation group. Scores over the cutoff point cannot be considered comparable to a clinical disorder but merely denote deviance from normative behavior.

Teacher ratings of child behavior problems (seven years)—Teachers' reports of children's behavior problems were collected using the well-validated 113-item Child Behavior Checklist (CBCL) from the Child Behavior Profile and the Teacher Report Form for classroom teachers (Achenbach, 1991). CBCL scores for internalizing and externalizing behaviors were standardized (T scores). Teachers also completed the report of CBCL maladaptive school functioning, yielding a summary measure of school maladaptation (T score). Scores for externalizing symptoms and maladaptive functioning in school were of interest in the current report and these scores were dichotomized according to the cutoffs representing the combined borderline and clinical ranges (scores at or above 95th percentile of standardization sample), cutoffs recommended by Achenbach (1991) as minimizing false negatives and false positives.

Analytic Procedures

Analyses involving the full cross-sectional sample of 120 families are presented first, followed by analyses involving only the 56 families seen longitudinally since infancy. For both cross-sectional and longitudinal analyses, associations between the individual predictor variables and ASPD features on the SCID-II were first evaluated. Hierarchical linear regression models (Cohen, Cohen, West, & Aiken, 2003) were then calculated using SPSS software to assess the relative contributions of the significant predictor variables, as well as to evaluate the proposed mediational models. For the cross-sectional analyses, a follow-up analysis controlling for comorbid depression and substance abuse was also computed to evaluate whether the obtained relations were a function of comorbidities or were specific to ASPD features.

Statistical mediation was evaluated following Baron and Kenny (1986). All tests were two-tailed. Effect size calculations used r for two continuous variables; Nagelkerke R for a continuous predictor of a dichotomous variable; η for a dichotomous predictor of a continuous variable; φ for two dichotomous variables(Cohen et al., 2003). For longitudinal analyses, following recommendations of Schafer and Graham (2002), multiple imputation procedures were used to estimate missing data. Missing data were imputed using the Markov Chain Monte Carlo procedure (MCMC) (Gilks, Richardson, & Spiegelhalter, 1996), with SAS software. The percentages of missing data ranged from 0-37.5%, with a total missing rate of 18.4%. This range is well within the recommended allowances for imputation procedures and imputation is strongly recommended over analysis of the raw data alone (McCartney, Burchinal, & Bub, 2006; Schafer & Graham, 2002) As noted under Results, longitudinal findings were similar whether raw data alone or raw data with imputation procedures were used. In the present study, 10 data sets were generated with excellent efficiency (power to detect a significant effect was 97%) according to Rubin's guidelines (Rubin, 1987).

Results

Prevalence of ASPD Features and Diagnosis

Number of ASPD features in the sample ranged from 0 to 4 (m .81; s.d. 1.25); 47% (n=48) met criteria for one or more features; 7.4 % (n=8) met criteria for diagnosis. Given the small number of those meeting diagnostic criteria, only the analyses with the continuous measure of extent of ASPD features are reported here. However, all cross-sectional results were the same when contrasting the small group with the ASPD diagnosis with the rest of the sample (analyses available from the last author). Socioeconomic variables were unrelated to ASPD features and diagnosis, including family income, mother's education, mother's single parenthood, and young adult age and minority status (effect sizes -.24 to .19, all p n.s.). Male gender was associated with both ASPD features (effect size=.31) and ASPD diagnosis (effect size=.27, both p<.05), so gender was controlled in all analyses. Means and standard deviations for independent variables are shown in Table 1.

Correlates of ASPD: Full Sample Analyses

Presence of childhood abuse—Presence of abuse before age18 was significantly related to extent of ASPD features (Table 2). Notably, verbal abuse was as strongly associated with ASPD features as sexual/physical abuse (verbal abuse only: features mean 1.00, s.d. 1.56, n = 10; sexual/physical abuse: features mean 1.14, s.d. 1.48, n = 47; no abuse: features mean .38, s.d. .97; n = 51).

Clinical risk in infancy—Early clinical risk was also related to extent of ASPD features, both when the clinical risk group was contrasted with the other families seen longitudinally and when the risk group was contrasted with the families seen only cross-sectionally (Table 2). Participants referred in infancy for clinical services had significantly more ASPD features at age 20 than other longitudinal participants not referred in infancy (based on screening occurring in infancy), β *(with gender controlled)* = .28, p = .05. Participants referred in infancy also had significantly more ASPD features at age 20 than participants recruited in young adulthood who had not been referred in infancy (based on self-report) (β *(with gender controlled)* = .28, p = .01). It should be noted that any *failure to report* early services among the cross-sectional group would *decrease* the possibility of detecting differences between this group and the referred group. Comparing the early referred group to the combined non-referred groups, clinical risk identified in infancy accounted for 7% of variance in ASPD features.

Abuse as potential mediator—One possible reason for the significant prediction from early clinical risk is that early risk is a predictor of later abuse, and the occurrence of later abuse is the important mediator of the link to ASPD. Therefore, mediational analyses assessed whether childhood abuse could account for the variability in outcomes associated with early clinical referral. Presence of childhood abuse was significantly associated with extent of ASPD features (Table 2) and was also related to early clinical risk (phi=.28, p<. 003). However, abuse did not meet criteria as a possible mediator of the effect of clinical risk because with early clinical risk controlled, abuse no longer reached significance as an independent predictor of ASPD features. However, as predicted in Hypothesis 1, with variance related to *abuse* controlled, early clinical risk remained a significant predictor of ASPD features (Table 2).

Specificity of Results in Relation to Substance Abuse and Major Depression

As expected (Odgers et al., 2008; O'Donohue, Fowler, & Lilienfeld, 2007), there was significant comorbidity of substance abuse with ASPD outcomes (38.0% substance abuse diagnosis (n = 41); association with ASPD features, β = .39, p <.000, gender controlled).

Similarly, major depression was comorbid with ASPD features (32.4% major depressive episodes (n = 35); association with ASPD features, β = .21, p =.03, gender controlled). Substance abuse was also related to male gender (phi = .20, p = .04), and depression (phi = . 23, p = .02), and marginally related to childhood abuse (phi = .17, p = .09). Depression was unrelated to childhood abuse (phi = .01, n.s.) and neither depression nor substance abuse was related to early clinical risk (phi = -.04 and .13, respectively). Consistent with Hypothesis 2, with substance abuse and major depression controlled, the effect of early clinical risk on ASPD features remained significant (β = .19, p = .03). The effect of childhood abuse did not reach significance with depression and substance abuse controlled (β = .14, p = .10).

Prospective Longitudinal Assessments

In the longitudinal subsample, three additional prospectively assessed measures of the parent-child relationship were available, including infant attachment security at 18 months, disrupted maternal communication at 18 months, and disorganized-controlling attachment behavior at age eight. Range of ASPD features in the longitudinal subsample was 0-4 (m 1.21; s.d. 1.51). Due to the small sample size, ASPD categorical diagnosis was not analyzed. The associations among the longitudinal independent variables are shown in Table 3, with gender controlled. The relation between each longitudinal variable and ASPD features, with gender controlled, is shown in Table 4. All significant results in Table 4 were also significant without imputing missing data.

Mother-infant attachment and interaction as predictors—Our first longitudinal hypothesis was that infancy variables would account for variance in ASPD features beyond that accounted for by childhood abuse. Security of the infant's attachment behavior was not predictive of later ASPD outcomes (Table 4). However, maternal disrupted communication in the form of maternal withdrawal from the infant's attachment signals was a significant predictor of ASPD features (Table 4). Other dimensions of maternal disrupted communication with the infant were not predictive.

We then evaluated whether this prediction from maternal withdrawal could be accounted for by the occurrence of abuse in childhood. The occurrence of childhood abuse just missed significance as a predictor in this smaller longitudinal cohort (p = .10; Table 4). In addition, regression analysis on ASPD features confirmed that occurrence of childhood abuse could not account for the effects of early maternal withdrawal on later ASPD features, consistent with Hypothesis 3. After controlling for gender and occurrence of childhood abuse, early maternal withdrawal continued to account for significant independent variance in later ASPD features (maternal withdrawal: t = 2.54, df (1,53), p =. 01).

Disorganized-controlling attachment behavior in middle childhood as predictor—Hypothesis 4 was that middle childhood attachment behavior would also predict ASPD features and would add to the prediction available from the infancy variables. Controlling-punitive child attachment behavior toward the parent was a marginally significant predictor of ASPD features (p = .09), and disorganized attachment behavior was a significant predictor (p = .04) (Table 4). Caregiving behavior was not associated with ASPD features. Thus, our expectation that child punitive and disorganized behavior would be especially predictive of later ASPD features was confirmed. However, disorganized behavior in the presence of the parent was a somewhat stronger predictor.

In order to evaluate whether child disorganized behavior at age 8 added to the prediction of ASPD features after parental withdrawal in infancy was accounted for, an additional regression model was computed. With child gender and maternal withdrawal entered first

into the model (these statistics available in Table 4), the contribution of child disorganized behavior at age eight to ASPD features did not drop significantly (Sobel test= .43, SD=.03, p= .67) and child disorganization remained marginally significant as a predictor of ASPD features (Estimate = .23, SE .14, t-ratio= 1.74, d.f. (1, 52), p=.08). Therefore, consistent with Hypothesis 4, the prediction from the middle childhood assessment was not accounted for by the extent of earlier maternal withdrawal.

Teacher-rated behavior problems: Do childhood symptoms mediate long-term prediction to ASPD?—Our fifth hypothesis was that children with disturbed interactions who were *not symptomatic* in childhood would not show later ASPD features. Teacher ratings of behavior problems were obtained in kindergarten and second grade. Hostile and hyperactive behavior at age 5 and externalizing behavior problems and general maladaptive behavior at age 7 were first examined as potential precursors to later ASPD features. With gender controlled, both hyperactive behavior at age 5 and general maladaptive behavior in the classroom at age 7 predicted extent of ASPD features, while hostile behavior at age 5 and externalizing behavior at age 7 did not reach significance (Table 4). Notably, then, it was the child's hyperactivity and general maladaptation at school, rather than hostile or externalizing behavior per se, that best predicted later ASPD features.

We then evaluated whether these child symptoms at 5 and 7 years would account for the variability in ASPD features associated with the quality of parent-child interaction in middle childhood. That is, we expected that children who had poor quality parent-child relations *and* were symptomatic in childhood would show later ASPD features, while those who were *not* symptomatic in the early school years would not show later elevated ASPD features. To assess this possibility, we ran a series of regressions to assess mediation, with gender always controlled on the first step (see note to Table 4 for Step 1 values).

The first set of analyses revealed that hyperactive behavior at age five was unrelated both to maternal withdrawal in infancy (t = 1.01, df 1,53, n.s.) and to child disorganization at age eight (t = 1.42, df 1, 53, n.s.), so could not be a mediator of those effects. Furthermore, hyperactive behavior no longer added to prediction of ASPD features once maladaptive behavior at age seven was accounted for (t = 1.07, df 1,52, n.s.). However, maladaptive behavior at age seven continued to predict ASPD features significantly with hyperactive behavior controlled (t = 1.95, df 1,52, p = .05). Therefore, only maladaptive behavior at age seven was analyzed further as a mediator.

Maladaptive behavior at school at age seven was unrelated to maternal withdrawal in infancy (t = .56, df 1, 53, n.s.), but was significantly related to child disorganized behavior toward the parent at age eight (t = 2.67, df 1, 53, p = .05). With both middle childhood assessments entered into the equation on the same step, maladaptive behavior at school at age 7 continued to account for marginally significant variance in later ASPD features (t = 1.77, df 1,52, p = .08), while disorganized attachment behavior at age 8 did not continue to account for variance in ASPD features in young adulthood (see similar values in Table 5, where childhood abuse is also controlled). The Sobel test for mediation by maladaptive behavior at age 7 was insignificant, however, Sobel test = 1.58, S.D. =.37, p=.11. Therefore, the two middle childhood assessments are best described as sharing substantial variance in predicting ASPD features (compare to Table 4: maladaptation p = .004 to .08; disorganization p = .04 to .ns). Thus, disorganized children who later displayed ASPD features tended to be symptomatic by age 7 and, conversely, those displaying maladaptive behavior at school at age 7 were also likely to show signs of unusual, out-of-context behavior in their interactions with their mothers at age 8.

The effect of age 7 maladaptation on ASPD features could not be accounted for by maternal withdrawal in infancy. After gender and maternal withdrawal were entered into the equation (see values in Table 4), age 7 maladaptation continued to account for significant additional variance in ASPD features ($t = 2.42$, df 1, 53, $p = .02$)

The final regression model in Table 5 assessed whether the prediction from early maternal withdrawal to later ASPD features could be accounted for by the two middle childhood assessments. Contrary to Hypothesis 5, child problem behavior at school age did not account for the variance in young adult ASPD features associated with early maternal withdrawal. Early maternal withdrawal added significantly to the prediction of young adult ASPD features even after accounting for teacher-rated behavior problems at school age, as well as the extent of disorganized attachment behavior in middle childhood and the occurrence of childhood abuse. This analysis indicates that some children of withdrawing mothers who show elevated ASPD features in young adulthood are not symptomatic at school in middle childhood. In summary, an additive, rather than mediated, model was supported in which both infancy and middle childhood assessments added significantly to the prediction of later ASPD features.

Discussion

Disentangling components of the childhood environment related to ASPD is important for understanding its etiology. First, the results of the current study converge with other available studies on ASPD which have documented significant contributions of childhood maltreatment. Second, these prospective longitudinal findings extend the literature by indicating that the quality of the early caregiving environment explains variance in ASPD features not explained by childhood abuse.

Quality of early care, indexed first by clinician referral for problems in the parent-infant relationship during the first 18 months of life, was a significant predictor of ASPD features and diagnosis almost 20 years later. This finding was replicated with similar effect sizes both when the two prospectively observed longitudinal groups were compared, and when the referred group was compared to the group assessed retrospectively in young adulthood. Moreover, early clinical risk remained predictive of ASPD features and diagnosis after eliminating the covariance of ASPD with substance abuse and major depression. One important implication of these findings is that the independent effect of quality of early caregiving is not adequately captured by standard measures of childhood abuse, so that quality of early care should be conceptualized and measured separately.

In the longitudinal subsample, additional independent measures of quality of care were rated from videotape. Infant security of attachment assessed at 18 months of age was not a significant predictor of extent of later antisocial symptoms. However, maternal behavior observed on videotape in infancy was an important predictor of later ASPD outcomes. The coding of maternal withdrawal includes such behaviors as interacting silently, failing to greet the infant, and using toys instead of the self to soothe the infant. These ways of interacting with the infant produce an affectively dead, emotionally distanced feel to the interaction and would seem to communicate to the infant the parent's reluctance to participate in a physically close and emotionally engaged relationship. Such emotionally unavailable parental responses to infant attachment behaviors fail to provide adequate regulatory support for the infant at times of stress, with earlier work confirming that maternal withdrawal is associated with infant disorganized attachment behaviors (Lyons-Ruth, Bronfman, & Parsons, 1999). Further, Bugental et al. (2003) have linked maternal withdrawal to elevations in the infant's basal cortisol levels.

Further work is needed to examine the biological consequences of a withdrawing parental stance for the regulation of child stressful arousal and neurotransmitter function (Glenn & Raine, 2008). To date, randomized animal models have shown that less attentive early care not only produces prolonged enhancement of infant stress responses in the HPA axis (Barr et al., 2004; Francis et al., 1999), but also alters noradrenalin, serotonin and dopamine systems in ways that can persist into adulthood and may influence aggressive behaviors (Bremner et al., 1998; Bennett et al., 2002). Molecular genetic studies indicate that there are gene-environment interactions in the development of antisocial behavior involving serotonin-related genes (Caspi et al., 2002; Ducci et al., 2008; Taylor & Kim-Cohen, 2007). Therefore, the role of disturbed caregiving in ASPD could take effect through altering the expression of genes related to serotonin pathways and stress responsiveness. These results extend the link between low maternal affection and nurturance as self-reported at age 16 and ASPD at age 22 in the work by Horwitz et al. (2001), by demonstrating a similar link starting in infancy and assessed by direct observational methods.

Security of attachment in infancy was not a predictor of young adult ASPD features, contrary to prediction. However, the prominence of early maternal withdrawal in the pathways toward ASPD features suggests one potential explanation for this lack of effect. In previous work on the infancy data from this sample, David and Lyons-Ruth (2005) reported that high levels of maternal withdrawing behavior were associated with high levels of infant approach behavior toward the mother. This infant approach behavior sometimes included clear disorganized behaviors, yielding a disorganized attachment classification for these infants. However, for other infants disorganization was not apparent and only approach behavior was seen, which resulted in a secure classification for the infant. We noted in that paper that other data on those families clearly indicated disturbance in the parent-infant relationship (e.g. protective service involvement), so that maternal withdrawal in some cases resulted in misclassification of infant attachment behavior as secure.

In addition to these effects associated with early care, the child's disorganized-controlling patterns of interaction with the parent at age eight were also significantly predictive of ASPD features. We had expected that controlling-punitive interactions with the parent in childhood might be one early predictor of ASPD, given the large literature relating hostile and coercive parent-child interactions from at least age three to later aggressive behavior and delinquency (e.g. Trentacosta & Shaw, 2008). Few of these studies have used diagnostic criteria for ASPD, however, which also emphasize passive-aggressive failures to meet familial and societal obligations, with less emphasis on the interpersonal forms of aggression more prominent in conduct disorder and delinquency studies. Therefore, the parent-child interactive antecedents of ASPD may also be somewhat distinct. Contrary to expectations, controlling-punitive behavior was only marginally significant as a predictor. This marginal effect may simply reflect the low power available in our longitudinal cohort. In contrast, disorganization in the child's responses to the parent in middle childhood *were* an early danger sign for ASPD. It is also notable that disorganized attachment at age 8 added marginally to prediction even after effects associated with maternal withdrawal were accounted for. Further work on the role of disorganized and controlling forms of attachment as antecedents of ASPD is clearly indicated.

The convergence of two independent assessments of the quality of early care, clinician's judgments and laboratory observations, strengthens the finding that an adverse environment in infancy contributes unique variance to the development of later ASPD. The extent to which these effects of infant care are *specific* to ASPD cannot be answered from the present analyses. However, the bivariate correlations revealed no relation between early clinical risk and either depression or substance abuse. In addition, in other longitudinal analyses in this sample, Bureau et al. (2009) have found that young adult depression was not predicted by

maternal withdrawal or early clinical risk, but was significantly predicted by maternal depression in infancy. Taken together, these results suggest some specificity to the current findings in relation to later depression and substance abuse. .

The impact of early care does seem to generalize across ASPD and borderline personality disorder features, however. In other recent work (Lyons-Ruth, Bureau, Holmes, Easterbrooks, & Hennighausen, 2009), both maternal withdrawal and disorganized-controlling forms of attachment behavior in middle childhood were also predictors of later borderline personality disorder (BPD) features. Kendler et al. (2008), using behavioral genetic techniques, have also reported that ASPD and borderline personality disorder share common (unspecified) environmental factors. Taken together, these results imply that deviations in the early attachment relationship may be part of a common environmental contribution to borderline and antisocial personality disorders, a contribution that in both cases is independent of childhood abuse.

We also assessed the hypothesis that by the early school years, child behavioral problems would become evident among a subset of those with disturbed attachment relationships, and children with early problems would be those more likely to show later ASPD features. Early behavior problems as rated by teachers *were* found to be predictors of later ASPD features. Teacher's ratings of child maladaptation on the CBCL proved to be the most important precursor to ASPD features because they were significantly related to both hyperactive behavior at age 5 and to externalizing behavior at age seven (Table 3), but captured the prediction to later ASPD better than these two more clearly externalizing behavior problems. In addition, maladaptive behavior at school at age seven was related to disorganized behavior toward the parent at age 8 (Table 3).

Contrary to our hypotheses, however, parental withdrawal in infancy was not associated with externalizing behavior problems in childhood, though it was a predictor of later abuse and of child caregiving behavior toward the mother (Table 3). Therefore, we found no mediators of the long-term effects associated with maternal withdrawal, either in childhood behavior problems, childhood disorganized attachment, or childhood abuse. The prediction from childhood disorganized behavior to ASPD features also was not accounted for by maladaptive behavior at schoolage, even though these two middle childhood variables were significantly related and overlapped substantially in prediction. Therefore, over both cross-sectional and longitudinal analyses, the results suggest that the lack of investment in societal standards and demands in young adulthood that is indexed by ASPD features may be contributed to by disturbances in parental care that begin in the first years of life. Prediction of ASPD features is further enhanced by assessment of maladaptive behavior evident in the early school years, by disorganized attachment strategies in middle childhood, and by abuse experiences before age 16.

Several limitations of the current study should be considered. It is important to note that the reported effects are occurring in the context of relatively low family income. ASPD features were not associated with socioeconomic variations within this sample. However, given the restricted range in the sample, the obtained effects may represent interactions with socioeconomic stress rather than main effects and thus may not apply to more advantaged family settings. In addition, a relatively small number of families contribute to the longitudinal analyses and there are a small number of young adults who meet full diagnostic criteria for ASPD. In addition, given the 20-year span of the study, missing data were inevitably present and recommended statistical procedures were followed to estimate missing observations and minimize data loss. While the amount of missing data here was well within the recommended allowances for imputation procedures (McCartney, Burchinal, & Bub, 2006; Shafer & Graham, 2002), it is important to keep in mind this additional source

of error. Finally, we did not attempt to quantify developmental timing of abuse, nor to specify type of abuse or perpetrators of abuse in these analyses. It may be that a particular timing or type of abuse would emerge as a stronger mediator of the effects associated with the quality of the parent-child relationship.

Despite these limitations, there are important clinical implications of the current findings. The prospective longitudinal data underscore the long developmental pathways culminating in ASPD, as well as the need for effective intervention strategies in the first years of life. Such early intervention efforts should target subtle forms of maternal withdrawing behavior, in particular, as these emerged as the most robust predictors of later ASPD features over a twenty year period. In addition, the additive nature of the model supports the need for additional intervention efforts to prevent later childhood abuse, to target disorganized attachment relationships in childhood, and to monitor the emergence of hyperactive and maladaptive behavior at school. These results suggest that effective reduction in any of these target behaviors should have measurable effects in reducing the symptoms of antisocial personality disorder in young adulthood.

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Table 1

Descriptive Data for Study Variables

Full Sample[a]	
Gender (% female)	57.5 % (69)
Early clinical risk	
Not clinically referred-cross-sectional	53.3 % (64)
Not clinically referred-longitudinal	22.5 % (27)
Clinically referred-longitudinal	24.2 % (29)
Childhood abuse	51.7% (62)

Longitudinal Subsample[b]	**Mean**	**S.D.**
Continuous variables		
Security of infant attachment (18 mos.)	2.25	.90
Maternal disrupted communication (18 mos.)		
Affective communication errors	5.20	4.68
Role confusion	5.10	7.35
Disorientation	3.05	3.72
Negative intrusiveness	2.37	3.71
Withdrawal	3.21	3.55
Middle childhood attachment (8 yrs.):	2.24	.69
Punitive behavior	2.25	.34
Caregiving behavior	3.09	.42
Disorganized behavior	1.90	.40
Categorical variables		
Gender (% female)	41.1% (23)	
Early clinical risk		
Not clinically referred-longitudinal	48.2 % (27)	
Clinically referred-longitudinal	51.8 % (29)	
Hostile behavior-clinical cutoff (5 years)	35.6 % (16)	
Hyperactive behavior-clinical cutoff (5 years)	24.4 % (11)	
Externalizing behavior-clinical cutoff (7 years)	8.8 % (3)	
Maladaptive behavior-clinical cutoff (7 years)	30.3 % (10)	
Childhood abuse	55.4% (31)	

[a] Full sample with SCID data N = 108.

[b] Longitudinal N = 56

Table 2

Regression Models and Effect Sizes for ASPD Symptoms: Full Sample Analyses

Extent of ASPD Symptoms					
	FΔ	***df***	***P***	***R²Δ***	**β**
Model 1:					
Step 1					
Male gender	13.73	1, 106	.000	.12	.34***
Step 2					
Early clinical risk	3.55	2,104	.03*	.06	
Early clinical risk vs longitudinal comparison					.28*
Early clinical risk vs cross-sectional comparison					.28**
Model 2:					
Step 1					
Male gender	13.73	1, 106	.000	.12	.34***
Step 2					
Childhood abuse	5.07	1,105	.03	.04	.22*
Model 3: Evaluating Mediation by Childhood Abuse					
Step 1					
Gender[a]	13.73	1,106	.000	.12	.34***
Step 2					
Childhood abuse	5.07	1, 105	.03	.04	.20*
Step 3					
Early clinical risk	4.59	1, 104	.04	.04	.20*

Note. N = 108.

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Table 3

Associations Among Longitudinal Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Early clinical risk	1														
2. Infant attachment security	.18	1													
3. Affective communication errors	.43**	.24	1												
4. Role confusion	-.05	.31*	.21	1											
5. Disorientation	.23	.22	.33*	.34*	1										
6. Negative-intrusiveness	.25	.31*	.51***	.55***	.42**	1									
7. Withdrawal	.39**	-.04	.57***	-.2	.27	.07	1								
8. Hyperactive behavior (5 yrs.)	.21	.11	.21	.05	.01	.19	.18	1							
9. Aggressive behavior (5 yrs.)	.36**	.39**	.21	.28*	.02	.18	.002	.41**	1						
10. Externalizing behavior (7 yrs.)	.02	.08	.21	-.11	.07	.09	.06	.40**	.15	1					
11. Maladaptive behavior (7 yrs.)	.43**	.09	.16	-.09	.01	.08	.10	.35*	.07	.43**	1				
12. Punitive behavior (8 yrs)	.21	.24	.18	-.06	-.01	.28	-.08	.20	.25	.28	20	1			
13. Caregiving behavior (8 yrs.)	-.11	-.16	.05	-.05	-.02	-.24	.38*	.05	-.11	-.24	-0.14	-0.33	1		
14. Disorganized behavior (8 yrs.)	.44*	.1	-.04	-.14	.1	-.14	.09	.28*	.15	.14	.53**	0.34	0.01	1	
15. Childhood abuse	.36*	.07	.24	-.07	.05	-.01	.39**	.27	.26	.13	0.17	0.26	-0.09	0.2	1

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Table 4

Regression Estimates for Individual Infancy and Childhood Predictors of ASPD Symptoms: Longitudinal Cohort

Predictors of ASPD Symptoms[a]	Estimate	*S.E.*	*t*-ratio	*d.f.*	*p*
Parent-infant interaction (18 mos.):					
Infant attachment security	-.26	.26	-1.00	1, 53	n.s.
Maternal disrupted communication					
Affective communication errors	-.00	.05	-.01	1, 53	n.s.
Role confusion	-.04	.03	-1.44	1, 53	n.s.
Disorientation	.09	.06	1.51	1, 53	n.s.
Negative-intrusiveness	.02	.07	1.35	1, 53	n.s.
Withdrawal	.17	.06	3.19**	1, 53	.002
Childhood behavior problems					
Hyperactive behavior (5 yrs.)	1.01	.44	2.31*	1, 53	.02
Aggressive behavior (5 yrs.)	-.67	.48	-1.38	1, 53	.17
Externalizing behavior (7 yrs.)	-1.22	.81	-1.50	1, 53	.13
Maladaptive behavior (7 yrs.)	1.50	.51	2.92**	1, 53	.004
Middle childhood attachment (8 yrs.)[b]:					
Punitive behavior	-.30	.16	-1.85x	1, 51	.09
Caregiving behavior	.09	.12	.08	1, 51	n.s.
Disorganized behavior	.37	.19	2.10*	1, 51	.04
Childhood abuse	.60	.37	1.62	1, 53	.10

Note. N = 56.

*** $p < .001$

[a] Gender controlled in all analyses; Step 1 gender t-ratio = .3.72, p=.002.

[b] All components entered on a single step in the regression model.

* $p < .05$

** $p < .01$

Table 5

Childhood Assessments Do Not Account for the Effects of Early Maternal Withdrawal on Later ASPD Symptoms

Predictors of ASPD Symptoms	**Estimate**	**S.E.**	***t*-ratio**	***d.f.***	***p***
Step 1					
Male gender	1.34	.40	3.72	1,54	.002**
Step 2					
Childhood abuse	.68	.41	1.66	1,53	.10
Step 3					
Maladaptive behavior age 7 years	.86	.49	1.74	1,51	.08[x]
Disorganized behavior at age 8 years	.08	.17	.51	1,51	n.s.
Step 4					
Maternal withdrawal age 18 months	.16	.07	2.27	1,50	.03*

Note. N=56.