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Neurocognitive Impairments in Boys on the Life-Course Persistent Antisocial Path

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This study addresses 5 unresolved issues in the neuropsychology of antisocial behavior using a community sample of 325 school boys in whom neurocognitive measures were assessed at age 16–17 years. Antisocial behavior measures collected from age 7–17 years were cluster analyzed and produced 4 groups: control, childhood-limited, adolescent-limited, and life-course persistent. Those on the life-course persistent path and also on the childhood-limited path were particularly impaired on spatial and memory functions. Impairments were independent of abuse, psychosocial adversity, head injury, and hyperactivity. Findings provide some support for the life-course persistent versus adolescent-limited theory of antisocial behavior and suggest that (a) neurocognitive impairments are profound and not artifactual and (b) childhood-limited antisocials may not be free of long-lasting functional impairment.

The growing neuropsychological literature on violent and antisocial behavior is confirming beyond a reasonable doubt what some have argued for a long time—that antisocial and violent offenders have neuropsychological impairments (Henry & Moffitt, 1997; Ishikawa & Raine, 2002; Moffitt, 1990b; Morgan & Lilienfeld, 2000; Raine, 1993). Such research is important at both theoretical, treatment, and prevention levels. Neurocognitive impairments may be a key route through which genetic and psychosocial influences on antisocial behavior find expression, and early prevention or remediation of these impairments could, in theory, help reduce levels of antisocial behavior. Yet, despite recent empirical progress on neurocognition and antisocial behavior, at least five issues remain in question:

1. Do offenders show spatial in addition to verbal impairments?
2. Are they impaired on memory functions?
3. Do neuropsychological impairments characterize life-course persistent (LCP) but not adolescent-limited (AL) individuals?
4. Do neurocognitive impairments also characterize childhood-limited (CL) antisocial individuals, or are these individuals protected from later antisocial behavior by showing particularly good cognitive functioning?
5. Are impairments in antisocial groups attributable to comorbid attention-deficit/hyperactivity disorder (ADHD), abuse, social adversity, or head injury?

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Do Offenders Show Spatial in Addition to Verbal Impairments?

The classic view of cognitive functioning in antisocial populations is that spatial functions are either much less impaired than verbal functions, or they are not impaired at all (e.g., Quay, 1987; Wilson & Herrnstein, 1985). Such conclusions have been based largely on institutionalized offenders, and recent community studies are beginning to question the assumption of the primacy of verbal impairments and antisocial behavior. For example, several recent studies found that early spatial impairments characterize young antisocial children, sometimes in addition to verbal impairments (e.g., Dietz, Lavigne, Atrend, & Rosenbaum, 1997; Speltz et al., 1999) and sometimes in the absence of verbal impairments (e.g., Raine et al., 2002). In adult psychopaths, one study found no evidence for verbal impairments but did find strong impairments on Block Design, a spatial task (Smith, Arnett, & Newman, 1992). A more recent study found spatial but not verbal impairments in adult psychopaths (Raine et al., 2003). Similarly, psychopathic-like children with callous-unemotional traits show an absence of verbal impairments but tend to have poor spatial ability (Loney, Frick, Ellis, & McCoy, 1998).

Theoretically, findings in children have been interpreted on the basis of an early starter spatial impairment model of antisocial behavior (Raine, Yaralian, Reynolds, Venables, & Mednick, 2002), which suggests that early visuospatial (right hemisphere) impairments can predispose an individual to persistent antisocial behavior by interfering with early attachment and emotion recognition and regulation. Regional cerebral blood flow research on infants and young children has shown that the right (not left) hemisphere is dominant from 1–3 years (Chiron et al., 1997), suggesting that right hemisphere dominance regulates nonverbal orienting, attention, arousal, and affective facial expression and recognition that are in evolutionary terms essential for survival in the preverbal infant (Saugstad, 1998). Compromising of these systems would be expected to both impair the infant's orienting to, and recognition of, its mother's facial expression and, in turn, limit the infant's reciprocal expressive response to the mother. In transactional terms, this could elicit more negative parenting from the mother, with the combined effects leading to disruption of early mother–infant bonding and attachment, which in turn could predispose the child to affectionless, psychopathic-like behavior (Raine, 1993). At a later age, right hemisphere dysfunction may contribute to social-information-processing deficits that predispose an individual to antisocial behavior (Dodge, 1991) as well as reduced ability to recognize the negative facial emotions of anger and fear (Adolphs, Damasio, Tranel, & Damasio, 1996; Borod, St. Clair, Koff, & Alpert, 1990). The inability of the growing child to accurately process and recognize signals of negative affect such as anger and fear in a protagonist during a fractious social encounter could contribute to inappropriate responding and escalation into an aggressive response. Thus, early visuospatial deficits may interfere with mother–infant bonding and may reflect right hemisphere dysfunction that disrupts emotion processing and regulation, which in turn contributes to life-course antisocial and aggressive behavior. Nevertheless, additional testing is required to further test the hypothesis that antisocial individuals are characterized by poor spatial ability.

Are Offenders Impaired on Memory Functions?

Neuropsychological reviews of antisocial behavior have focused almost exclusively on executive and verbal functions, with little or no mention of memory functions (Henry & Moffitt, 1997; Fishbein, 2000; Ishikawa & Raine, 2002; Moffitt, 1990b; Nigg & Huang-Pollock, 2002; Raine, 1993). This may partly be due to the fact that studies fail to find memory impairments in male antisocial groups (e.g., Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001; Famularo, Fenton, Kinscherff, & Barnum, 1992; Hare, Frazelle, Bus, & Jutai, 1980; Bergvall, Wessely, Forsman, & Hansen, 2001). There have even been suggestions that psychopathic offenders in particular have superior memories (Andrew, 1982). In contrast, several recent studies do find memory impairments in antisocial and violent groups (Brickman, McManus, Grapentine, & Alessi, 1984; R. A. Cohen, Rosenbaum, Kane, Warnken, & Benjamin, 1999; Dolan & Park, 2002; Moffitt & Caspi, 2001; Seguin, Arsenault, Boulerice, Harden, & Tremblay, 2002; Sigurdsson, Gudjonsson, & Peersen, 2001; Teichner, Golden, Van Hasselt, & Peterson, 2001; Vermeiren, De Clippele, Schwab-Stone, Ruchkin, & Deboutte, 2002). Because the hippocampus subserves memory and because structural and functional hippocampal impairments have been found in violent and psychopathic offenders (Raine, Buchsbaum, & LaCasse, 1997; Raine et al., 2004), it could be predicted that antisocial individuals would show memory impairments. In addition to the question of whether memory impairments in general reliably characterize offenders, few previous studies have systematically explored verbal and spatial impairments in memory tasks to see whether this classic Verbal IQ < Performance IQ profile extends to the memory domain.

Do Neuropsychological Impairments Characterize LCP but Not AL Offenders?

One influential theory of offending has argued for a critical distinction between those whose antisocial behavior starts in childhood and persists into adolescence and early adulthood and those whose antisocial behavior is limited to the adolescent period (Moffitt, 1993). This LCP theory argues that early neurocognitive and psychosocial impairments in part cause persistent antisocial behavior, whereas, in contrast, AL offenders do not evidence these impairments (Moffitt, 1993; Moffitt, Caspi, Rutter, & Silva, 2001). Several studies have confirmed that LCP offenders in particular have neurocognitive impairments (Donnellan, Ge, & Wenk, 2000; Fergusson, Horwood, & Nagin, 2000; Kratzer & Hodgins, 1999; Moffitt & Caspi, 2001; Nagin, Farrington, & Moffitt, 1995; Piquero, 2001), although one study has failed to observe this effect (Aguilar, Sroufe, Egeland, & Carlson, 2000).

Do Neurocognitive Impairments Also Characterize CL Antisocial Individuals?

CL antisocial individuals are a fascinating but understudied group. These individuals are just as severely antisocial in childhood as the LCP group, but their antisocial behavior is significantly attenuated in adolescence (Lahey, Waldman, & McBurnett, 1999; Moffitt, Caspi, Harrington, & Milne, 2002). It is conceivable that this group is protected from antisocial behavior during adolescence by virtue of having particularly good neurocognitive functioning, as high IQ has been shown to protect against adult crime (Kandel, Mednick, Kirkegaard-Sorensen, & Hutchings, 1988). A second, competing prediction, however, is that this group will show the same neurocognitive impairments as the LCP group (Moffitt, 2002). Although this group was initially labeled as the "recovery" group (Moffitt, Caspi, Dickson, Silva, & Stanton, 1996), later analyses showed this label to be overly optimistic, because in adulthood they went on to show both low-level offending and multiple psychological functional impairments (Moffitt, 2002; Moffitt et al., 2002). Yet a third possibility is suggested by the only prior published work on neurocognitive functioning and CL antisocial behavior. This prospective study found age 3 and age 11 cognitive functioning in the CL group to be intermediate between that of nonantisocial control participants and the LCP group (Raine et al., 2002). Do CL antisocial children share the neurocognitive and psychosocial impairments found in LCP offenders, or are there important differences that could give clues to their ability to resist further antisocial behavior?

Are Impairments an Artifact of Comorbid Conditions?

The fifth unresolved issue concerns whether neurocognitive impairments in antisocial groups are an artifact of comorbid conditions. Lynam, Moffit, and Stouthamer-Loeber (1993) demon-

strated that the delinquency–low-IQ relationship was not accounted for by test motivation, school failure, and self-control in a community sample of 13-year-old boys, but other confounds need to be ruled out. Although some have argued that neurocognitive impairments characterize antisocial behavior after controlling for ADHD (e.g., Seguin, Boulerice, Harden, Tremblay, & Pihl, 1999; Seguin et al., 2002; Sergeant, Geurts, & Oosterlaan, 2002), others argue that antisocial boys lacking ADHD do not show neuropsychological impairments (e.g., Moffitt, 1990a; Speltz et al., 1999). A related question concerns whether environmental influences such as abuse, psychosocial adversity, and history of head injuries can account for neurocognitive impairments in antisocial children. Although there are strong genetic contributions to both brain structure and function (de Geus, Wright, Martin, & Boomsma, 2001; Posthuma et al., 2002), twin studies have demonstrated significant environmental influences on cognitive ability (Finkel, Pedersen, & Harris, 2002). Child abuse, psychosocial adversity, and a history of head injury are all processes that could conceivably cause neurocognitive impairments yet are not frequently examined. For example, although at least one prior study has shown that neuropsychological impairments in aggressive boys are independent of family adversity (Seguin, Pihl, Harden, & Tremblay, 1995), few studies have assessed whether increased abuse in antisocial groups could account for neurocognitive–antisocial relationships. Both male and female delinquent populations have been found to have significantly higher rates of trauma exposure than control groups (Cauffman, Feldman, Waterman, & Steiner, 1998; Steiner, Garcia, & Matthews, 1997), and traumatized patients have been reported to have neuropsychological impairments, particularly those related to hippocampal functioning (Bremner, 1999; Bremner, Randall, Vermetten, & Staib, 1997). Consequently, neurocognitive impairments in antisocial groups could be a function of abuse history or comorbidity with ADHD.

The current study attempts to make a partial contribution to addressing these gaps in the neurocognitive literature on antisocial behavior using a community sample of boys repeatedly assessed on antisocial behavior measures from age 7–17 years. Neurocognitive functioning and history of head injury were assessed at age 17, whereas environmental factors and ADHD were assessed at age 7 years. Because the creation of antisocial groups frequently relies on the use of cut-off scores that can seem arbitrary, cluster analysis was used as a statistical technique to obviate the use of cut-offs and to assess whether LCP, AL, CL, and nonantisocial control groups emerge when groupings are not "forced" into this mold by artificial cut-offs on antisocial measures.

Method

Participants

Participants consisted of the youngest of the three samples making up the Pittsburgh, PA, Youth Study. Full details of background characteristics and initial participant recruitment in 1987–1988, when children (all male) were aged 7 years, are given in Loeber, Farrington, Stouthamer-Loeber, and van Kammen's (1998) work. Briefly, 868 Grade 1 boys from public schools in Pittsburgh, PA, were assessed by caretakers, teachers, and the boys themselves on 21 serious antisocial behaviors. The 250 most antisocial boys were selected for further study, together with 253 boys randomly selected from the remainder, to make a total sample of 503. As such, this population-based community sample, although representative of the children in public schools in the city of Pittsburgh, PA, was nevertheless weighted toward containing more antisocial boys.

Of the original sample of 503 individuals, 335 individuals (66.6%) participated in a substudy on the biosocial bases of aggressive and violent behavior. The 10-year attrition of 168 individuals (33.4%) for the substudy broke down as follows: 31 living out of the area; 20 in jail; 45 refused the larger Pittsburgh, PA, Youth Study; 35 refused the biosocial study; 27 repeatedly cancelled appointments; and 10 failed to decide on participation. Participants were compared with nonparticipants on initial data collected at age 7 to assess for bias. Odds ratios, confidence intervals (CI), and statistical significance were computed as follows: socioeconomic status (SES; odds ratio = 0.99, CI = 0.65–1.49, $p = .94$), ethnicity (African American vs. White: odds ratio = 1.15, CI = 0.79–1.67, $p = .46$), initial risk status (odds ratio = 1.09, CI = 0.75–1.58, $p = .64$), delinquency seriousness (no or minor delinquency vs. moderate or serious delinquency: odds ratio = 0.83, CI = 0.55–1.25, $p = .37$), and violence seriousness (no violence vs. gang fighting and attacks: odds ratio = 0.87, CI = 0.56–1.36, $p = .55$). As all odds ratios were nonsignificant, there was no evidence of selective attrition as based on early data. The 335 participants had a mean age of 16.15 years at the time of testing ($SD = 0.89$), with 41.2% Caucasian and 58.8% African American affiliation. Full written informed consent was obtained from the boys and their parents, and study protocols were approved by the Institutional Review Boards at both the University of Southern California and the University of Pittsburgh.

Delinquency Seriousness Classification Groups (Age 7–17 Years)

Delinquency measures were obtained from parents (through the use of an extended version of the Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1979), teachers (using an extended version of the Teacher Report Form; Edelbrock & Achenbach, 1984), and boys (using the Self-Report Delinquency Scale; Elliott, Ageton, Huizinga, Knowles, & Canter, 1983). Full methodological details of the classification are provided in Loeber et al.'s (1998) work. Assessments were taken every 6 months from ages 7–11 years and every year thereafter up to 17 years. At each stage, participants were classified into six levels of delinquency seriousness based on an extension of the four-stage delinquency seriousness classification (see Loeber et al., 1998). Most behaviors in the classification system were represented by more than one question and more than one respondent. Classifications were based on the most serious act reported by any of the informants.

Items from the three informant sources were weighted for seriousness using severity ratings developed by Wolfgang, Figlio, Tracy, and Singer (2002) and used to construct delinquency seriousness categories for each of the 14 assessments as follows: no delinquency (scored 0), minor delinquency at home (e.g., stealing from parents; scored 1), minor delinquency other (e.g., shoplifting; scored 2), moderately serious delinquency (e.g., gang fighting; scored 3), serious delinquency (e.g., car theft; scored 4), and serious-violent delinquency (e.g., attack to seriously hurt or kill; scored 5). The mean 1-year test–retest reliability for the classification system was 0.41 ($p < .001$).

Neuropsychological Measures (Age 17 Years)

Neuropsychological measures were interspersed throughout the test day in order to help avoid fatigue and lack of motivation. Test order was as follows: Continuous Performance Task, Wisconsin Card Sorting Task (WCST), Verbal Dichotic Listening, Verbal and Visuospatial Memory, Vocabulary, Information, Block Design, and Picture Completion.

Verbal and spatial IQ. The Vocabulary, Information, Block Design, and Picture Completion subtests of the Wechsler Intelligence Scale for Children (WISC–III; Wechsler, 1991) were administered according to manual guidelines. Total IQ was estimated from the sum of all four scales. An estimate of verbal IQ was made from the sum of Vocabulary and Information, while spatial IQ was estimated from the sum of Block Design and Picture Completion.

Verbal memory. Immediate and delayed verbal memory were assessed using the Logical Memory subscale of the Wechsler Memory Scale (Wechsler, 1945). Two stories were read to the participant, and, immediately after each story, the participant recalled as many memory units or ideas of the story as possible. Thirty minutes later, the participants were again asked to recall the stories. Separate scores were computed for immediate and delayed recall components.

Visual-spatial memory. Immediate and delayed visuospatial memory were assessed using the Visual Reproduction subtest of the Wechsler Memory Scale (Wechsler, 1945). Four cards with printed designs were shown to the participant for 5 s, after which the participant attempted to draw the design from memory. After a 30-min delay, the participant was again instructed to redraw the designs. Separate scores were computed for immediate and delayed recall components.

Frontal Functioning

Continuous Performance Task (CPT). Version 4.08 of the degraded stimulus version of the CPT (Nuechterlein, Parasuraman, & Jiang, 1983) was administered according to the author's guidelines. Visually degraded numbers ranging from 0 to 9 were flashed on a computer screen (placed 1 m from the participant in his line of vision) for 40 ms at the rate of one per second. The participants' task was to press a response button on a Gravis joystick every time they saw the figure "0" but to not respond to all other stimuli. Targets had a 0.25 probability of occurrence. After 10 presentations of the target stimulus only, participants were given two practice blocks with 80 trials/block (for a total of 160 trials). Thereafter, 6 blocks with 80 trials in each bock (for a total of 480 trials) were presented, lasting 8 min.

Hits, false alarms, mean reaction time to hits, and mean reaction times to false alarms were computed. To reduce the number of errors and the likelihood of Type I error, these variables were factor analyzed. Only one principal component accounting for 61.8% of the variance was extracted with loadings as follows: hits (.82), false alarms (−.64), reaction time to hits (−.81), and reaction time to false alarms (−.86). Factor scores from the first principal component were calculated using the regression method to assess CPT performance, with higher scores indicating better performance. Coefficient alpha for this composite was 0.88.

WCST. A computerized version of the WCST (Grant & Berg, 1948) was administered in which participants sorted a pack of 64 cards according to color, shape, and number. Visual feedback (right or wrong) was provided after each card placement. This task reflects abstract reasoning, cognitive flexibility, and the ability to maintain and change set. Number correct, number of perseverative errors, number of nonperseverative errors, number of categories achieved, trials to complete the first category, and failure to learn set were computed.

Factor analysis using principal-component analysis and Varimax rotation produced two factors. The first (55.0% of variance) had loadings of correct answers (.88), perseverative errors (−.73), nonperseverative errors (−.65), categories (.92), and trials to complete first categories (−.72). The second (21.1% variance) had inconsistent loadings of failure to learn set (−.96), number correct (−.45), and nonperseverative errors (.45). Because some of these latter loadings were inconsistent (i.e., failure to learn set associated with more correct), only factor scores were saved from the first factor, with high scores reflecting better WCST performance. Coefficient alpha for this composite was .84.

Verbal Dichotic Listening. Stimuli consisted of pairs of consonants and vowels (*ba*, *da*, *ga*, *pa*, *ta*, and *ka*) of 350-ms duration and 85-dB intensity, which had been synthesized to closely align their temporal and spectral characteristics (Raine, O'Brien, Smiley, Scerbo, & Chen, 1990). The six consonant–vowel stimuli were paired with each other to form a total of l5 trials with the same pairings repeated on a second sequence of 15 trials. After 30 trials, ear of presentation was reversed and the sequence repeated to counterbalance for any channel differences on the headphones, making a total of 60 trials. Pairs were randomized within this 30-trial sequence with the proviso that no stimulus should occur on three successive trials. Intertrial interval was set at 6 s. On each trial, one consonant–vowel stimulus was presented to the left ear and a different consonant–vowel stimulus was presented to the right ear. The participants' task in this experiment was to report verbally the one stimulus they heard. Because there are six possible responses (the six pairs of consonants and vowels) for each trial and because there are two possible correct responses, chance performance on the set of 60 trials is 20 (33%).

Potential Psychiatric and Psychosocial Confounds (Age 7–17 Years)

Attention deficit hyperactivity disorder (ADHD). A diagnosis of *DSM–III–R* ADHD was assessed when the child was aged 7 years through the revised Diagnostic Interview for Children—Parent version (DISC–P; Costello, Edelbrock, Kalas, Kessler, & Klaric, 1982) administered to the mother. To meet diagnostic criteria, individuals had to have present at least 8 out of 14 behaviors, with at least 6 months duration of the disorder. The base rate of ADHD in this sample at this age was 14.1%. To help ensure standardization of the administration of this structured instrument, we made sure that interviewers were given practice on the instrument in the laboratory and were then monitored by a supervisor on their initial field interviews. This instrument has been found to have acceptable levels of reliability and validity (Jensen et al., 1995; Shaffer et al., 1996), and support for the validity of parent report of child psychopathology is given in works by Loeber, Green, Lahey, and Stouthamer-Loeber (1989) and Loeber, Green, and Lahey (1990).

Child abuse. Court-reported child abuse data (from birth to 13 years) was collected from official records at the Children and Youth Services (CYS) offices of Allegheny County, PA, in 1993. This information covered the time span from the participant's birth to the time of data collection (age 13 years). Substantiated maltreatment was defined according to the Maltreatment Classification System (Barnett, Manly, & Cicchetti, 1993) and covered all forms of significant child abuse (e.g., physical, sexual, neglect, emotional maltreatment) requiring the intervention of Children and Youth Services.

Psychosocial adversity. Measures of psychosocial adversity were derived from interviews of the boy's caregiver when he was aged 7 years. Nineteen measures were selected that reflect items similar to those contained in psychosocial adversity indices used previously by others (Moffitt, 1990a; Rutter, 1978). Full description of these variables and their coding are outlined in Loeber et al.'s (1998) work.

Because (a) many of these characteristics reflect single items, (b) usual measures of reliability and validity could not be derived, and (c) Type I error posed a problem, a factor analytic approach was taken to the establishment of psychosocial adversity constructs using principal-component analysis and an Oblimin rotation. Missing data on these variables varied from 1 (0.3%) to 45 (13.4%) participants. To prevent major data loss in the factor analysis requiring complete data on all participants, we replaced missing values with the variable mean. Although seven factors had eigenvalues > 1, the scree test indicated three factors. Factors 4–7 were ill-defined and often loaded only two items, items which were represented in Factors 1–3. Consequently, only the 1st three factors were rotated using an Oblimin criterion.

Factor 1 (Poverty) was defined by family on welfare (.75), nonintact family (.64), bad neighborhood (.62), low SES (.61), few rooms (.53), teenage mother (.51), mother poorly educated (.47), and mother unemployed (.41).

Factor 2 (Parental Psychopathology) was defined by parent anxious/depressed (.78), parental substance abuse (.75), father behavior problems (.74), and nonintact family (.51)

Factor 3 (Parental Neglect) was defined by bad relationship with primary caregiver (.57), little time spent with child (.55), caretaker does not enjoy child (.50), little positive parenting (.50), caretaker does not talk to child about activities (.50), few shared family activities (.45), low SES (.48), poor education of mother (.42), poor parental supervision (.35), and parental stress (.34).

All 19 variables loaded on one of the three factors, and the direction of loadings were in every case theoretically consistent, although simple structure was not obtained as 3 variables loaded on two factors. In these cases, the variable was deleted from the factor on which it loaded least highly. All 19 variables were *z* transformed, and variables loading on each factor were summated in order to operationalize the three factors of psychosocial adversity. Coefficient alphas for each of the three composites were as follows: poverty (.70), parental psychopathology (.69), and parental neglect (.56).

Head injury. A lifetime history of head injury was assessed in an interview with the participant using a structured head injury questionnaire (Raine, Lencz, Bihrle, LaCasse, & Colletti, 2000). The following variables were coded: number of head injuries suffered (from falls, traffic accidents, playing sports, fighting, being attacked), number of times experiencing nausea or vomiting after head injury, number of times knocked unconscious, total number of minutes of unconsciousness, hospitalization for head injury (yes/no), and number of days hospitalized.

Statistical Analyses

Hierarchical cluster analysis was conducted on the age 7–17 year delinquency data for 325 participants. Ward's method was used to optimize minimum variance between clusters in conjunction with a squared Euclidian distance measure to assess similarity between participants. Inspection of the dendrogram and fusion coefficients were used to help identify number of clusters (Everitt, 1993). Omnibus multivariate analyses of variance (MANOVAs) were conducted on each set of variables and one-way analyses of variance ANOVAs on individual measures were followed up with Tukey's honestly significant difference tests to minimize family-wise error rates and to help reduce Type I error. For chi-square analyses conducted on the six possible pairings across the four participant groups on an individual measure, a Bonferroni correction was applied ($\alpha = .0083$). Effect sizes were computed using Cohen's *d* for *t* tests (J. Cohen, 1988) and η^2 for MANOVAs.

Results

Cluster Analysis

Inspection of the dendrogram indicated two large clusters (antisocial and nonantisocial groups), as well as four clusters with *n* sizes of 156, 57, 68, and 44. Similarly, inspection of the plot of fusion coefficients indicated a large jump in values at the two-cluster level as well as a jump at the four-cluster level. Antisocial behavior data for these four clusters across ages 7–17 years are shown in Figure 1. Groups differed significantly on delinquency scores at all ages, $F(3, 321) > 24.4$, $p < .0001$. It can be seen that one group (control participants, $n = 156$) remains stably low on antisocial behavior from age 7 to 17. A second group (on the AL path, $n = 68$) starts off at exactly the same level as the control participants but progresses to significant levels of antisocial behavior by late adolescence. A third group (on the CL path, $n = 57$) starts off with high levels of antisocial behavior up to age 11 but then declines. A fourth group (on the LCP path, $n = 44$) starts off high and shows even higher levels of antisocial behavior during late adolescence. Because of the theoretical interest in the composition of these four clusters, they were retained for further validation analyses.

Antisocial Group Differences on Neurocognitive Measures

An omnibus MANOVA conducted on all neurocognitive variables produced a significant main group effect, $F(24, 915) = 2.46$, $p < .0001$, $\eta^2 = .061$, indicating that groups differed on overall neurocognitive functioning.

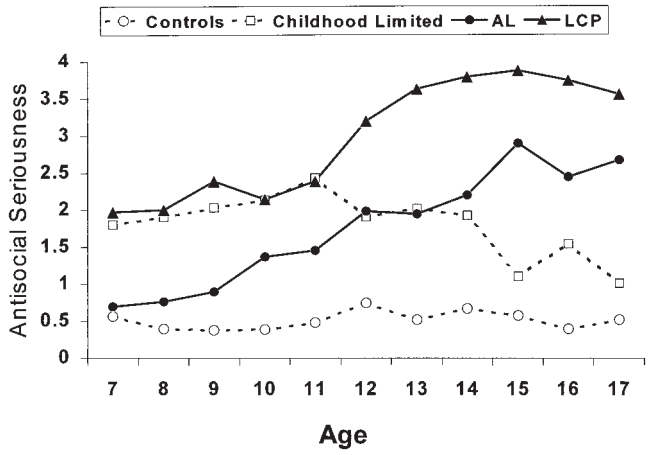

Figure 1. Group mean delinquency seriousness scores for the four clusters from ages 7 to 17 years. AL = adolescent limited; LCP = life-course persistent.

Intelligence. A MANOVA performed on verbal and spatial IQ showed a main group effect (see Table 1). Groups differed significantly on both forms of intelligence. Specifically, the LCP group had lower verbal ($d = 0.65$) and total ($d = 0.56$) IQs than control participants and additionally had lower verbal IQs compared with ALs ($d = 0.53$). The CL group also scored lower than control individuals on all three summary IQ measures (mean $d = 0.44$). In contrast, the AL group showed no significant differences compared with control participants on any of the three measures. A similar pattern of findings was observed for the four intelligence subtests (see Table 1), with the effect for Block Design being somewhat stronger than for Picture Completion. In addition, the LCP group showed significantly impaired functioning on Block Design compared with control participants ($d = 0.69$), in part explaining the nonsignificant reduction (after correction for family-wise error rate) in spatial IQ ($p = .023$, $d = 0.37$) in this group compared with control participants.

Memory. Groups differed significantly on verbal and spatial memory, both in terms of immediate and delayed recall (see Table 1). The LCP group showed spatial (mean $d = 0.73$) but not verbal memory impairments compared with control participants, while the CL group showed impairments in three of the four measures (mean $d = 0.48$) compared with control participants.

Frontal functions. Individual ANOVAs were conducted on the WCST and CPT because they reflect distinct features of frontal lobe functions. The ANOVA for the CPT was significant ($p = .011$) while that for the WCST was not ($p = .56$).[1] On the CPT, the LCP group performed more poorly than control participants ($d = 0.52$, see Table 1). The CL group showed a trend for poorer

[1] Means, standard deviations (in parentheses), and results of one-way analyses of variance for the control participants and childhood-limited, adolescent-limited, and life-course persistent groups on two commonly reported WCST indices were as follows: perseverative errors, 10.8 (10.8), 12.5 (7.5), 11.1 (6.0), 12.2 (8.1), $F(3, 311) = 1.1$, $p > .34$; categories achieved, 2.6 (1.3), 2.4 (1.2), 2.4 (1.2), 2.2 (1.2), $F(3, 311) = 1.2$, $p > .30$.

Table 1
Group Comparisons on Neurocognitive Measures

Neurocognitive measure	Controls (C; $n = 156$)	Childhood limited (CL; $n = 57$)	Adolescent limited (AL; $n = 68$)	Life-course persistent (LCP; $n = 44$)	F	dfs	p	Contrasts[a]
Intelligence								
Multivariate[b]					3.4	6, 642	.002	
Information	9.4	7.7	8.1	7.1	10.4	3, 321	.0001	LCP, CL, AL < C
	(3.0)	(2.9)	(3.0)	(2.5)				
Vocabulary	8.1	6.5	7.2	7.0	4.1	3, 321	.007	CL < C
	(2.9)	(3.0)	(2.8)	(4.4)				
Block Design	8.4	6.3	7.3	5.8	8.6	3, 321	.0001	LCP, CL < C
	(3.8)	(3.4)	(3.5)	(3.5)				
Picture Completion	11.5	9.8	10.8	10.5	4.6	3, 321	.003	CL < C
	(3.2)	(3.0)	(3.8)	(2.9)				
Verbal IQ	91.9	85.1	89.4	81.5	6.1	3, 321	.0001	LCP < C, AL CL < C
	(16.5)	(15.9)	(15.2)	(14.7)				
Spatial IQ	98.9	90.1	95.4	91.1	3.6	3, 321	.013	CL < C
	(21.4)	(17.2)	(18.9)	(18.9)				
Total IQ	94.6	86.2	91.3	84.6	5.5	3, 321	.001	LCP, CL < C
	(18.5)	(16.8)	(16.8)	(16.5)				
Memory								
Multivariate					3.0	12, 960	.0001	
Verbal: immediate	21.2	17.9	18.2	19.4	4.5	3, 321	.004	CL, AL < C
	(7.3)	(7.2)	(7.8)	(6.9)				
Verbal: delayed	17.5	13.7	15.0	16.4	4.4	3, 321	.005	CL < C
	(7.3)	(7.3)	(7.9)	(7.1)				
Spatial: immediate	34.2	32.4	32.3	30.8	6.9	3, 321	.0001	LCP, AL < C
	(4.3)	(5.3)	(5.9)	(4.6)				
Spatial: delayed	31.1	28.0	28.9	26.8	6.4	3, 321	.0001	LCP, CL < C
	(6.1)	(7.3)	(7.7)	(7.2)				
Frontal								
WCST	.08	−.01	−.04	−.15	0.7	3, 311	.56	
	(1.0)	(1.1)	(1.0)	(0.9)				
CPT	.17	−.19	−.03	−.34	3.7	3, 311	.011	LCP < C
	(.97)	(1.1)	(.93)	(1.1)				
Dichotic Listening								
Multivariate					2.5	6, 640	.023	
Left	31.5	30.3	30.3	29.9	1.8	3, 320	.15	
	(4.9)	(5.0)	(5.6)	(4.3)				
Right	35.2	32.8	35.6	35.3	3.2	3, 320	.023	CL < C, AL
	(5.6)	(5.9)	(5.8)	(4.4)				

Note. WCST = Wisconsin Card Sorting Task; CPT = Continuous Performance Test.
[a] Using Tukey's honestly significant difference test. [b] Only verbal and spatial IQ measures are entered into the multivariate analysis of variance.

CPT functioning compared with control participants (uncorrected $p = .027$, $d = 0.37$).

Dichotic listening. The MANOVA indicated an overall group effect with respect to performance on the Dichotic Listening Task (see Table 1). In particular, groups differed on right-ear performance, with the CL group performing at significantly lower levels compared with the control ($d = 0.42$) and AL ($d = 0.47$) groups (see Table 1).

Antisocial Group Differences on Child Abuse, Adversity, Head Injury, and ADHD

Abuse. Groups differed significantly in rates of court-recorded abuse (see Table 2). Specifically, the LCP group had almost four times the rate of abuse than control participants and were significantly higher (2.7-fold increase) than the AL group who did not differ from control participants. The CL group had a significantly higher (2.4-fold) abuse rate than control participants but did not differ from the LCP group ($p < .12$).

Psychosocial adversity. Groups differed significantly on all three measures of psychosocial adversity (see Table 2). LCP offenders had higher levels of poverty ($d = 0.45$) than control participants and higher rates of neglect than both control participants ($d = 0.78$) and AL offenders ($d = 0.46$), although rates of parental psychopathology were not high in this group. In contrast, the AL group was not characterized by adversity. The CL group was, however, characterized by higher scores on all three factors of adversity compared with control participants (mean $d = 0.47$) but did not differ from the LCP group on any measure.

History of head injury. The MANOVA was nonsignificant (see Table 2), but as head injury was viewed as a potential confound, to be conservative, we nevertheless computed univariate F tests to assess whether the LCP group would be characterized by more head injuries as well as uncorrected post hoc comparisons.

Table 2
Group Comparisons on Psychosocial, Abuse, ADHD, and Head Injury Variables

Possible confound	Controls (C; n = 156)	Childhood limited (CL; n = 57)	Adolescent limited (AL; n = 68)	Life-course persistent (LCP; n = 44)	F	dfs	p	Contrasts
ADHD								
% with diagnosis	14.8	28.1	11.8	31.8	χ^2 = 11.9	3	.008	LCP, CL > C, AL
Child abuse								
% abused	11.5	28.1	16.2	43.2	χ^2 = 25.0	3	.0001	LCP > C, AL CL > C
Psychosocial adversity								
Multivariate					5.4	9, 963	.0001	
Poverty	−1.65	.15	−.68	−.12	9.1	3, 321	.002	LCP > C
	(3.5)	(3.5)	(3.9)	(3.1)				CL > C
Parental psychopathology	−.39	.60	.31	−.08	3.2	3, 321	.021	CL > C
	(2.0)	(2.5)	(2.6)	(2.1)				
Neglect	−.96	.54	−.12	1.7	8.4	3, 321	.0001	LCP > C, AL
	(3.2)	(3.7)	(3.8)	(4.2)				CL > C
Head injury								
Multivariate					0.9	21, 246	.54	
No. of head injuries	1.26	1.32	1.36	1.36	0.3	3, 321	.83	
	(2.3)	(1.8)	(2.4)	(2.1)				
No. of unconscious	0.37	0.33	0.44	0.70	2.6	3, 321	.06	LCP > C, CL
	(0.7)	(0.61)	(.85)	(1.02)				
No. of min. unconscious	5.8	8.9	92.6	48.0	1.2	3, 321	.32	
	(34.2)	(29.9)	(703.9)	(220.0)				
% hospitalized	38.0	42.0	31.0	43.0	0.8	3, 321	.51	
	(0.49)	(0.50)	(0.47)	(0.50)				
No. of days in hospital	0.21	2.38	1.40	0.75	1.4	3, 321	.26	
	(0.78)	(12.5)	(10.9)	(2.46)				
No. of disoriented/sick	0.42	0.28	0.16	0.15	0.6	3, 321	.59	
	(2.15)	(1.11)	(0.53)	(0.55)				

Note. ADHD = attention-deficit/hyperactivity disorder; min. = minutes.

On only one variable (number of times knocked unconscious) was there any evidence that the LCP group had a greater history of head injuries compared with control (d = 0.41) and CL (d = 0.46) groups (see Table 2).

ADHD. There was a significant relationship between ADHD and antisocial grouping, $\chi^2(3) = 11.9$, $p = .008$; see Table 2). Rates of ADHD were twice as high in both LCP (31.8%) and CL (28.1%) groups as compared with control (14.8%, $p = .011$ and .027, respectively) and AL (11.8%, $p = .009$ and .021, respectively) groups. These comparisons were all statistically nonsignificant after applying a Bonferroni correction ($p = .0083$; see Table 2).

Influence of Possible Confounds on Delinquency–Neurocognitive Relationships

Because of the higher rates of ADHD in both LCP and CL groups, ADHD was entered as a covariate in an omnibus MANOVA, with all neurocognitive variables as the dependent variables and with antisocial grouping as the independent variable. The main group effect remained significant, $F(24, 909) = 2.3$, $p = .0001$, with η^2 at .057 being almost unchanged from the prior (ADHD uncorrected) level of .061. Consequently, antisocial–neurocognitive links were independent of the influence of ADHD.

The LCP group also had higher rates of court-recorded abuse than control participants. After entering abuse as a covariate, the main group effect remained significant, $F(18, 921) = 2.4$, $d = 21$, $p = .001$, $\eta^2 = .051$.

In assessing whether psychosocial adversity was a confound, after simultaneous entry of poverty, parental psychopathology, and neglect as covariates in the MANOVA, we discovered that the main effect of group remained significant, $F(24, 906) = 2.04$, $p = .002$, $\eta^2 = .051$, indicating that group differences in neurocognitive functioning were largely independent of psychosocial adversity.

Because there was some limited evidence that LCP offenders were more likely to be knocked unconscious compared with control participants and the CL group, this variable was entered as a covariate in the omnibus MANOVA on neurocognitive variables. The main effect of group remained significant, $F(18, 921) = 2.8$, $p < .0001$.[2]

Discussion

Results of this study provide initial answers to the five questions posed above. First, community offenders show spatial as well as verbal impairments. Second, they are impaired on memory as well as nonmemory cognitive tasks. Third, LCP offenders are particularly impaired on both neurocognitive and psychosocial factors relative to control participants. Fourth, CL antisocial individuals possess neurocognitive impairments. Fifth, impairments are not attributable to ADHD, child abuse, psychosocial adversity, or head

[2] Analyses were rerun for the individual neurocognitive tests after controlling for confounds and pairwise contrasts remain unchanged.

injury. These findings provide partial support for the LCP versus AL theory of antisocial behavior, indicate that these neurocognitive impairments are profound and not easily explained away by confounds and artifacts, and suggest that CL antisocials may not be free of long-lasting functional impairment.

The fact that group differences arose on spatial in addition to verbal neurocognitive measures questions the strong emphasis in the literature on verbal impairments in antisocial and delinquent populations. Specifically, group differences were found for spatial IQ, spatial memory, and visuospatial continuous performance. These findings on community antisocial individuals are consistent with other community studies of antisocial children that do find evidence for spatial impairments (see Raine et al., 2002, for a review). Although both verbal and spatial IQ impairments have been found to characterize persistently antisocial children from ages 8 to 17 years, these children show spatial, but not verbal, impairments at age 3 years (Raine et al., 2002). Early spatial impairments have been hypothesized to reflect disruption in right hemisphere affect regulation, which predisposes individuals to persistent antisocial behavior by interfering with early bonding and attachment (Raine et al., 2002). The fact that spatial neurocognitive impairments (both in terms of Block Design and memory functions) were found in the present community sample of LCP individuals provides further support for the potential significance of spatial impairments as a risk factor for such behavior and encourages their further assessment in future neuropsychological studies of antisocial groups. At the same time, the extent of the spatial impairments in the LCP (mean Cohen's $d = 0.58$) and CL groups ($d = 0.42$) suggests that the impairment is relative rather than absolute and is moderate in size (J. Cohen, 1988). Because there are stronger motor components in the tasks that most discriminated the LCP group from control participants ($d = 0.77$ for visual reproduction, $d = 0.69$ for block design), compared with a reduced motor component in the CPT ($d = 0.52$) and almost no motor component in Picture Completion ($d = 0.32$), future studies could further test the hypothesis that visuomotor ability is particularly impaired in the LCP group, as opposed to conceptual, constructional, or speed components of spatial ability.

Memory impairments were also observed in the antisocial groups, with spatial memory impairments being in relative terms the strongest of all neurocognitive measures. Such memory impairments have been traditionally associated with temporal lobe dysfunction, particularly to mesial temporal lobe structures such as the hippocampus. Recent brain-imaging research has uncovered evidence that the hippocampus is both structurally and functionally impaired in murderers (Raine et al., 1997), violent offenders (Soderstrom, Tullberg, Wikkelsoe, Ekholm, & Forsman, 2000), violent inpatients (Critchley et al., 2000), alcoholic psychopaths (Laakso et al., 2001), and unsuccessful psychopaths (Raine et al., 2004), while reduced blood flow in the right temporal cortex has also been observed in abused violent offenders during performance of a working-memory task (Raine et al., 2001). Extensive animal research has also found that the hippocampus, along with the amygdala and other subcortical structures, regulates the intensity of rage and aggression exhibited by cats (Gregg & Siegel, 2001) and that lesions to the septal-hippocampal-frontal system result in behavioral disinhibition and a hypersensitivity to immediate reward (see Gorenstein & Newman, 1980, for review). Furthermore, hippocampal lesions applied at birth in rats result in increased aggressive behavior in adulthood (Becker, Grecksch, Bernstein, Hollt, & Bogerts, 1999). Consequently, widespread memory impairments may be associated with antisocial behavior because they may be a marker for hippocampal dysfunction, which impairs affect regulation and inhibitory processes; these processes, in turn, may predispose individuals to antisocial behavior. Furthermore, such memory impairment in LCP offenders would theoretically be consistent with the view that dysfunction to the septo-hippocampal system underlies the response modulation deficits observed in psychopaths (Newman et al., in press). The facts that the hippocampus is critically important in memory and spatial representation formation and also that life-course antisocials were found in this study to have both memory and spatial impairments raises the hypothesis that hippocampal impairment may predispose individuals to life-course, psychopathic behavior (Newman et al., in press; Raine et al., 2004).

The finding that LCP, but not AL, offenders show neurocognitive and psychosocial impairments compared with control participants is consistent with Moffitt's LCP theory of antisocial behavior (Moffitt, 1993; Moffitt, Caspi, Rutter, & Silva, 2001). In particular, the LCP group had lower verbal IQs, more abuse, more neglect, and nonsignificantly (uncorrected $p = .009$) higher rates of ADHD than the AL group. There were, however, exceptions to this general pattern of increased risk factors in the LCP group. Specifically, the AL, but not LCP, group were more impaired than control participants on immediate memory. Furthermore, the two groups did not differ significantly on spatial IQ, although the effect was in the expected direction. These effects would not be directly predicted by Moffitt's theory. It is conceivable that while many in the AL group at age 17 will not go on to become adult offenders, some may well go on to become what have been termed "late-onset" offenders who lack childhood antisocial behavior. The limited research on these offenders shows that they possess some of the biological and social risk factors for adult crime shown by LCP offenders (Ishikawa, Raine, Lencz, Bihrle, & LaCasse, 2001). Future research studies could usefully test the hypothesis that a neurocognitively impaired subgroup of AL offenders progress to adult offending.

Neurocognitive impairments in antisocial groups were not attributable to comorbid ADHD, a finding that provides support for the notion that such impairments may be of etiological significance in shaping antisocial behavior rather than being an artifact of the comorbid condition of ADHD. In addition to this psychiatric variable, several important environmental processes that might be expected to account for the neurocognitive impairments (abuse, psychosocial adversity, history of head injury) failed to do so, findings that are consistent with other research on offenders (Nigg & Huang-Pollock, 2002; Seguin et al., 1995). Because neurocognitive functioning is significantly influenced by genetic processes and because genetic processes have been implicated in serious antisocial behavior (Rhee & Waldman, 2002), the possibility that the neurocognitive impairments are genetically mediated needs to be seriously considered in future research. In contrast, the possibility that effects are mediated by important psychosocial influences that were not measured in this study (e.g., impaired mother–infant bonding) cannot be discounted.

One interesting finding from this study is that the CL antisocial group was characterized by across-the-board neurocognitive (as well as psychosocial) impairments. Indeed, they showed significant impairments (compared with control participants) on more neurocognitive tests than the LCP group, despite the fact that they

had much lower age 17 antisocial scores than the LCP group. This finding suggests that neurocognitive impairments may be an important component of the explanation as to why this group is antisocial in the first place. The complimentary question is why this group eventually resists later antisocial behavior. An answer to this question has so far been elusive (Moffitt et al., 1996, 2002), but the present study provides a provisional pointer. The CL group was significantly lower (before correction for Type I error) than the LCP group on number of times knocked unconscious. Consequently, the absence of head injury in the lives of these children may explain why, despite the fact that they share much in common with the LCP group, they avoid a negative antisocial outcome in later life. If this finding can be replicated and extended in future studies, one implication is that prevention studies that target head injury in at-risk children may become more effective in reducing adult offending. Alternatively, future research should address the possibility, as outlined by Moffitt et al. (2002), that this group does not truly resist all forms of antisocial behavior, that they are later impaired in adult life, and that they may suffer from internalizing psychopathology.

Perhaps the most salient null result of this study is that no significant group differences were found on the WCST, a standard neuropsychological indicator of frontal impairments. The effect size between the LCP and control groups was −0.23 and consequently represents a small effect in the predicted direction. Nevertheless, this compares with a similar value of −0.28 for the WCST reported by Morgan and Lilienfeld (2000) in a meta-analytic review of frontal functioning and antisocial behavior. Consequently, the size of the effect observed, although nonsignificant, appears to be consistent with the larger literature. On the other hand, stronger frontal neurocognitive impairments were indicated in the LCP group by the CPT ($d = -0.51$). This suggests some limited support for the theoretical perspective that frontal impairments may be implicated in the etiology of antisocial behavior (Raine, 2002) but also indicates that there may be some specificity within the prefrontal cortex of the neurocognitive predisposition to antisocial behavior. In particular, positron emission tomography, near-infrared spectroscopy, and magnetoencephalography imaging studies have shown that while WCST bilaterally activates widespread regions of the prefrontal cortex (Fallgatter & Strik, 1998; Wang, Kakigi, & Hoshiyama, 2001), the CPT shows a relatively more lateralization pattern of right frontal activation consistent with broader findings of right hemisphere dominance for attention (Fallgatter & Strik, 2000; Hager, Volz, Gaser, Mentzel, Kaiser, & Sauer, 1998; Riccio, Reynolds, Lowe, & Moore, 2002). As such, the stronger effects for the CPT than the WCST in the LCP group would be consistent with a right hemisphere dysfunction hypothesis of antisocial behavior (Raine et al., 2001, 2002).

Seven limitations to this study should be acknowledged. First, female individuals were not assessed, so it is unknown whether such findings would generalize to this group. For example, recent research indicates that memory impairments are found in male, but not female, LCP offenders (Moffitt et al., 2001). Second, neurocognitive measures were not assessed prior to onset of antisocial behavior and, consequently, a direct causal pathway cannot be confirmed. Nevertheless, the fact that other studies find early neurocognitive impairments in LCP offenders (Moffitt et al., 2001; Raine et al., 2002) suggests that these impairments may play an etiological role. Third, findings pertain strictly to those on the *path* to LCP offending. Follow-up past age 17 years and into adulthood is needed to confirm that these individuals are truly LCP, although it is likely that the majority of the LCP group will retain their status into adulthood. Fourth, the neurocognitive battery was not as extensive as that used in some other studies, although it is more representative than others in that it includes memory functions. The fact that spatial impairments were found on the WISC, Wechsler Memory scale, and CPT suggests that spatial impairments are pervasive, but future studies could use a wider battery of visuospatial measures to clarify further the nature of spatial impairment in antisocial groups. Fifth, because 20 participants were in prison and could not be tested, results cannot be fully generalized to an unselected community sample, and these results place limits on the power to detect effects for the LCP group. Sixth, the measure of psychosocial adversity was based on indicators at age 7 years and may or may not reflect continued adversity up to age 16. Seventh, although we found no evidence for selective attrition on a number of demographic and antisocial variables, it is not known whether the sample is biased in terms of neurocognitive variables as these were not taken at age 7 years.

Despite these limitations, the current findings indicate that neurocognitive perspectives are especially applicable to LCP offending, that both spatial and memory impairments are salient, that neurocognitive impairments cannot be easily explained by psychiatric or psychosocial confounds, and that, instead, they may stem from very early environmental or genetic influences. Because few if any neurocognitive studies have assessed antisocial behavior developmentally in order to test a developmental theory of subtypes of antisocial behavior, it is felt that the current findings make a nontrivial contribution to the literature on neurocognition and antisocial behavior.

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Correction to Elsesser et al. (2004)

In the article "Attention, Heart Rate, and Startle Response During Exposure to Trauma-Relevant Pictures: A Comparison of Recent Trauma Victims and Patients With Posttraumatic Stress Disorder," by Karin Elsesser, Gudrun Sartory, and Axel Tackenberg (*Journal of Abnormal Psychology*, 2004, Vol. 113, No. 2, pp. 289–301), Table 1 contained an error.

On p. 293, the values in the "Control," "Without ASD," and "Total" columns of the "Attentional bias score" row of Table 1 are incorrect. The correct values are given below in bold.

Group Means (and Standard Deviations) of Age, Questionnaire Scores, Attentional Bias Derived From the Dot-Probe Task, and Prestimulus Heart Rate

	Group means				
		Recent trauma			
Variable	Control (N = 31)[a]	Without ASD (N =20)[b]	With ASD (N =17)[c]	Total (N = 37)[d]	PTSD (N =18)[e]
Age	41.19 (11.38)	43.80 (13.10)	36.18 (8.56)	40.32 (11.70)	41.72 (12.42)
State anxiety	32.13 (6.13)	35.15 (7.51)	40.41 (8.60)	37.57 (8.35)	45.17 (13.62)
Trait anxiety	31.10 (6.57)	34.95 (9.37)	39.35 (8.54)	36.97 (9.15)	49.33 (11.84)
Depression (BDI)	3.45 (3.30)	3.75 (4.17)	9.35 (6.15)	6.32 (5.83)	16.50 (9.87)
Impact of Event Scale					
Intrusion		8.40 (6.62)	13.41 (8.04)	10.70 (7.63)	20.83 (7.72)
Avoidance		6.75 (6.19)	12.94 (9.57)	9.59 (8.41)	16.06 (9.35)
Hyperarousal		7.90 (5.98)	13.88 (7.73)	10.65 (7.39)	19.11 (9.16)
Attentional bias score	**10.68 (49.89)**	**8.08 (42.46)**	−4.88 (42.93)	**1.96 (42.57)**	17.93 (56.92)
Prestimulus HR (bpm)	71.60 (8.86)	72.21 (13.12)	72.12 (6.42)	72.63 (10.03)	65.41 (8.98)

Note. Positive scores of the attentional bias variable indicate attention directed toward the trauma-related pictures and negative scores attention directed away. ASD = acute stress disorder; PTSD = posttraumatic stress disorder; BDI = Beck Depression Inventory; HR = heart rate; bpm = beats per minute.
[a] 15 men, 16 women. [b] 11 men, 9 women. [c] 7 men, 10 women. [d] 18 men, 19 women. [e] 8 men, 10 women.