Development and Psychopathology, **14** (2002), 25–44
Copyright © 2002 Cambridge University Press
Printed in the United States of America

Spatial but not verbal cognitive deficits at age 3 years in persistently antisocial individuals

ADRIAN RAINE,[a] PAULINE S. YARALIAN,[a] CHANDRA REYNOLDS,[a] PETER H. VENABLES,[a,b] AND SARNOFF A. MEDNICK[a]
[a]*University of Southern California; and* [b]*University of York*

Abstract
Previous studies have repeatedly shown verbal intelligence deficits in adolescent antisocial individuals, but it is not known whether these deficits are in place prior to kindergarten or, alternatively, whether they are acquired throughout childhood. This study assesses whether cognitive deficits occur as early as age 3 years and whether they are specific to persistently antisocial individuals. Verbal and spatial abilities were assessed at ages 3 and 11 years in 330 male and female children, while antisocial behavior was assessed at ages 8 and 17 years. Persistently antisocial individuals ($N = 47$) had spatial deficits in the absence of verbal deficits at age 3 years compared to comparisons ($N = 133$), and also spatial and verbal deficits at age 11 years. Age 3 spatial deficits were independent of social adversity, early hyperactivity, poor test motivation, poor test comprehension, and social discomfort during testing, and they were found in females as well as males. Findings suggest that early spatial deficits contribute to persistent antisocial behavior whereas verbal deficits are developmentally acquired. An early-starter model is proposed whereby early spatial impairments interfere with early bonding and attachment, reflect disrupted right hemisphere affect regulation and expression, and predispose to later persistent antisocial behavior.

Reviews of cognitive and neuropsychological deficits in delinquents and criminals have highlighted the repeated finding that antisocial offenders have lower IQs than nonoffenders (Binder, 1988; Moffitt, 1993a, 1993b; Raine, 1993; Wilson & Herrnstein, 1985). Despite literally hundreds of studies supporting this finding (over 350 prior to 1931; Binder, 1988), surprisingly little is known on whether these intellectual deficits *precede* the onset of delinquency. Specifically, are cognitive deficits firmly in place prior to kindergarten age before the onset of conduct disorders, or do they develop as a consequence of antisocial behavior and its concomitant substance abuse and history of head injury which interfere with schooling and the development of verbal intellectual abilities?

A second and related issue concerns the question of the specificity of cognitive deficits in antisocial individuals to verbal IQ. Cross-sectional studies of juvenile delinquents are frequently cited as supporting the view that there are verbal, but not spatial, cognitive deficits in antisocial groups (Binder, 1988; Moffitt, 1990; Moffitt, 1993b; Quay, 1987; Raine, 1993; Rutter, Giller, & Hagell, 1998; Wil-

This research was conducted with the support of a Research Scientist Development Award (K02 MH01114–01) and grants from NIMH (RO1 MH 46435–02) and the Borchard Foundation (to A. R.); grants from the Medical Research Council, Leverhulme Trust, and Mental Health Foundation (to P. V.); a Research Scientist Award (to S. M.) from NIMH (5 K05MH00619–08); and a grant from the Ministry of Health of the Mauritian Government. We wish to thank Marie-Clare Calambay, Meena Calinghen, Athene Chiriaca, Cyril Dalais, and Devi Jaganathen for help in data collection, and Frank Manis for comments on the manuscript.

Address correspondence and reprint requests to: Adrian Raine, Department of Psychology, University of Southern California, Los Angeles, CA 90089-1061; E-mail: raine@usc.edu.

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

son & Herrnstein, 1985). A closer look at this older literature indicates that antisocial groups do not have the intact spatial functions that they are commonly believed to have and that evidence for spatial deficits has been overlooked. For example, Berman and Siegal (1976) found a verbal–performance IQ deficit in juvenile delinquents, but they found not only a significant 8-point reduction in spatial IQ in delinquents compared to controls but also that performance IQ makes a more powerful contribution in predicting group membership than verbal IQ. Similarly, Virkkunen and Luukonen (1977) found significant reductions in *both* spatial and verbal IQ in individuals with antisocial personality disorder compared to controls with other personality disorders. More recent studies also provide evidence for the existence of spatial deficits in antisocial groups. The criminal offspring of criminal fathers show equivalent spatial and verbal IQ deficits compared to noncriminal controls (Kandel et al.,1988). Frost, Moffitt, and McGee (1989) show that boys with conduct disorder compared to nondisordered boys have a 9.4-point performance IQ deficit compared to a 6.0-point verbal IQ deficit, while Moffitt and Silva (1988) find that delinquents, irrespective of whether they have been caught by the police or not, have similar deficits in verbal IQ (approximately 6 points) and performance IQ (approximately 5 points) compared to nondelinquents. In this light, it is not too surprising that a recent meta-analysis of executive functions in antisocial behavior found the strongest effect size (d = .80) for a visuospatial measure (mazes) and the weakest effect size (d = .26) for a verbal measure (word fluency; Morgan and Lilienfeld, 2000).

Similarly, the few longitudinal studies starting in preschool years tend to find more evidence for spatial than verbal deficits in antisocial children from community samples. While Stattin and Klackenberg–Larsson (1993) did find early language deficits to be predictive of criminality, Moffitt (1990) reported visuomotor deficits at age 3 years as characteristic of persistently antisocial behavior. Similarly, Richman, Stevenson, and Graham (1982) found that spatial but not verbal deficits at age 3 years characterize persistently antisocial children. Feshbach and Price (1984) found no relationship between verbal IQ and aggression in kindergarten children but did find a significant relationship between low spatial IQ and increased aggression in these children. These latter findings are not consistent with the claim that verbal deficits are primary in the etiology of delinquency and that spatial deficits play little if any role (Wilson & Herrnstein, 1985). Indeed, if anything, these studies on 3-year-olds suggest that poor spatial ability represents an early correlate of antisocial behavior. Findings from these studies may differ from those of institutionalized juvenile delinquents which emphasize verbal deficits because (a) they are unselected, community samples as opposed to institutional samples; and (b) IQ is being measured much earlier in life when it is less confounded by the effects of leading a delinquent way of life.

Other studies on children have also found visuospatial deficits early in life in antisocial individuals which are equal or greater than verbal deficits at the same age. Lipsitt, Buka, and Lipsitt (1990) found a 3.1-point deficit in verbal IQ at age 7 years in children who went on to became juvenile delinquents, compared to a 4.6-point deficit in spatial IQ. Richman et al. (1982) observed an 11-point decrement in persistently antisocial boys in verbal IQ compared to a 13-point spatial deficit. Loney, Frick, Mesha, and McCoy (1998) found that children (age 6–13 years) with severe conduct problems who also show callous and unemotional traits (i.e., more psychopathic-like children) do not show a verbal IQ deficit and instead showed a trend towards a spatial IQ deficit. Dietz, Lavigne, Atrend, and Rosenbaum, (1997), in a study of 2- to 5-year-olds, found that performance IQ as well as verbal IQ were related to externalizing behavior problems. Speltz, DeKlyen, Calderon, Greenberg, and Fisher (1999) found that boys with early onset conduct disorder compared to controls score more poorly on performance as well as verbal IQ and have visuomotor as well as verbal neuropsychological impairments at age 4–5 years. Owens, Shaw, and Giovannelli (2001) found both verbal and spatial IQ deficits in 5-year-old boys from low income families with both externalizing and attention

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

problems. Taken together, these findings suggest that the current literature may be mistaken in ignoring the role of early spatial deficits in the etiology of antisocial behavior.

One possible resolution to these conflicting findings lies in the concept of life-course persistent antisocial behavior. Moffitt (1993a) has argued that neuropsychological and cognitive deficits are specific to "life-course persistent antisocial individuals," (i.e., those who both show antisocial behavior early in life and continue to show it up to age 18 years) but not those who are transiently antisocial. Thus, the reason why Moffitt (1990) found that nonverbal deficits (poor motor coordination) as early as age 3 years predicted antisocial behavior at age 15 years may be that the children were defined as life-course persistent antisocial individuals. This possibility receives support from an earlier study by Richman et al. (1982), who found that while IQ at age 3 years did not predict to antisocial behavior at age 8 years, boys who were categorized as having behavior problems at age 3 years and also classified at age 8 years as having "antisocial disorder" (N = 27) had significantly lower visuospatial ability (but not verbal ability) at age 3 years compared to boys who had behavior problems at age 3 years but who desisted from antisocial behavior at age 8 years (N = 14). Thus, this study suggests that boys who show continuity in antisocial behavior (at least throughout childhood) are characterized by spatial but not verbal deficits as early as age 3 years. Similarly, persistent delinquents who had committed delinquent acts for the previous 4–5 years were found by Yeudall, Fromm–Auch, and Davies (1982) to show *right* (not left) hemisphere neuropsychological impairments, again indicating the potential importance of taking persistency of antisocial behavior into account. Persistent antisocial individuals may therefore be especially likely to show early cognitive deficits (although see Aguilar, Sroufe, Egeland, & Carlson, 2000, for one recent failure to observe any early neuropsychological deficits in early childhood in persistent antisocial individuals, and Heller, Baker, Henker, & Hinshaw, 1996, for a failure to obtain any link between preschool IQ and externalizing problems in first grade).

Findings on spatial deficits may have been minimized in previous studies because there is currently no model to explain why such deficits should predispose to antisocial behavior. An early-starter spatial impairment model of antisocial behavior based on spatial deficits proposed here suggests that early visuospatial (right hemisphere) impairments can predispose to persistent antisocial behavior by interfering with early attachment and emotion recognition and regulation. Recent regional cerebral blood flow research on infants and young children has shown that the right (not left) hemisphere is dominant from 1 to 3 years (Chiron et al., 1997), a finding consistent with the notion that right hemisphere dominance regulates nonverbal orienting, attention, arousal, and emotional processes that are in evolutionary terms essential for survival in the preverbal infant (Saugstad, 1998). Right hemisphere dominance for facial recognition has been found as early as 4 months (De Schonen & Deruelle, 1991), while the right posterior hemisphere plays a crucial early role in mediating affective facial expression in infants (Reilly, Stiles, Larsen, & Trauner, 1995); compromising of these systems would be expected to impair both the infant's orienting to and recognition of its mother's facial expression and in turn to limit the infant's reciprocal expressive response to the mother. In transactional terms (Hinshaw & Anderson, 1996) 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 can predispose to affectionless, psychopathic-like behavior (Raine, 1993). At a later age, right hemisphere dysfunction may contribute to social information processing deficits that predisposes to antisocial behavior (Dodge, 1991). For example, right hemisphere deficits are associated with reduced ability to recognize both negative facial emotions, including anger and fear (Adolphs, Damasio, Tranel, & Damasio, 1996; Borod, St. Clair, Koff, & Alpert, 1990), and fear in prosodic information (Schmitt, Hartje, & Willmes, 1997). Inability by the growing child to accurately process and recognize signals of negative affect, such as anger and fear in a protagonist during a

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

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 reflect right hemisphere dysfunction that disrupts emotion processing and regulation, which in turn contributes to life-course antisocial and aggressive behavior.

The present study set out to contribute to the growing developmental literature on antisocial behavior by measuring verbal and spatial abilities at ages 3 and 11 years in persistently antisocial individuals (those who are antisocial throughout childhood and adolescence) and comparisons. Persistently antisocial individuals in particular should have verbal IQ and reading deficits at age 11 years compared to well-behaved comparisons, as is strongly suggested by the literature in general and Moffitt's life-course persistent theory in particular. If deficits in verbal skills are primary in the etiology of antisocial behavior, these deficits at age 11 years should also be prefaced by verbal ability deficits at age 3 years but not by spatial deficits at this age. Conversely, spatial but not verbal deficits at age 3 years would raise a serious question against conventional accounts of cognitive deficits in conduct-disordered behavior but would support a key assumption of the spatial deficits early starter model of antisocial behavior. While hypothesis testing is focused on comparisons between persistently antisocial individuals compared to nonantisocial comparisons, performance of other groups of transiently antisocial individuals (childhood limited and adolescent onset antisocial individuals) are also included for exploratory purposes.

A final important issue concerns the role of mediating factors. While some studies of juvenile delinquents and criminals have demonstrated IQ deficits after controlling for social adversity (Lynam, Moffitt, & Stouthamer–Loeber, 1993; Rutter et al., 1998), social adversity has rarely if ever been controlled in studies of early cognitive deficits in antisocial children, and one study that did control for adversity failed to find effects for verbal ability (Aguilar et al., 2000). Similarly, only two previous studies appear to have taken into account test-taking confounds such as poor attention, restlessness, and motivation (Lynam et al., 1993; Speltz et al., 1999). Consequently, the current study also set out to assess the role of social adversity and test behavior as potential mediators in any cognitive–antisocial relationship.

Method

Participants

Participants consisted of a subsample of 330 participants (177 male, 153 female) who were derived from a larger sample of 1,795 children from the island of Mauritius, a small tropical country lying in the Indian Ocean between Africa and India. These participants consisted of all those with complete data from the four test phases (ages 3, 8, 11, and 17 years) that form the focus of this study.[1] All children born in 1969 in two towns on the island were originally recruited into the study when age 3 years between September 1972 and August 1973. The larger sample consisted of males (51.4%) and females (48.6%) and with ethnic distribution as follows: 68.7% Indian, 25.7% Creoles (African origin), and 5.6% others (Chinese, English, and French). Due to the small number of Chinese, English, and French in the subsample, these groups were dropped from analyses below, which were based on the two predominant ethnic groups of Indians and Creoles. Informed consent was obtained from the mothers of the participants. A full list of variables administered to the participants across age levels is given in Table 1, while recent findings from the Mauritius Child Health Project of relevance to antisocial behavior may be found in Raine, Reynolds, Venables, and Mednick (1997), Raine, Reynolds, Venables, Mednick, and Farrington (1998), Raine, Venables, and Mednick (1997), and Scerbo, Raine, Venables, and Mednick (1995).

1. The major reason for missing data was that 1,452 children were tested at age 3 years, 1,123 at age 8 years, 1261 at age 11 years, and 606 at age 17 years. Subjects were missing at a phase due to lack of funding, not due to subject refusal, and as such the 606 tested at age 17 years were not all represented at earlier ages.

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

Table 1. *List of constructs* (*and their components*) *measured at different ages*

Age (Years)	Construct
3	Verbal ability (information, number, color, classification)
	Spatial ability (block assembly and copying shapes)
	Social adversity (father uneducated, mother uneducated, semiskilled or unskilled occupation, teenage mother, single parent status, separation from parents, large family size, poor health of mother, overcrowded home)
	Test behavior (early hyperactivity, poor comprehension of test instruction, test motivation, relationship with experimenter)
8	Antisocial behavior
11	Verbal IQ (similarities and digit span)
	Spatial IQ (block design, object assembly, coding, mazes, and picture completion)
	Holborn Reading Scale
	Scholastic ability (English, French, mathematics, and environmental studies)
	Social adversity (rented accommodation, no electricity/water, no toys/books, no TV, poor housing, father uneducated, mother uneducated, parent psychiatrically ill, parent physically ill, teenage mother, single parent status, separation from both parents, five or more siblings, overcrowding)
17	Antisocial behavior (conduct disorder and socialized aggression)

Formation of antisocial groups

Antisocial behavior was assessed at age 8 years by teachers using the Childrens Behavior Questionnaire (CBQ; Rutter, 1967; Venables et al., 1983) and at age 17 years by parents, teachers, or employers[2] using the combined conduct disorder (CD) and socialized aggression (SA) subscales of the Revised Behavior Problem Checklist[3] (RBPC; Quay & Peterson, 1987). Both were scored using same-gender norms. The eight-item Antisocial Behavior scale from the CBQ had an α of 0.83 (M = 1.47, SD = 2.31, range 0–11) and was comprised of the following items: "truants from school," "often destroys own or others belongings," "frequently fights with other children," "irritable and quick to 'fly off the handle'," "is often disobedient," "often tells lies," "has stolen things on one or more occasions," and "bullies other children." The 39 items of the combined CD and SA scale of the RBPC had an alpha of 0.89 (M = 9.23, SD = 9.09, range 0–60).

Males in Mauritius were comparable to 12- to 16-year-olds rated by their parents from Dade County, Florida, on both CD (Ms = 7.76, 7.06, respectively) and SA scales (Ms = 2.20, 1.33, respectively) of the RBPC as indicated in the RBPC manual (Quay & Peterson, 1987). Similarly, females in Mauritius and Dade Country scored at a similar level for both CD (Ms = 7.74, 7.02, respectively) and SA (1.88, 1.61, respectively). For the CBQ, somewhat higher proportions of Mauritian males (29.6%) and females (23.7%) scored above Rutter's cutoff of 9 on the CBQ as compared to his normative sample of 9- to 13-year-old males (9.7%) and female (4.5%) schoolchildren from Aberdeen; these Mauritian rates are much lower however than 9- to 13-year-old males (77.9%) and females (67.5%) attending psychiatric clinics in Aberdeen (Rutter, 1967).

2. The source of the RBPC data was as follows: parents (N = 127), teachers (N = 146) and employers (N = 57). The source from which the RBPC data were derived did not moderate the age 3 years cognitive data, F (6, 318) = 1.54, p = .162, or the age 11 years F (12, 636) = 0.06, p = .64, cognitive data. In addition, entering source as a covariate did not abolish the group effect for either age 3 years F (3, 325) = 3.21, p = .023, or age 11 years F (6, 650) = 2.62, p = .016, cognitive data.

3. Initial findings of adult criminal data collected so far show that those with a criminal record by their late 20s (N = 46) have significantly higher scores on the RBPC antisocial measure (M = 12.43) than those without a criminal conviction (N = 560, M = 9.37, t = 2.2, p < .03), indicating some degree of validity for the age 17 years rating.

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

Four discrete groups were formed on the basis of whether participants fell into the top 33% cutoffs on either the age 8 or age 17 years antisocial measures. Comparisons (N = 133; 40.3% of total sample, 54% male, 82% Indian) were defined as those who did not fall into the top 33% of scores at both ages 8 and 17 years. The childhood-limited antisocial group (N = 90, 27.3% of total sample, 60% male, 69% Indian) were defined as those who were in the top 33% of scorers at age 8 years but not at age 17 years. The adolescent-onset group (N = 60, 18.2% of total sample, 53% male, 78% Indian) were in the top 33% of scorers at age 17 years, but not age 8 years. The persistently antisocial group (N = 47, 14.2% of total sample, 64% male, 57% Indian) fell into the top 33% of scores at both ages 8 and 17 years. Means and standard deviations for the four groups on the age 8 and 17 years antisocial behavior measures were as follows: comparisons (age 8 years: M = 0.04, SD = 0.21; age 17 years: M = 4.07, SD = 3.65), childhood limited (age 8 years: M = 2.98, SD = 2.36; age 17 years: M = 4.81, SD = 3.42); adolescent onset (age 8 years: M = 0.02, SD = 0.15; age 17 years: M = 19.50, SD = 8.05), persistently antisocial (age 8 years: M = 3.51, SD = 2.79; age 17 years: M = 19.17, SD = 9.16).

Groups differed on ethnicity (χ^2 = 11.7, df = 3, $p < .009$), with Creoles more likely than Indians to be classified as persistently antisocial. There was also a trend for group differences on sex (χ^2 = 6.7, df = 3, $p < .09$), with more males in the persistently antisocial group. Consequently, sex and ethnicity were entered as factors in analyses below to assess for any moderating effect of these demographic variables on cognitive outcome.

To assess whether this population of 330 was representative of the initial population (N = 1,795), comparisons were made between those contained in the study and the rest of the sample on cognitive, antisocial, and demographic measures. Groups did not differ on sex (χ^2 = 0.5, $p > .46$), race (χ^2 = 1.4, $p > .23$), social adversity (t = 0.9, df = 1793, $p > .36$), age 3 years verbal ability (t = 0.3, df = 1385, $p > .76$), age 3 years spatial ability (t = 0.99, df = 1450, $p > .32$), age 11 years verbal ability (t = 1.37, df = 1259, $p > .17$), age 11 years spatial ability (t = 0.80, df = 1259, $p > .44$), age 8 years antisocial behavior (t = 0.1, df = 1067, $p > .99$), and age 17 years antisocial behavior (t = 1.1, df = 604, $p > .27$). Consequently, the subsample was representative of the larger sample on these measures.

Intelligence and cognitive ability at ages 3 and 11 years

Age 3 years. Measures of verbal and spatial cognitive ability were derived from subtests of the Boehm Test of Basic Concepts—Preschool Version (Boehm, 1986). This test is designed to assess children's ability to grasp basic verbal and spatial concepts that are fundamental for early school achievement. The type of abilities and concepts that are measured include relational concepts about persons, objects, and situations; construction and copying ability; and making judgments of space, quantity, and time. Concepts are assessed at increasingly complex levels of abstraction and took approximately 30 min to administer. While testing the children at age 11 years posed few difficulties, pilot testing of the Boehm on 3-year-olds indicated that some changes in format were necessary. In addition to pilot tests in the laboratory, visits were made to the homes of children to observe them in a more natural context. This piloting led to minor modifications of the test for use with Mauritian children. For example, sugarcane sticks were used for judgments of length (Mauritius has a predominantly sugarcane-related economy), local rocks were used for judgments of size, pictures of Mauritian children were used for identification of body parts of children, and a tea set was used to assess ability to follow directions (tea drinking is ubiquitous in Mauritius).

The modified test had 6 components: (a) block assembly (making constructions from blocks; e.g., bridge, circle, tower), (b) copying shapes (copying circle, triangle, and square), (c) information (identifying body parts, pictures of boys and girls), (d) number–size–length

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

concepts (simple numeric ability, size and length discriminations), (e) color concepts (naming and pointing to different colors), and (f) classification (making discriminations between same or different objects). Many of these abilities parallel cognitive skills found in the Wechsler Preschool and Primary Scale of Intelligence (WPPSI; Wechsler, 1967), for example, copying shapes and "geometric design," same–different discriminations and "similarities." Consequently, scale construction initially followed a face validity approach to form indices of verbal and spatial ability. Each scale was first normalized by transforming the raw scores to percentiles and then finding the standard score for each percentile (Allen & Yen, 1979). Scales were then standardized to have a mean of 10 and standard deviation of 3.

Two of the scales (block assembly and copying shapes) were spatial–constructional in nature and were similar in nature to the block design and geometric design spatial tests of the WPPSI. These tests were summed and further standardized to a mean of 100 and a standard deviation of 15 to form an index of age 3 years spatial ability. The remaining six scales were verbal in nature. Some involved a verbal response (e.g., picture content, numbers) while others required verbal comprehension and knowledge of the names of objects (information). Several of the Boehm verbal tests had parallels with WPPSI verbal tests (e.g., information and Information, classification and Similarities, number–size–length and Arithmetic). These subscales were summed and standardized in the same way as the spatial tests to form an index of age 3 years verbal ability. This face validity approach to scale construction was followed up with confirmatory factor analyses (see below).

Age 11 years. Estimates of verbal and spatial IQ were assessed at age 11 years using seven subtests of the Wechsler Intelligence Scale for Children (WISC; Wechsler, 1967). Raw scores on the WISC subscales were normalized and standardized in the same way as the age 3 years scales. "Similarities" and "digit span" formed an estimate of verbal IQ, while "block design," "object assembly," "coding," "mazes," and "picture completion" formed an estimate of spatial IQ.

Scholastic ability and reading ability at age 11 years

Reading ability was assessed at age 11 years using the Holborn Reading Scale (Pumfrey, 1985). This word-recognition test consisted of 33 sentences of increasing difficulty. Total scores (number of sentences correctly read) were standardized into reading quotients. This reading measure correlated 0.54 with verbal IQ (see Table 2).

The measure of scholastic ability was based on scores on four standardized academic tests (Certificates of Primary Education), which were given to all 11-year-old children throughout the country: English, French, mathematics, and environmental studies. Scores on these tests (graded 0–5) were summed to form an overall index of scholastic ability. The correlation between scholastic ability and reading ability was $r = .69$, $N = 1094$, $p < .0001$. Intercorrelations between scholastic ability and reading scores and cognitive ability measures at age 3 and 11 years are shown in Table 3.

Social adversity

Social adversity at age 3 years. The age 3 years social adversity index was based on nine variables collected by social workers who visited the homes of the children at age 3 years. The index was created along lines similar to those described by Rutter (1978) and Moffitt (1990). A total adversity score was created by adding 1 point for each of the following nine variables: father uneducated (no schooling, 30.0%), mother uneducated (no schooling, 29.4%), semiskilled or unskilled occupation (occupational status 3 or less on an 8-point occupational scale: 0, *unemployed*; 4, *factory worker*; *cook*; 8, *academic, head of large business*; 55.5%), teenage mother (age 19 years or younger when child was born, 14.2%), single-parent status (2.1%), separation from parents (orphaned or raised by sub-

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

Table 2. *Intercorrelation between age 3 years cognitive and spatial subtests*

	Number	Block	Color	Copying	Information
Block	.195				
Color	.306	.202			
Copying	.251	.310	.260		
Information	.360	.237	.373	.306	
Classification	.292	.192	.302	.248	.355

Note: All correlations are statistically significant ($p < .0001$).

Table 3. *Correlations between age 3 and 11 years cognitive measures* (*rows*) *and age 3 years motor and verbalization measures*, *and age 11 years reading ability*, *scholastic achievement*, *and intelligence measures* (*columns*)

	Age 3 Years		Age 11 Years			
	Motor	Verbalizations	Reading	Achievement	VIQ11	PIQ11
Verbal 3 years	.09	.24	.25	.29	.25	.24
Spatial 3 years	.23	.11	.16	.25	.13	.24
Verbal 11 years	.11	.13	.54	.58	—	—
Spatial 11 years	.23	.12	.48	.59	.60	—

Note: All correlations $p < .002$; *N*s = 971–1455.

stitute mother, 0.9%), large family size (sibling order fifth or higher by age 3 years, 30.0%), poor health of mother (coded 1 on a 3-point scale: 3, *above average*; 2, *average*; 1, *below average*; 3.3%), overcrowded home (five or more family members per house room, 28.8%). Scores ranged from 0 to 7 ($M = 1.94$, $SD = 1.39$).

Social adversity at age 11 years. The age 11 years social adversity index was based on 14 variables collected by social workers who visited the homes of the children at age 11 years. A total adversity score was created by adding 1 point for each of the following 14 variables: living in rented accommodation (20.7%), house without electricity or water (15.6%), child has neither good toys nor good books (35.7%), no television (22.5%), living in poor housing (24.7%), father uneducated (30.0%), mother uneducated (29.4%), parent psychiatrically ill (4.0%), parent physically ill (2.0%), teenage mother (age 19 years or younger when child was born, 14.2%), single-parent status (8.3%), separation from both parents (1.1%), five or more siblings (30.7%), and overcrowded home (five or more family members per house room, 12.1%). Scores ranged from 0 to 6 ($M = 2.04$, $SD = 1.61$).

Sustained childhood adversity. An index of sustained social adversity throughout childhood (ages 3 to 11 years) was created by computing the product of the age 3 years and age 11 years adversity indices. High scores on this index indicated relatively high adversity at both ages, low scores indicated low adversity at both ages, and medium scores indicated moderate or unstable adversity throughout childhood.

Behavioral ratings during testing at age 3 years

During cognitive testing at age 3 years the experimenter rated components of the child's behavior using 4-point scales for the following five behaviors. Each of these behaviors were a potential confound, as they were each significantly related to age 3 years cognitive ability as follows: attention–distraction, F (6, 636) = 3.62, $p = .002$; activity level, F (6, 636) = 2.6, $p = .017$; motivation, F (6, 636) = 12.73,

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

$p < .0001$; comprehension of directions, F (6, 636) = 9.97, $p < .0001$; and ease of relationship with tester, F (6, 634) = 12.01, $p < .0001$.

Attention–Distraction. Coded: 1, *easily distracted;* 2, *some distraction with noises or movements of others;* 3, *attends to tester most of the time;* 4, *focuses attention easily*.

Activity level. Coded: 1, *out of seat, body constantly in motion;* 2, *much movement;* 3, *some squirming;* 4, *sits quietly*.

Motivation. Coded: 1, *refuses;* 2, *attempts task after much encouragement;* 3, *attempts task briefly;* 4, *persists with task*.

Comprehension of directions. Coded: 1, *does not appear to comprehend most directions;* 2, *slow to comprehend;* 3, *comprehends after several repetitions;* 4, *comprehension of most directions*.

Ease of relationship with tester. Coded: 1, *very reluctant or fearful;* 2, *shy;* 3, *warms up to tester;* 4, *immediately friendly*.

Reliability and validity of the age 3 years and age 11 years cognitive measures

Confirmatory factor analysis (CFA) was employed to assess whether the two-factor (verbal–spatial) model derived from the face validity approach (a) was a significantly better fit to the data than a one-factor (general cognitive ability) model and (b) provided a good fit to the data. The intercorrelation matrix for the six verbal and spatial subtests is given in Table 2. The two-factor verbal–spatial model was a significant improvement in fit compared to the one-factor model, χ^2 (1) = 34.79, $p < .0001$. Fit indices together with factor loadings for the two-factor model are shown in Table 4 and indicate a strong fit, with fit indices exceeding .90 and the root mean-square error of approximation falling below .10.

The two subcomponents of the age 3 years spatial measure correlated significantly with age 11 years spatial IQ (range .22–.25, $p < .0001$). Similarly, all subcomponents of the age 3 years verbal measure correlated significantly with age 11 years verbal IQ (range .19–.27, $p < .0001$). Intercorrelations between cognitive, motor, reading, and achievement measures are given in Table 3. It can be seen that the age 3 years spatial measure correlated .24 with the age 11 years spatial IQ measure, while the age 3 years verbal measure correlated .25 with the age 11 years verbal IQ measure. Age 3 years verbal ability correlated significantly with age 11 years reading ability. Both verbal and spatial ability at age 3 years measures predicted to scholastic ability at age 11 years. Age 3 years verbal ability correlated with a rating of the amount of verbalizations the child made to the experimenter at age 3 years (Raine et al., 1998), while age 3 years spatial ability correlated significantly with a measure of motor ability (jumping, hopping, balancing on one foot) at age 3 years. Data from 73 subjects who were given the Reynell Developmental Language Scale (Reynell & Huntley, 1972) at age 6 years show a .36 correlation ($p < .002$) with the age 3 years verbal ability measure compared to a .25 correlation

Table 4. *Results of confirmatory factor analysis (factor loadings and fit indices) for the two-factor model of verbal and spatial ability at age 3 years*

Factor Loadings	Spatial	Verbal
Copying	.63	—
Block	.49	—
Information	—	.66
Color	—	.56
Classification	—	.54
Number	—	.54
Fit Indices		
χ^2	42.6 ($p < .0001$)	
Bentler–Bonett Normed Fit Index	.96	
Bentler–Bonnett Non-Normed Fit Index	.94	
Comparative Fit Index	.97	
Bollen Fit Index	.97	
McDonald Fit Index	.99	
Goodness-of-Fit Index	.99	
Adjusted Goodness-of-Fit Index	.97	
Root-Mean-Square Error of Approximation	.048	

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

($p < .005$) with the spatial age 3 years measure ($p < .025$); while the difference between these two correlations in not statistically significant due to the modest sample size, the verbal age 3 years measure predicts twice the amount of variance in age 6 years language than the spatial age 3 years measure. Not given in Table 4 is the intercorrelation between age 3 years total cognitive score (verbal + spatial) and age 11 years estimated total IQ, which was .30 ($N = 969$, p < .0001). This latter correlation is modest but in keeping with the facts that (a) IQ does not stabilize until later childhood and correlations between early cognitive ability and later IQ are relatively small (Gottfried, Gottfried, Bathurst, & Guerin, 1994) and (b) different cognitive measures were used in the age 3 and 11 years test sessions.

Coefficient alpha at the subtest level for the verbal scale was .66. Coefficient alpha at the subtest level for this spatial scale was 0.46. Age 3 years verbal ability correlated significantly with age 3 years spatial ability ($r = .41$, $N = 1{,}387$, $p < .0001$).

Results

Intelligence and persistently antisocial behavior

To assess the main research question of whether the persistently antisocial group have cognitive deficits, all cognitive measures were entered into a multivariate analysis of variance (MANOVA). In order to test exploratory questions and to assess on which specific measures groups differ, one-way analyses of variance (ANOVAs) were also conducted. The MANOVA showed a main effect of group, F (12, 975) = 2.0, $p < .02$, indicting overall group differences in cognitive ability.

One-way ANOVAs were conducted on each of the four cognitive measures (verbal and spatial ability at ages 3 and 11 years). At age 3 years, there was no main group effect on verbal ability, F (3, 326) = 0.3, $p > .89$, but there was a main group effect for spatial ability, F (3, 326) = 3.4, $p < .02$ (see Figure 1). Persistently antisocial individuals had significantly lower age 3 years spatial ability than both comparisons ($t = 2.7$, $df = 178$, $p < .007$, $d = 0.46$) and adolescent-onset offenders ($t = 2.3$, $df = 135$, $p < .02$, $d = .48$).

At age 11 years, there was a significant main group effect for verbal IQ, F (3, 326) = 5.3, $p < .001$, but not for spatial IQ, F (3, 326) = 2.0, $p < .11$. A breakdown of verbal IQ showed that persistently antisocial individuals had lower age 11 years verbal IQ than both comparisons ($t = 3.8$, $df = 178$, $p < .0001$, $d = 0.65$) and childhood-limited individuals ($t = 2.3$, $df = 135$, $p < .03$, $d = 0.42$), while in addition adolescent-onset antisocial individuals scored lower than comparisons ($t = 2.5$, $df = 191$, $p < .02$, $d = 0.38$; see Figure 2). An a priori comparison of spatial ability showed significantly lower spatial ability in the persistently antisocial group than in comparisons ($t = 2.5$, $df = 178$, $p < .02$, $d = 0.42$), but the persistently antisocial and adolescent-onset groups did not differ significantly on verbal IQ ($t = 1.23$, $df = 106$, $p > .22$, $d = 0.24$).

Scholastic attainment and reading ability at age 11 years

For the overall measure of scholastic ability, there was a main effect of group, F (3, 287) = 4.0, $p < .008$, with post hoc tests indicating lower scores compared to comparisons for persistently antisocial individuals ($t = 3.3$, $df = 159$, $p < .0001$, $d = 0.61$) and adolescent-onset antisocial individuals ($t = 2.0$, $df = 171$, $p < .05$, $d = 0.34$; see Figure 3). However, these two antisocial groups did not differ significantly ($t = 1.27$, $df = 93$, $p > .21$, $d = 0.26$).

Group differences on age 11 years reading ability were marginally significant, F (3, 326) = 2.6, $p < .052$. An a priori comparison showed that persistently antisocial individuals have lower reading ability than Comparisons ($t = 2.7$, $df = 178$, $p < .007$, $d = 0.46$).

Sex and ethnicity

Although no sex or race interactions were predicted, the above omnibus MANOVA was repeated with sex and ethnicity entered as factors in order to explore possible moderating

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

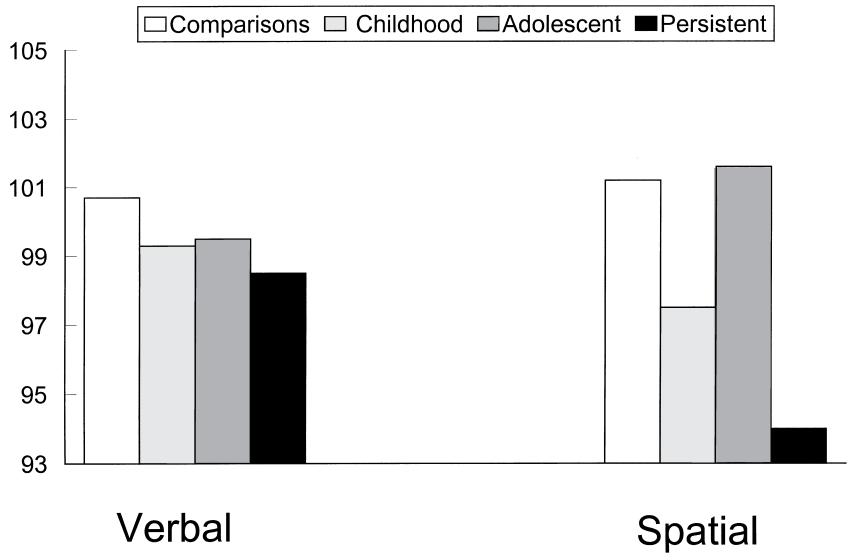


Figure 1. Age 3 years verbal and spatial ability in comparisons, childhood-limited, adolescent-onset, and persistently antisocial groups.

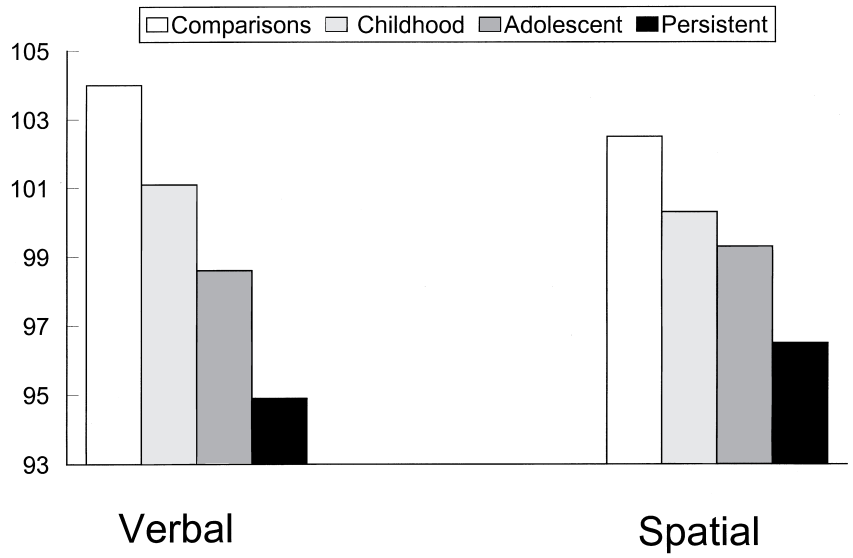


Figure 2. Age 11 years verbal and spatial ability in comparisons, childhood-limited, adolescent-onset, and persistently antisocial groups.

effects of these variables. There were no interactions between sex and group ($p > .88$) or between ethnicity and group ($p > .89$). In addition, the three-way Group × Sex × Ethnicity interaction was nonsignificant ($p > .63$). Although antisocial groups differed on ethnicity, with a trend also for differences on sex (see Methods section), these factors do not moderate persistently antisocial—cognitive relationships. In particular, the effect size for the difference between the persistently antisocial group and comparisons were very similar for

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

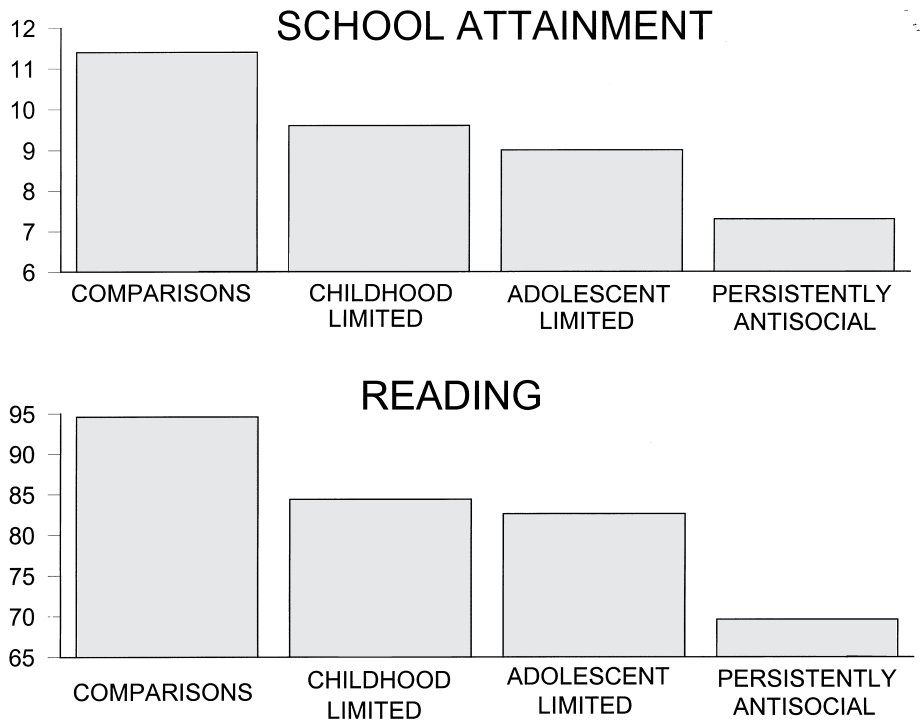


Figure 3. Age 11 years scholastic ability (upper half) and reading ability (lower half) in comparisons, childhood-limited, adolescent-onset, and persistently antisocial groups.

males and females (d = .50 d = .44 for males and females, respectively, for age 3 years spatial ability, .52 and .58 for age 11 years spatial ability, and .68 and .71 for age 11 years verbal ability).

Different cutoffs for defining persistent antisocial group

Different cutoffs (50%, 33% 25%, 15%) were used to define groups to assess whether the key finding (early spatial but not verbal deficits in persistently antisocial individuals) changes as a function of cutoff. The resulting effect size (d) for persistently antisocial versus comparison differences for age 3 years verbal and spatial ability, sample size of the persistently antisocial group, and statistical significance for each cutoff (including the 33% cutoff used in the study) are as follows: for 50% cutoff, N = 67 for persistently antisocial individuals, spatial d = 0.51 ($p < .0001$), verbal d = 0.14 ($p > .36$); for 33% cutoff, N = 47 for persistently antisocial individuals, spatial d = 0.46 ($p < .007$), verbal d = 0.13 ($p > .39$); for 25% cutoff, N = 16 for persistently antisocial individuals, spatial d = 0.54 ($p < .032$), verbal d = 0.10 ($p > .70$); for 15% cutoff, N = 11 for persistently antisocial individuals, spatial d = 0.85 ($p < .006$), verbal d = 0.094 ($p > .74$). Results indicate that the key finding is not a function of the cutoff used, although they suggest the possibility that a stronger effect size for age 3 years spatial (but not verbal) ability may be observed in future studies with larger sample sizes employing a more extreme cutoff.

Possible artifactual explanations of the IQ–persistently antisocial relationship

Persistently antisocial individuals compared to well-behaved comparisons have lower age 3 years spatial ability and lower age 11 years verbal IQ, spatial IQ, and school performance. Are these relationships artifactual? We tested a number of hypotheses in which possible mediators were entered as covariates in analyses

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

of covariance (ANCOVAs) with comparisons versus persistently antisocial individuals as the independent variable and cognitive ability as the dependent variable. We also controlled for several age 3 years behavioral measures to assess whether poor performance on age 3 years spatial tests in the persistently antisocial group could in some way be a function of factors such as early hyperactivity, poor comprehension of test instruction, low test motivation, or being ill at ease with the experimenter at this age.

After controlling for the effects of social adversity at age 3 years, group differences remained significant for age 3 years spatial ability, F (1, 177) = 7.3, $p < .007$, age 11 years verbal ability, F (1, 177) = 14.9, $p < .0001$, age 11 years spatial ability, F (1, 177) = 6.5, $p < .02$, age 11 years school performance, F (1, 158) = 11.8, $p < .001$, and age 11 years reading ability, F (1, 177) = 7.5, $p < .007$. To assess whether sustained social adversity throughout childhood could explain cognitive deficits at age 11 years (particularly age 11 years verbal deficits that were not present at age 3 years), the measure of sustained social adversity was entered as a covariate. Group differences remained significant for age 11 years verbal ability, F (1, 170) = 10.5, $p = .001$, age 11 years school performance, F (1, 153) = 9.6, $p = .002$, and age 11 years reading ability, F (1, 170) = 4.3, $p = .04$. The group difference for age 11 years spatial ability was rendered marginally significant, F (1, 170) = 2.8, $p = .095$, with the size of the original group difference reduced slightly from 6.21 to 5.68.

To assess whether group differences in early hyperactivity–attention deficit were in place by age 3 years and could account for group differences on age 3 years spatial ability, the two groups were compared on two early behavioral signs of hyperactivity (attention–distraction and movement) measured during age 3 years cognitive testing. After entering these two covariates, the group difference on spatial ability remained significant, F (1, 171) = 10.4, $p < .002$, with the difference in spatial ability increasing slightly from 8.0 to 8.3.

The rating of receptive linguistic ability taken during age 3 years testing was entered as a covariate to assess whether age 3 years spatial differences were a function of inability of the persistently antisocial group to understand test directions. The group difference remained significant, F (1, 172) = 9.7, $p < .002$, with a slight increase (8.1) in the group difference. Age 3 years test motivation was also entered as a covariate to test the hypothesis that poor age 3 years spatial performance in the persistently antisocial group was due to poor motivation. The group difference remained significant, F (1, 172) = 8.9, $p < .003$, with the group difference (7.7) being almost unchanged. Finally, ease of the child's relationship was entered as a covariate to assess whether lower spatial scores in the persistently antisocial group could be due to social discomfort. The group difference remained significant, F (1, 172) = 9.9, $p < .002$, with a slight increase in the group difference (8.1).

Prediction of persistently antisocial behavior

In order to assess whether cognitive deficits predict persistent antisocial behavior over and above demographic variables, ethnicity, sex, and social adversity, measures were entered in the first block of a logistic regression in the prediction of persistently antisocial versus comparison group membership using forward entry and the Wald chi square. These measures explained 17.1% of the variance in group membership (Nagelkerke statistic). On the second block, age 3 years and age 11 years cognitive measures were entered. Both age 3 years spatial ability ($\chi^2 = 7.47$, $df = 1$, $p < .007$) and age 11 years school performance ($\chi^2 = 12.2$, $df = 1$, $p < .0005$) significantly predicted group membership over and above demographic measures and doubled the explained variance in group membership from 17.1 to 33.3%. Alternatively, age 3 years verbal ability ($p > .85$), age 11 years verbal IQ ($p > .11$), age 11 years spatial IQ ($p > .87$), and age 11 years reading ability ($p > .12$) all failed to significantly increased variance in group membership.

Discussion

A large cross-sectional literature on cognitive deficits in delinquents and criminals has fueled the conclusion that verbal but not spatial

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

deficits[4] are of etiological significance to antisocial behavior (Binder, 1988; Moffitt, 1993b; Quay, 1987; Raine, 1993; Rutter et al., 1998; Wilson & Herrnstein, 1985). Results of this longitudinal study question this conclusion. A strong version of the verbal deficit hypothesis makes the prediction that verbal cognitive deficits will be in place as early as age 3 years before the onset of persistent antisocial behavior. Instead, persistently antisocial individuals (whether female or male) were found to have significantly lower *spatial* ability at age 3 years (as operationalized by block assembly and copying shapes) than both comparisons and adolescent-onset antisocial individuals, with normal performance on verbal ability at this early age. These conflicting findings do not stand alone. Close scrutiny of the few and infrequently cited prospective longitudinal studies based on community samples often fail to find strong support for verbal deficits and, on balance instead indicate early spatial deficits in antisocial children (Feshbach & Price, 1984; Lipsitt et al., 1990; Moffitt, 1990; Richman et al., 1982). Taken together, these findings raise a question mark against the heavy emphasis on verbal (left hemisphere) deficits as playing an etiological role in antisocial behavior and instead suggest the possible etiological role of early spatial (right hemisphere) deficits.

A key finding from this study is that persistent spatial cognitive deficits are specific to persistently antisocial individuals. While childhood-limited and adolescent-onset groups both show cognitive deficits at one age but not another, only the persistently antisocial group showed significant cognitive deficits at ages 3 and 11 years. This result in turn supports the hypothesis of Moffitt (1993a) that neurocognitive deficits are associated with persistent antisocial offending, and appears to represent the only longitudinal study to have assessed early verbal and spatial cognitive deficits at an early age in persistently antisocial individuals. While studies of adult antisocial personality are increasingly finding evidence of cognitive deficits (Barratt, Stanford, Kent, & Felthous, 1997; Bechara et al., 1997; Kosson, 1996; Newman & Schmitt, 1998), an important question in these studies concerns whether these deficits were present early in life and are thus of potential etiological significance, or alternatively whether they are the product of living an antisocial life with its associated substance abuse and head trauma. By assessing cognition as early as age 3 years, the current study circumvents these important methodological confounds. On the other hand, it must be recognized that the term "persistent" antisocial behavior in this study is operationally defined as ranging from 8 to 17 years, and while males predominated in this group, a higher than expected percentage (36%) were females. As such, this study should not be construed as a replication of Moffitt (1990) and Moffitt, Caspi, Dickson, Silva, and Stanton (1996), and it remains to be seen whether all of these individuals go on to remain persistently antisocial over the life course (Moffitt, 1993a).

The finding of early spatial deficits in persistently antisocial individuals is consistent with the early-starter spatial impairment model of antisocial behavior. Nevertheless, there are two important caveats. First, findings from this study cannot be taken to show that early spatial deficits cause later persistent antisocial behavior. Second, the notion that spatial cognitive deficits in the first 3 years of life can impair bonding assumes that there are links between cognitive deficits and quality of attachment. This assumption is supported by the meta-analysis of 25 studies that show a significant positive relationship between attachment and intelligence (van Ijzendoorn, Dijkstra, & Bus, 1997), but it should be noted that the link between attachment and conduct problems is less well established. Furthermore, the current findings do not establish that spatial deficits lead to antisocial behavior via bonding impairments, and several other pathways are feasible based on the well-established relationship between spatial functions and right hemisphere functioning. First, as noted earlier, spatial deficits may be a marker for right hemisphere dysfunction that impairs emotion recognition

4. The term "deficit" is used to signify a relative between-group reduction in test scores and does not necessarily signify brain damage or a clinical deficit.

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

and can lead children to make inappropriate responses in social encounters, escalating into aggression.

Second, reductions in right hemisphere functioning (particularly the anterior regions) have been associated with deficits in the withdrawal system, a system that promotes retreat from aversive and dangerous situations (Davidson, 1998; Davidson, Eckman, Saron, Senulis, & Friesen, 1990). Reduced right hemisphere functioning and a consequent weaker withdrawal system could make children less averse to dangerous, risky situations that increase the probability of antisocial behavior. Third, antisocial groups have been consistently found to show poor fear conditioning (Raine, 1993), and two positron emission tomography studies in humans have shown that fear conditioning preferentially activates right frontotemporal regions (Furmark, Fischer, Gustav, Larsson, & Mats, 1997; Hugdahl, 1998). Consequently, relatively weaker right hemisphere activation would predispose children to poor fear conditioning, lack of conscience development, and antisocial behavior. Fourth, the right hemisphere is viewed as dominant for the processing of pain (Hari, Portin, Kettenmann, Jousmaeki, & Kobal, 1997; Hsieh, Hannerz, & Ingvar, 1996), and stably aggressive individuals show reduced pain thresholds (Seguin, Pihl, Boulerice, & Tremblay, 1996); reduced pain thresholds could make a child less susceptible to socializing punishments. Fifth, fMRI studies in children show that response inhibition tasks activate right hemisphere frontostriatal circuitry (Casey, Castellanos, Giedd, & Marsh, 1997); children with right hemisphere (spatial) impairments would therefore be expected to be more likely to fail to inhibit antisocial behavioral responses.

When taken together, these five factors (poorer recognition of affect, poor fear conditioning, reduced pain perception, deficient withdrawal system, and poor response inhibition), especially in combination, could constitute a significant predisposition towards persistent antisocial behavior. Nevertheless, it should be emphasized that spatial ability measures derived here from 3-year-olds are only indirect neuropsychological indices of right hemisphere functioning, functioning that is not directly measured in this study. Compelling evidence from brain imaging studies do, however, support the conclusion from earlier lesion studies that visuospatial, holistic functions are subserved by the right hemisphere (Flitman, O'Grady, Cooper & Grafman, 1997; Jonides, Smith, Koeppe, & Awh, 1993; Owen, Milner, Petrides, & Evans, 1996; Smith, Jonides, & Koeppe, 1996; van Horn et al., 1998), although functional specialization of the cerebral hemispheres is by no means absolute. Furthermore, the type of spatial constructional tasks (drawing and modeling block designs) used at age 3 years have been found to be compromised in 4- to 5-year-old children who suffered pre- or perinatal right hemisphere injuries (Stiles, Stern, Trauner, & Nass, 1996; Stiles, Trauner, Engel, & Nass, 1997). In the absence of fMRI studies on 3-year-olds to more directly assess right hemisphere functioning, such neuropsychological tasks are likely to continue to serve as indirect indicators of right hemisphere functioning for this age group.

The significant but modest correlation between spatial ability at age 3 years and spatial ability at age 11 years is not surprising given developmental plasticity in growing children, yet such plasticity might dictate that persistently antisocial individuals would outgrow their early spatial deficits. Speltz et al. (1999) speculated that their young 4- to 5-year-old children with early onset conduct problems may eventually overcome their visuomotor deficits in time. This does not appear to be the case in the present sample: the persistently antisocial group had spatial deficits at age 11 years that were almost as strong (d = 0.42) as spatial deficits at age 3 years (d = 0.46). Early perinatal right hemisphere lesions lead to spatial deficits which are more intractable than the language problems associated with left hemisphere perinatal lesions (Johnson, 1999; Stiles, 1996), and this reduced recovery of function may account for the relative stability of the spatial deficit in persistently antisocial individuals.

Results of the logistic regression analyses showed that after accounting for demographic

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

variables (sex, ethnicity, and social adversity), age 3 years spatial ability significantly increased variance explained in predicting persistently antisocial versus comparison group membership. In contrast, age 11 years verbal and spatial ability did not add further to discriminating group membership after taking into account age 3 years spatial ability. These analyses suggest that while persistently antisocial individuals have lower verbal and spatial ability at age 11 years, these deficits do not predict antisocial outcome over and above demographic and early spatial deficits. Nevertheless, these findings do not necessarily negate the potential contribution of verbal deficits in predisposing to persistent antisocial behavior. It is possible that these later, developmentally acquired verbal deficits may be a result of early spatial deficits and may actively contribute to persistent antisocial behavior. Early dysfunction to the right hemisphere could lead to interhemispheric reorganization of functions whereby the left hemisphere, after becoming dominant after age 3 years (Chiron et al. 1997), takes over spatial functions in addition to its own verbal functions. Such "crowding" of the left hemisphere over time would increasingly compromise verbal abilities, while spatial functions which are now more bilaterally represented become increasingly less compromised. Such a perspective may help explain why studies of adolescent delinquents find stronger verbal than spatial deficits.

Other lines of evidence lend some support to the view that spatial deficits may contribute to later verbal deficits. Early right hemisphere damage in children is known to impair language and comprehension (Eisele, Lust, & Aram, 1998; Trauner, Ballantyne, Friedland, & Chase, 1996), while children with right hemisphere lesions are more likely than children with left hemisphere lesions to show decrements in verbal IQ over time and show deteriorations in intellectual ability (Aram & Eisele, 1994). The right hemisphere of children is more activated than that of adults during verbal fluency as indicated by fMRI, implicating significant involvement of the right hemisphere in this verbal task in children (Gaillard et al., 2000). While spatial deficits (relative right hemisphere impairment) may reflect disruption to emotion regulation processes that contribute to antisocial behavior, verbal deficits could additionally interfere with the development of language-based mechanisms of self-control and contribute to poor communication skills that result in difficult parent–child relationships and antisocial behavior (Moffitt, 1993a; Tarter, Hedegus, Winsten, & Alterman, 1984). Furthermore, spatial ability in young children is positively correlated with their ability to describe affective states (Speltz et al., 1999), an ability that is hypothesized to facilitate emotion regulation. Thus, a "double cognitive hit" involving both spatial and verbal processes, of the type seen here in the persistently antisocial group by age 11 years, may help explain why a minority of antisocial children persist in their antisocial behavior.

Cognitive deficits in persistently antisocial individuals could not be accounted for by poor scholastic ability, social adversity, early hyperactivity, poor test motivation, poor test comprehension, or social discomfort during testing. In addition, the possibility that the failure to observe verbal deficits at age 3 years may be due to lower reliability or validity of the verbal measure relative to the spatial measure must be considered, particularly since there were different numbers of indicators of the verbal and spatial constructs at each age. Four lines of evidence are inconsistent with this alternative scenario. First, coefficient alpha for the verbal age 3 years measure (with six subscales) is .76, whereas internal reliability for the spatial age 3 years measure (with only two subscales) is only 0.46. Second, as indicated in Table 4, the verbal age 3 years measure correlated .25 with age 11 verbal IQ, with the spatial age 3 years measure correlating at a similar level ($r = .24$) with the age 11 spatial IQ. Third, the age 3 years verbal ability measure is at least as good as the spatial age 3 years measure in predicting to reading and school achievement levels at age 11 years (see Table 4). Fourth, data from 73 subjects who were given the Reynell Developmental Language Scale (Reynell & Huntley, 1972) at age 6 years show a .36 correlation ($p < .002$)

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

with the age 3 years verbal ability measure compared to a .25 correlation ($p < .005$) with the spatial age 3 years measure ($p < .025$); while the difference between these two correlations in not statistically significant due to the modest sample size, the verbal age 3 years measures predicts twice the amount of variance in age 6 language than the spatial age 3 years measure. These findings for internal reliability and predictive validity demonstrate that the lack of age 3 years verbal deficits in persistently antisocial individuals cannot be easily explained by differential reliability or invalidity of the age 3 years verbal ability measures, particularly since this verbal index had more indicators and higher reliability than the spatial age 3 years measure, which did yield significant group differences.

A number of caveats to these findings need to be outlined. First, the measures of cognitive ability at both ages are not comprehensive. Second, the age 3 years measures of attention–distraction, activity level, motivation, comprehension of directions, and ease of relationship with the tester, which were entered as covariates, were only measured by one research assistant, and lack of reliability could contribute to their failure to abolish group differences in cognitive ability. Third, because antisocial behavior was measured at only two time points, because only teachers measured antisocial behavior at age 8 years, because there were a variety of sources for the age 17 years antisocial ratings, and because the use of a 33% cutoff resulted in a group of persistent antisocial individuals of whom only 64% were male, findings of the present study cannot be directly generalized to studies in Western societies that have used multiple assessment points and more stringent criteria for group formation that result in more male-dominated and potentially more serious forms of persistent antisocial behavior. In particular, not all childhood onset cases as defined in other studies (starting by age 10–12 years) would have been included in the childhood onset cases in the present study (defined as antisocial by age 8 years), while some adolescent onset cases as defined in other studies may have already desisted from engaging in antisocial behavior by age 17 years. Fourth, we caution that no clear conclusions can be drawn from this study about the precise mechanisms that may underlie the early spatial deficits–persistent antisocial behavior relationship. Furthermore, there may be no simple, direct explanations for why spatial deficits are linked to persistent antisocial behavior; transactional processes in which cognitive deficits in the antisocial child elicit negative parenting that further exacerbates antisocial behavior are likely to provide more realistic pictures of the developmental complexities that surround persistent antisocial behavior (Hinshaw & Anderson, 1996).

In conclusion, this study finds that persistently antisocial individuals have fixed spatial deficits at both ages 3 and 11 years, whereas verbal deficits are present at age 11 years, but not age 3 years and are thus developmentally acquired. Findings suggest that sustained spatial deficits may contribute to both sustained antisocial behavior and the development of verbal deficits that contribute further to antisocial processes. An early-starter spatial impairment model is proposed in which early spatial impairments may interfere with mother–infant bonding processes and be a marker for disrupted right hemisphere regulation of affect. Despite several limitations, it is hoped that this study may encourage further consideration of spatial deficits in the development of persistent antisocial behavior and provide one explanatory model that can be empirically tested in future studies.

References

Adolphs, R., Damasio, H., Tranel, D., & Damasio, A. R. (1996). Cortical systems for the recognition of emotion in facial expressions. *Journal of Neuroscience, 16,* 7678–7687.

Aguilar, B., Sroufe, L. A., Egeland, B., & Carlson, E. (2000). Distinguishing the early-onset/persistent and adolescence-onset antisocial behavior types: From birth to 16 years. *Development & Psychopathology, 12,* 109–132.

Allen, M. J., & Yen, W. M. (1979). *Introduction to measurement theory.* Monterey, CA: Brookes/Cole.

Aram, D. M., & Eisele, J. A. (1994). Intellectual stability

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

in children with unilateral brain lesions. *Neuropsychologia, 32,* 85–95.
Barratt, E. S., Stanford, M. S., Kent, T. A., & Felthous, A. (1997). Neuropsychological and cognitive psychophysiological substrates of impulsive aggression. *Biological Psychiatry, 41,* 1045–1061.
Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1997). Deciding advantageously before knowing the advantageous strategy. *Science, 275,* 1293–1294.
Berman, A., & Siegal, A. (1976). A neuropsychological approach to the etiology, prevention, and treatment of juvenile delinquency. In A. Davids (Ed.), *Current perspectives in child personality and psychopathology.* New York: Wiley.
Binder, A. (1988). Juvenile delinquency. *Annual Review of Psychology, 39,* 253–282.
Boehm, A. (1986). *Boehm Test of Basic Concepts—Preschool Version*. San Antonio, TX: Psychological Corporation.
Borod, J. C., St. Clair, J., Koff, E., & Alpert, M. (1990). Perceiver and poser asymmetries in processing facial emotion. *Brain & Cognition, 13,* 167–177.
Casey, B. J., Castellanos, F. X., Giedd, J. N., & Marsh, W. L. (1997). Implication of right frontostriatal circuitry in response inhibition and attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child & Adolescent Psychiatry, 36,* 374–383.
Chiron, C., Jambaque, I., Nabbout, R., Lounes, R., Syrota, A., & Dulac, O. (1997). The right brain is dominant in human infants. *Brain, 120,* 1057–1065.
Davidson, R. J. (1998). Anterior electrophysiological asymmetries, emotion, and depression: Conceptual and methodological conundrums. *Psychophysiology, 35,* 607–614.
Davidson, R. J., Eckman, P., Saron, C. D., Senulis, J. A., & Friesen, W. V. (1990). Approach-3 withdrawal and cerebral asymmetry: Emotional expression and brain physiology I. *Journal of Personality and Social Psychology, 58,* 330–341.
Deitz, K. R., Lavigne, J. V., Atrend, R., & Rosenbaum, D. (1997). Relation between intelligence and psychopathology among preschoolers. *Journal of Clinical Child Psychology, 26,* 99–107.
De Schonen, S., & Deruelle, C. (1991). Visual field asymmetries for pattern processing are present in infancy: A comment on T. Hatta's study on childrens' performances. *Neuropsychologia, 29,* 335–337.
Dodge, K. A. (1991). The structure and function of reactive and proactive aggression. In D. J. Pepler & K. M. Rubin (Eds.), *The development and treatment of childhood aggression* (pp. 201–218). Hillsdale, NJ: Erlbaum.
Eisele, J. A., Lust, B., & Aram, D. M. (1998). Presupposition and implication of truth: Linguistic deficits following early brain lesions. *Brain & Language, 61,* 376–394.
Evans, J. R., & Park, N. S. (1997). Quantitative EEG findings among men convicted of murder. *Journal of Neurotherapy, 2,* 31–39.
Feshbach, S., & Price, J. (1984). Cognitive competencies and aggressive behavior: A developmental study. *Aggressive Behavior, 10,* 185–200.
Flitman, S., O'Grady, J., Cooper, V., & Grafman, J. (1997). PET imaging of maze processing. *Neuropsychologia, 35,* 409–420.
Frost, L. A., Moffitt, T. E., & McGee, R. (1989). Neuropsychological correlates of psychopathology in an unselected cohort of young adolescents. *Journal of Abnormal Psychology, 98,* 307–313.
Furmark, T., Fischer, H., Gustav, W., Larsson, M., & Mats, F. (1997). The amygdala and individual differences in human fear conditioning. *Neuroreport, 8,* 3957–3960.
Gaillard, W. D., Hertz–Pannier, L., Mott, S. H., Barnett, A. S., LeBihan, D., & Theodore, W. H. (2000). Functional anatomy of cognitive development: fMRI of verbal fluency in children and adults. *Neurology, 54,* 180–185.
Gottfried, A. W., Gottfried, A. E., Bathurst, K., & Guerin, D. W. (1994). *Gifted IQ: Early developmental aspects.* New York: Plenum.
Hari, R., Portin, K., Kettenmann, B., Jousmaeki, V., & Kobal, G. (1997). Right-hemisphere preponderance of responses to painful CO/2 stimulation of the human nasal mucosa. *Pain, 72,* 145–151.
Heller, T. L., Baker, B. L., Henker, B., & Hinshaw, S. P. (1996). Externalizing behavior and cognitive functioning from preschool to first grade: Stability and predictors. *Journal of Clinical Child Psychology, 25,* 376–387.
Hinshaw, S. P., & Anderson, C. A. (1996). Conduct and oppositional defiant disorders. In E. J. Mash & R. A. Barkley (Eds.), *Child psychopathology* (pp. 113–149). New York: Guilford.
Hsieh, J. C., Hannerz, J., & Ingvar, M. (1996). Right-lateralized central processing for pain of nitroglycerin-induced cluster headache. *Pain, 67,* 59–68.
Hugdahl, K. (1998). Cortical control of human classical conditioning: Autonomic and positron emission tomography data. *Psychophysiology, 35,* 170–178.
Johnson, M. H. (1999). Cortical plasticity in normal and abnormal cognitive development: Evidence and working hypotheses. *Development and Psychopathology, 11,* 419–437.
Jonides, J., Smith, E. E., Koeppe, R. A., & Awh, E. (1993). Spatial working memory in humans as revealed by PET. *Nature, 363,* 623–625.
Kandel, E., Mednick, S. A., Kirkegaard–Sorensen, L., Hutchings, B., Knop, J., Rosenberg, R., & Schulsinger, F. (1988). IQ as a protective factor for subjects at high risk for antisocial behavior. *Journal of Consulting and Clinical Psychology, 56,* 224–226.
Kosson, D. S. (1996). Psychopathy and dual-task performance under focusing conditions. *Journal of Abnormal Psychology, 105,* 391–400.
Lipsitt, P. D., Buka, S., & Lipsitt, L. P. (1990). Early intelligence scores and subsequent delinquency: A prospective study. *American Journal of Family Therapy, 18,* 197–208.
Loney, B. R., Frick, P. J., Mesha, E., & McCoy, M. G. (1998). Intelligence, callous–unemotional traits, and antisocial behavior. *Journal of Psychopathology and Behavioral Assessment, 20,* 231–247.
Lynam, D., Moffitt, T., & Stouthamer–Loeber, M. (1993). Explaining the relation between IQ and delinquency: Class, race, test motivation, school failure, or self-control? *Journal of Abnormal Psychology, 102,* 187–196.
Moffitt, T. E. (1990). Juvenile delinquency and attention-deficit disorder: Developmental trajectories from age three to fifteen. *Child Development, 61,* 893–910.
Moffitt, T. E. (1993a). "Life-course-persistent" and "adolescent-limited" antisocial behavior: A developmental taxonomy. *Psychological Review, 100,* 674–701.

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

Moffitt, T. E. (1993b). The neuropsychology of conduct disorder. *Development and psychopathology, 5,* 135–151.

Moffitt, T. E., Caspi, A., Dickson, N., Silva, P., & Stanton, W. (1996). Childhood-onset versus adolescent-onset antisocial conduct problems in males: Natural history from ages 3 to 18 years. *Development and Psychopathology, 8,* 399–424.

Moffitt, T. E., & Silva, P. A. (1988). IQ and delinquency: A direct test of the differential detection hypothesis. *Journal of Abnormal Psychology, 97,* 227–240.

Morgan, A. B., & Lilienfeld, S. O. (2000). A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. *Clinical Psychology Review, 20,* 113–136.

Newman, J. P., & Schmitt, W. A. (1998). Passive avoidance in psychopathic offenders: A replication and extension. *Journal of Abnormal Psychology, 107*, 527–532.

Owen, A. M., Milner, B., Petrides, M., & Evans, A. C. (1996). A specific role for the right parahippocampal gyrus in the retrieval of object-location: A positron emission tomography study. *Journal of Cognitive Neuroscience, 8,* 588–602.

Owens, E. B., Shaw, D. S., & Giovannelli, J. (2001). *IQ and conduct problems among low-income preschool-aged boys.* Manuscript submitted for publication.

Pumfrey, P. D. (1985). *Reading tests and assessment techniques* (2nd ed). London: Hodder and Stroughton.

Quay, H. C. (1987). Intelligence. In H. C. Quay (Ed.), *Handbook of juvenile delinquency* (pp.106–117). New York: Wiley.

Quay, H. C., & Peterson, D. R. (1987). *Manual for the Revised Behavior Problem Checklist.* Coral Gables, FL: University of Miami, Department of Psychology.

Raine, A. (1993). *The psychopathology of crime: Criminal behavior as a clinical disorder.* San Diego, CA: Academic Press.

Raine, A., Reynolds, C., Venables, P. H., & Mednick, S. A. (1997). Biosocial bases of aggressive behavior in childhood: Resting heart rate, skin conductance orienting, and physique. In A. Raine, P. A. Brennan, D. P. Farrington, & S. A. Mednick (Eds.), *Biosocial bases of violence* (pp. 107–126). New York: Plenum.

Raine, A., Reynolds, C., Venables, P. H., Mednick, S. A., & Farrington, D. P. (1998). Fearlessness, stimulation-seeking, and large body size at age 3 years as early predispositions to childhood aggression at age 11 years. *Archives of General Psychiatry, 55,* 745–751.

Raine, A., Venables, P. H., & Mednick, S. A. (1997). Low resting heart rate at age 3 years predisposes to aggression at age 11 years: Findings from the Mauritius Joint Child Health Project. *Journal of the American Academy of Child & Adolescent Psychiatry, 36,* 1457–1464.

Reilly, J. S., Stiles, J., Larsen, J., & Trauner, D. (1995). Affective facial expression in infants with focal brain damage. *Neuropsychologia, 33,* 83–99.

Reynell, J., & Huntley, R. M. (1972). New scales for the assessment of language development in young children. *Journal of Learning Disabilities, 4,* 549–557.

Richman, N., Stevenson, J., & Graham, P. J. (1982). *Preschool to school—A behavioral study*. London: Academic.

Rutter, M. (1967). A children's behaviour questionnaire for completion by teachers: Preliminary findings. *Journal of Child Psychology and Psychiatry, 8,* 1–11.

Rutter, M. (1978). Family, are, and school influences in the genesis of conduct disorders. In L. A. Hersov, M. Berger, & D. Shaffer (Eds.), *Aggression and antisocial behavior in childhood and adolescence* (pp. 95–113). New York: Wiley.

Rutter, M., Giller, H., & Hagell, A. (1998). *Antisocial behavior by young people*. Cambridge: Cambridge University Press.

Saugstad, L. F. (1998). Cerebral lateralisation and rate of maturation. *International Journal of Psychophysiology, 28,* 37–62.

Scerbo, A., Raine, A., Venables, P. H., & Mednick, S. A. (1995). The stability of inhibited/uninhibited temperament from ages 3 to 11 years in Mauritian children. *Journal of Abnormal Child Psychology, 23,* 607–618.

Schmitt, J. J., Hartje, W., & Willmes, K. (1997). Hemispheric asymmetry in the recognition of emotional attitude conveyed by facial expression, prosody and propositional speech. *Cortex, 33,* 65–81.

Seguin, J. R., Pihl, R. O., Boulerice, B., & Tremblay, R. E. (1996). Pain sensitivity and stability of physical aggression in boys. *Journal of Child Psychology & Psychiatry & Allied Disciplines, 37,* 823–834.

Smith, E. E., Jonides, J., & Koeppe, R. A. (1996). Dissociating verbal and spatial working memory using PET. *Cerebral Cortex, 6,* 11–20.

Speltz, M. L., DeKlyen, M., Calderon, R., Greenberg, M. T., & Fisher, P. A. (1999). Neuropsychological characteristics and test behaviors of boys with early onset conduct problems. *Journal of Abnormal Psychology, 108,* 315–325.

Stattin, H., & Klackenberg–Larsson, I. (1993). Early language and intelligence development and their relation to future criminal behavior. *Journal of Abnormal Psychology, 102,* 369–378.

Stiles, J., Stern, C., Trauner, D., & Nass, R. (1996). Developmental change in spatial grouping activity among children with early focal brain injury: Evidence from a modeling task. *Brain and Cognition, 31,* 46–62.

Stiles, J., Trauner, D., Engel, M., & Nass, R. (1997). The development of drawing in children with congenital focal brain injury: Evidence for limited functional recovery. *Neuropsychologia, 35,* 299–312.

Tarter, R. E., Hedegus, A. M., Winsten, N. E., & Alterman, A. L. (1984). Neuropsychological, personality, and familial characteristics of physically abused delinquents. *Journal of the American Academy of Child Psychiatry, 23,* 668–674.

Trauner, D. A., Ballantyne, A., Friedland, S., & Chase, C. (1996). Disorders of affective and linguistic prosody in children after early unilateral brain damage. *Annals of Neurology, 39,* 361–367.

van Horn, J. D., Gold, J. M., Esposito, G., Ostrem, J. L., Mattay, V., Weinberger, D. R., & Berman, K. F. (1998). Changing patterns of brain activation during maze learning. *Brain Research, 793,* 29–38.

van Ijzendoorn, M. H., Dijkstra, J., & Bus, A. G. (1995). Attachment, intelligence, and language: A meta-analysis. *Social Development, 4,* 115–128.

Venables, P. H., Fletcher, R. P., Dalais, J. C., Mitchell, D. A., Schulsinger, F., & Mednick, S. A. (1983). Factor structure of the Rutter Children's Behavior Questionnaire in a primary school population in a developing country. *Journal of Child Psychology and Psychiatry, 24,* 213–222.

Virkkunen, M., & Luukkonen, P. (1977). WAIS perfor-

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press

mances in antisocial personality (disorder). *Acta Psychiatrica Scandinavica, 55*, 220–224.

Wechsler, D. (1967). *Wechsler Preschool and Primary Scale of Intelligence*. San Antonio, TX: Psychological Corporation.

Wilson, J. Q., & Herrnstein, R. J. (1985) *Crime and human nature; The definitive study of the causes of crime*. New York: Touchstone.

Yeudall, L. T., Fromm–Auch, D., & Davies, P. (1982). Neuropsychological impairment of persistent delinquency. *Journal of Nervous and Mental Disease, 170,* 257–265.

https://doi.org/10.1017/S0954579402001025 Published online by Cambridge University Press