

Conduct Disorder and Cognitive Functioning: Testing Three Causal Hypotheses

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SCHONFELD, IRVIN SAM, SHAFFER, DAVID, O'CONNOR, PATRICIA, and PORTNOY, STEPHANIE *Conduct Disorder and Cognitive Functioning: Testing Three Causal Hypotheses* CHILD DEVELOPMENT, 1988, 59, 993-1007 The sample consisted of black adolescents who were members of the Columbia-Presbyterian chapter of the Collaborative Perinatal Project from birth to age 7. At age 17, subjects and their parents were administered a battery of instruments that included standardized psychiatric diagnostic interviews as part of a call-back study. Results from least-squares and logistic regression analyses were compatible with the hypothesis that deficiencies in cognitive functioning are causally related to adolescent conduct disorder as defined by DSM III. The results suggested that the relation of cognitive functioning to psychiatric status appears to be specific to conduct disorders. The results were incompatible with a "third" variable hypothesis (third factors included neurological status and environmental disadvantage) and the hypothesis that conduct problems lead to deficits in cognitive functioning. The 3 most (and equally) important factors in accounting for age-17 conduct disorder were cognitive functioning, parent psychopathology, and early aggression. A closer look at the data tentatively suggested that a broad deficiency in acculturational learning, rather than narrowly focused social cognitive differences or native endowment, constitutes a key element in the link between cognitive functioning and conduct disorder. Test bias was ruled out as a possible explanation for the results.

A widely observed finding in epidemiologic surveys and in studies of referred samples has been the association between conduct disturbance and deficits in cognitive functioning. On the Isle of Wight, Rutter, Tizard, and Whitmore (1970) found that, among 10- and 11-year-olds, children with conduct disorders tended to have lower IQ scores than neurotic and nondeviant control youngsters. These findings were replicated in an epidemiologic survey of an inner London

borough (Berger, Yule, & Rutter, 1975). In an epidemiologic survey of black youngsters residing in households in the Woodlawn section of Chicago, Kellam, Branch, Agrawal, and Ensminger (1975) found that aggression measured in the first grade was negatively associated with concurrent and third-grade IQ. In a total population survey of an upstate New York county, Huesmann, Eron, Lefkowitz, and Walder (1984) showed that a well-conceived measure of aggression at age 8 was

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significantly related to a contemporaneous measure of IQ Robins (1966), in a long-term follow-up of a clinic-referred sample of white St. Louis youngsters, found that educational level was inversely related to the rate at which individuals were diagnosed for sociopathic personality 30 years later Farrington (Farrington, 1978, West & Farrington, 1973) found that IQ was inversely related to official and self-reported delinquency among English working-class boys Lewis, Shanok, and Pincus (1981) found that, among incarcerated delinquents, those classified as very violent tended to perform less well on a psychoeducational battery that included IQ and reading and mathematics achievement tests than less violent peers

The relation between IQ and delinquency may be spurious since IQ test performance is related to race and social class, and race and social class are related to delinquency In response to such an argument, Hirschi and Hindelang (1977) adduced evidence from an array of research reports showing that IQ is related to both officially reported and self-reported delinquency *within* race and/or class That the relation holds for delinquency rates based on self-reports weakens the argument for spuriousness owing to biases in detection (Hirschi & Hindelang, 1977, West & Farrington, 1973)

At least three hypotheses explain the association between antisocial behavior or conduct disorder, on one hand, and deficits in cognitive functioning on the other One hypothesis is that conduct problems lead to inadequate school learning and attendant deficits in cognitive functioning Given the well-documented influence of education on cognitive development (Luria, 1976, Sharp, Cole, & Lave, 1979), the obstructing of classroom procedures by students exhibiting antisocial behaviors may have a retarding effect on cognitive development in those same students A second hypothesis is that cognitive deficits lead to antisocial behavior, either directly or through some mediating condition Rutter and Giller (1983) argued that one variable linking low IQ to conduct disorder or delinquency may be the individual's response to educational failure Low IQ might also be associated with inadequate social skills or coping behaviors, which in turn lead to frustrating experiences in interacting with others and, consequently, to aggression A third hypothesis is that cognitive deficits and antisocial behavior are spuriously associated because some explanatory antecedent factor

(or factors) is causally related to both (see Susser, 1973)

The first hypothesis (conduct difficulties → cognitive deficits) is inconsistent with a number of findings Since the average correlation between IQ test scores obtained at ages 5 and 17 is about .7 (e.g., Bloom, 1964), it is likely that depressed IQ test performance observed in adolescence is linked to depressed cognitive functioning earlier in the life span, temporally preceding delinquency if not more infantile expressions of antisocial behavior This is not to say that a somewhat stable entity like IQ is impervious to other influences, the results of three epidemiologic surveys, however, are inconsistent with the first hypothesis Kellam et al (1975) found an association between low IQ and aggressive behavior in first grade, early enough in the school careers of children to cast doubt on the conduct-problems-as-cause hypothesis Richman, Stevenson, and Graham (1982) found that low IQ was related to conduct problems as early as ages 3 and 4, well before entry into school In addition, Richman et al (1982) were unable to find changes in intellectual functioning to be associated with changes in conduct problems in their 5-year follow-up of 3- and 4-year-olds Rutter, Graham, Chadwick, and Yule (1976) and Rutter, Tizard, Yule, Graham, and Whitmore (1976) also found that changes in psychiatric status were not associated with changes in IQ in their 4-year follow-up of the 10- and 11-year-olds seen on the Isle of Wight The results of these studies indicate that remission in problem status was not associated with change in intellectual status, although it might take longer than 4 or 5 years for changes in intellectual status to appear

The second hypothesis (cognitive deficits → conduct difficulties) has modest and indirect support in the results of Rutter et al (1970) Their findings apply to the deficiencies in the cognitive skills pertaining to reading, but not IQ Families of conduct-disorder children with reading retardation had significantly lower rates of parental discord than families of conduct-disorder children without reading retardation, consistent with the view that there are at least two routes to conduct-disorder outcome response to educational failure and response to parental discord Response to educational failure may have led to conduct disorder in the reading-retarded subgroup since those children did not differ from controls in rate of parental discord By contrast, the families of antisocial children who

were not reading retarded tended to show significantly higher rates of parental discord than control youngsters

Other indirect support for the second hypothesis comes from Ayllon and Roberts (1974), who found that reduction in classroom antisocial behavior resulted from behavior modification efforts aimed specifically at improving academic performance. The contingencies employed were indifferent to the antisocial activities in which the children were engaged. Limitations of this study were that subjects came from predominantly upper-middle-class homes, and academic performance was, at a minimum, on grade level.

If conduct problems lead to cognitive deficits or, alternatively, if cognitive deficits lead to conduct problems, the outcomes would take some time to develop. The school can be expected to constitute the chief context in which these outcomes emerge because it is the site in which (a) social interaction with peers is accelerated and (b) cognitive-academic functioning takes a prominent place in the child's life. The third hypothesis (third variable → cognitive deficits, third variable → conduct difficulties) has noteworthy support in the Ruchman et al (1982) results, which show that IQ and conduct problems are associated as early as age 3, and in the Kellam et al (1975) results, which show that antisocial behavior is associated with low IQ as early as grade 1. Conduct problems and cognitive deficits are linked too early in life for one to cause the other over the long course schooling. In addition, Huesmann et al (1984) and Olweus (1979) found that aggressive behavior in males has much the same stability as IQ. It is therefore possible that, among males, the origins of conduct problems and deficiencies in cognitive functioning arise from common factors. Candidate factors include impaired socializing skills in parents owing, perhaps, to parent psychopathology and early temperamental differences in the child (Rutter et al, 1970). In the Huesmann et al (1984), Kellam et al (1975), and Ruchman et al (1982) samples, males exhibited more disturbance than females. Such findings are compatible with both socialization and temperament explanations. Counterevidence adduced by Rutter et al (1970) suggests that temperamental factors may not explain the association between poor cognitive functioning (as manifest in reading retardation) and conduct disorder because antisocial behavior appears to be associated with reading retardation that is either social or biological in origin.

In the present investigation, we studied the relation between cognitive functioning, as manifest in IQ and achievement test performance, and DSM III (*Diagnostic and Statistical Manual of Mental Disorders, Third Edition*) categories of conduct disturbance. This study constitutes an extension of research conducted prior to the development of the DSM III nomenclature, and our purpose was to test the validity of the rival hypotheses that potentially explain the association between cognitive functioning and conduct disturbance.

The sample studied consisted of black males who participated in a follow-up study of the psychiatric and medical sequelae, in adolescence, of neurological soft signs diagnosed at age 7 (Shaffer et al, 1985). Black male subjects were followed for practical reasons at the medical center at which the study was conducted, they had the highest prevalence rate of soft signs at the age of 7. This selection decision, however, limits the generalizability of the findings to other racial and ethnic groups, as well as to females. Nevertheless, the nature of the sample studied and the methodological strengths of the investigation make it possible to compare the three hypotheses mentioned. First, the sample was unreferred and unselected for conduct disorders, and the subjects were representative of youngsters from black northern Manhattan homes. Second, data capturing the environmental disadvantages of the subjects were collected. Third, the neurologic status of all subjects was assessed. Finally, IQ was measured at both ages 7 and 17 with the appropriate Wechsler scales. The linkages between psychopathology and performance on subtests reflecting relatively high and low levels of acculturational learning (e.g., Information and Block Design, respectively) could thus be assessed (see Cattell, 1963).

Method

Sample—Subjects were members of the Columbia-Presbyterian Medical Center chapter of the nationwide Collaborative Perinatal Project (CPP). At this site, between 1957 and 1963, approximately one in five mothers consecutively presenting for prenatal care were accepted into the study. Only planned adoption donors and mothers who failed to present for prenatal care were excluded. The research team followed the children until their eighth year. Call-back subjects followed by a different team at age 17 constituted two groups. One included nonretarded black, English-

speaking males who met the following criteria (a) membership in the 1962–1963 birth cohort, (b) a positive diagnosis for any one of a number of neurological soft signs during the age-7 medical examination, and (c) no evidence of other neurological problems. The second group consisted of nonretarded males matched to members of the first group on date of birth, race, and language. Members of the second group, however, were required to exhibit no evidence of soft or hard neurological signs.

At the time of the follow-up, 63 males who received index ratings for soft signs at age 7 were matched with 63 sign-free individuals. A total of 61 sign-positive males were assessed at age 17. Three of the 61 were later excluded because either a major neurological disorder or a tic, missed in an earlier review of the medical records, was documented in a later record check. Thus the sign-positive sample was reduced to 58. Of the 63 in the second sample, 57 were reexamined at age 17.

Procedures—At the time of the age-17 follow-up, psychiatric, neurological, and cognitive examinations were conducted on the same day for almost all of the subjects by independent examiners with no knowledge of the subjects' status on the archival measures. A description of the psychiatric assessment procedures can be found in Shaffer et al. (1985). The neurological examination is described in Stokman et al. (1986). Connors's (1969) Teachers Questionnaire, an inventory to assess the extent of deviant classroom behaviors (e.g., stealing, inattention), was completed by each of the subject's most recent teachers close to the time of the psychiatric examination.

Cognitive examination—The cognitive battery consisted of the Wechsler Adult Intelligence Scale (WAIS) and the Peabody Individual Achievement Tests (PIAT) for reading comprehension, mathematics, and spelling. Based on the work of Bannatyne (1971), scales were constructed from WAIS subtests to reflect success in acquiring knowledge (Information + Vocabulary + Arithmetic) and spatial ability (Block Design + Object Assembly + Picture Completion). The scales were constructed in order to locate the core of the IQ-related differences occurring with conduct disorder. While these scales have been used extensively with the WISC and the WISC-R (Kaufman, 1982), Bannatyne (personal communication, 1984) indicated that they apply equally to the WAIS. According to Horn

(1979), the acquired knowledge and spatial ability scales reflect, respectively, acculturational (crystallized) and native (fluid) aspects of intelligence. In order to assess the applicability of the scales in the present sample, a number of reliability coefficients were computed. The internal consistency reliabilities for the acquired knowledge ($\alpha = .82$) and spatial abilities ($\alpha = .76$) scales were satisfactory. Although the scales were significantly correlated with each other ($r = .54, p < .001$), the coefficient of the reliability of the differences (Cohen & Cohen, 1983) is consistent with the view that the scales measured different constructs ($r_{[A-B][A-B]} = .54$). A minimally acceptable value for such a coefficient has been thought to be .50 (Dohrenwend, Shrout, Egri, & Mendelsohn, 1980).

Psychiatric diagnoses—As part of the psychiatric evaluation of the adolescent, the subject and his parent (usually the mother) separately received semistructured interviews that were constructed from existing instruments (Shaffer et al. 1985). The interview of the adolescent included portions of the Schedule for Affective Disorders and Schizophrenia (Spitzer & Endicott, 1977) to assess affective and anxiety symptoms, elements of an interview written by Rutter and Graham (1968) to assess antisocial behaviors, and a number of new items designed to elicit symptoms needed to assign DSM III diagnoses. The parent interview was adapted from an instrument developed by Rutter and Brown (1966) to gather sociodemographic data on family members and psychiatric data on the adolescent. The General Well-Being Scale (GWB, Dupuy, 1974) was also incorporated into this interview. The GWB assesses psychiatric functioning in the caretaker, including visits to mental health clinicians and depressive symptoms.

At the conclusion of each parent and adolescent interview, the interviewer assigned the adolescent a Global Assessment Scale (GAS) rating (Endicott, Spitzer, Fleiss, & Cohen, 1976). The GAS is a continuous measure, ranging from 0 to 100, of psychiatric functioning in the adolescent. Higher scores reflect satisfactory social functioning and low levels of symptoms, and lower scores, impaired functioning and high levels of symptoms. If a subject received a rating of 70 or lower on either interview, or if a subject received a rating of 75 or lower when only one interview was conducted, a case conference on the subject's psychiatric status was held. In a separate study of a subsample of nine subjects involving four to seven raters blind to

each other's ratings, we obtained 79% agreement in assigning subjects GAS scores above and below the caseness marker of 70 (Shaffer et al., 1986)

At the case conference, two psychiatrists and one psychologist reviewed all relevant parent and adolescent interview data and teacher reports but no other contemporaneous records, case conferees did not know the subjects' status on the archival measures New GAS scores and as many Axis I DSM-III diagnoses (the clinical syndromes) as applicable were assigned by the conferees. No exclusionary rules were invoked, therefore, subjects could receive more than one diagnosis. Any adolescent who received a mean GAS rating of 70 or less from the conferees was assigned at least one diagnosis. Subjects whose high interview-based GAS values excluded them from the conference received no diagnosis. These individuals were assigned a final GAS rating that was the mean of the ratings assigned by the two interviewers.

Archival data—An earlier generation of researchers had collected data on all subjects in childhood (see Nichols & Chen, 1981, for a complete description of the early data). Retrieved psychological data included the Stanford-Binet and WISC IQ scores obtained during the age-4 and age-7 psychological examinations, respectively. Only seven subtests of the WISC (Verbal Information, Comprehension, Vocabulary, Digit Span, Performance Picture Arrangement, Block Design, Coding) were administered at age 7, age-7 IQ was prorated (see Nichols & Chen, 1981). Psychologists rated a number of subject behaviors, using forced-choice rating scales, during the age-7 psychological examination. The age-17 team, who did not know the subjects' status on other variables, selected three age-7 behavior ratings in constructing an a priori scale to reflect age-7 aggressive behavior ($\alpha = .78$, see Shaffer, O'Connor, Shafer, & Prupis, 1983). The three behaviors in the scale were negativism (0—responds to directions, 1—restive in response to directions, 2—extremely negative), dominating aggressiveness (0—unassertive to normally assertive, 1—forceful, 2—dominating aggressive behavior), and hostility (0—appropriate to negative affect, 1—uncooperative or angry, 2—overt physical or verbal attacks).

Health information retrieved from the early records included data from the age-7 neurological examination (Nichols & Chen, 1981). Six groups of neurological soft signs were diagnosed: awkwardness or poor coordination in finger-nose touching, finger pursuit,

and fine motor activity (the most frequently diagnosed neurological signs), dysdiadochokinesis (difficulties in performing rapid alternating movements of the hands or feet), mirror movements (inability to inhibit movements in the hand opposite the hand performing a simple task), tremor, dysgraphesthesia (incorrect identification of a predisplayed symbol traced on the palm when blindfolded), and astereognosis (incorrect identification of three-dimensional objects on the outstretched hand when blindfolded). Test-retest agreement on a CPP subsample was found to be 85% for the signs involving poor coordination (Nichols & Chen, 1981). Shaffer et al. (1985) provided additional, indirect evidence that the age-7 signs were reliably measured. Other types of archival data retrieved include (a) two single-item responses, one obtained at about the time of the subject's birth and another at the time of the age-7 examination, concerning the presence of "mental illness" in the family, and (b) an ordinal measure of age-7 family income.

Results

Classification of disorders—Table 1 enumerates all diagnoses given the age-17 call-back sample. For the purpose of data reduction, two psychiatrists and one psychologist sorted the diagnoses into five superordinate categories: affective disorders, conduct disorders, anxiety-withdrawal disorders, substance abuse, and psychotic disorder. The groupings are consistent with child psychiatry nosology (Rutter, Shaffer, & Sturge, 1975). Agreement on assignment to the categories exceeded 90%. A total of 64 adolescents were identified as having no disorder, and 30 were found to have a conduct disorder, either singly (18) or in combination with some other disorder (12).

Scale construction—Data from the parent interview were used to construct two a priori scales reflecting types of adversity that can affect the adolescent's psychiatric status: parent psychopathology and environmental disadvantage. The parent psychopathology scale comprised five variables: mother's dysphoric mood, mother or father having visited a psychiatrist/psychologist, criminality in either parent, and parental discord (based on interview items on parental quarreling). The environmental disadvantage scale comprised seven variables: low family income, mother not being married to the biological father, lower levels of education in the mother or father, four or more siblings, welfare dependency, and dissatisfaction with housing. Thus

TABLE 1
DISTRIBUTION AND GROUPING OF AGE 17 DSM III DIAGNOSES

	Frequency
Conduct	
312 00, Conduct disorder, undersocialized, aggressive	2
312 21, Conduct disorder, socialized, nonaggressive	14
312 23, Conduct disorder, socialized, aggressive	6
313 81, Oppositional disorder	5
314 01, Attention-deficit disorder, with hyperactivity	3
314 80, Attention-deficit disorder, residual	1
Affective	
295 70, Schizoaffective disorder	1
296 20, Major depression, single episode, unspecified	2
296 22, Major depression, single episode, without melancholia	2
296 32, Major depression, recurrent, with melancholia	1
296 36, Major depression, recurrent, in remission	2
296 56, Bipolar disorder depressed, in remission	1
296 82, Atypical depression	3
300 40, Dysthymic disorder	5
309 00, Adjustment disorder with depressed mood	3
Anxiety-withdrawal	
300 00, Atypical anxiety disorder	1
301 29, Schizotypal personality disorder	1
309 21, Separation anxiety disorder	1
313 00, Overanxious disorder	9
313 21, Avoidant disorder of adolescence	3
Substance abuse	
305 01, Alcohol abuse, continuous use	1
305 21, Cannabis abuse, continuous use	5
305 91, Other, mixed substance abuse, continuous use	1
Psychotic disorder	
295 32, Schizophrenia, paranoid type, chronic	1
Total	74

NOTE — This table presents a complete list of all diagnoses given. A subject may receive more than one diagnosis.

the environmental disadvantage scale encompasses the traditional socioeconomic indicators as well as other types of adversity.

Items for each scale were range standardized before being aggregated. In range standardizing the items, a score of zero was given to the optimal response category (e.g., college degree in the maternal education item) and a score of one was given to the most adverse response (e.g., elementary school education). Intermediate response alternatives were assigned proportionally intermediate values between zero and one. Thus in both scales higher scores reflected increasing levels of adversity. The alpha coefficients for the parent psychopathology and environmental disadvantage scales were .56 and .62, respectively. A substudy of the scales' convergent and discriminant validity suggested the scales were adequate. (a) *t* tests revealed that the archival record of the presence versus absence of family mental illness was significantly related to the age-17 parent psychopathology scale, $t(100) = 3.16$, $p < .01$, and

unrelated to the age-17 environmental disadvantage scale, $t(100) = 1.60$, (b) age-7 family income was significantly correlated to age-17 environmental advantage ($r_{sp} = -.47$, $p < .001$) but unrelated to age-17 parent psychopathology ($r_{sp} = .05$, $N.S.$)

The stability of IQ test performance — Cognitive functioning as measured by the age-17 WAIS IQ was found to be moderately correlated with age-7 WISC ($r = .66$, $p < .001$) and age-4 Stanford-Binet IQ ($r = .50$, $p < .001$). Age-7 and age-4 IQ were also moderately correlated ($r = .61$, $p < .001$).

Conduct disorder and cognitive functioning — Table 2 presents the mean scores of conduct-disorder and disorder-free adolescents, with and without soft signs, on the cognitive tests administered at age 17. For each measure of cognitive functioning a two-way (conduct disorder present/absent \times soft signs present/absent) analysis of variance was performed. The analyses revealed conduct-disorder main effects for full-scale IQ, $F(1,87)$

TABLE 2

AGE 17 TEST PERFORMANCE OF CONDUCT-DISORDER SUBJECTS WITH AND WITHOUT EARLY SOFT SIGNS

GROUP	WAIS			PEABODY			ACQUIRED KNOWLEDGE SCALE	SPATIAL SCALE
	FIQ	VIQ	PIQ	Reading Comp	Math	Spell		
Signs absent								
No disorder								
M	96 0	96 0	96 4	91 6	95 3	89 6	23 1	27 6
SD	9 8	9 6	12 1	13 8	10 8	13 4	5 1	7 0
Conduct disorder								
M	90 5	89 7	92 8	87 1	87 0	89 8	19 0	25 9
SD	8 5	8 0	12 3	12 1	11 5	12 8	4 4	6 9
Signs present								
No disorder								
M	90 5	91 5	90 5	88 7	90 3	86 0	22 2	24 3
SD	11 7	12 2	12 1	12 1	13 3	14 2	7 3	7 2
Conduct disorder								
M	83 8	85 2	84 0	82 3	82 3	78 9	18 0	22 1
SD	11 0	11 5	11 6	13 1	11 7	14 7	5 3	6 1

= 6.69, $p < .05$, verbal IQ, $F(1,87) = 6.96$, $p < .01$, PIAT Math, $F(1,87) = 9.31$, $p < .001$, and the acquired knowledge scale, $F(1,87) = 10.12$, $p < .01$. Marginal ($p < .10$) effects were found for performance IQ, $F(1,87) = 3.56$, and PIAT Reading, $F(1,87) = 3.52$. The analyses revealed soft-signs main effects for full-scale IQ, $F(1,87) = 6.74$, $p < .05$, performance IQ, $F(1,87) = 7.60$, $p < .01$, PIAT Spelling, $F(1,87) = 5.45$, $p < .05$, and the spatial scale, $F(1,87) = 5.50$, $p < .05$. Marginal effects were found for verbal IQ, $F(1,87) = 3.57$, and PIAT Math, $F(1,87) = 3.29$. No significant interactions were obtained.

Table 3 presents the mean scores of the conduct-disorder and disorder-free adolescents on cognitive tests administered at ages 4 and 7. For each measure of cognitive functioning, a two-way (conduct disorder present/absent \times soft signs present/absent) analysis of variance was performed. Because too few age-7 subtests were available to construct acquired knowledge and spatial scales, performance on Information and Block Design (subtests related to those constructs) was examined. The analyses revealed conduct-disorder main effects for full-scale WISC IQ, $F(1,89) = 4.62$, $p < .05$, verbal IQ, $F(1,90) =$

TABLE 3

AGE 4 AND 7 TEST PERFORMANCE OF CONDUCT-DISORDER SUBJECTS WITH AND WITHOUT EARLY SOFT SIGNS

GROUP	AGE 4 STANFORD- BINET	AGE 7 WISC			WISC SUBTESTS	
		FIQ	VIQ	PIQ	Infor- mation	Block Design
Signs absent						
No disorder						
M	99.0	98.4	96.0	101.3	9.8	10.7
SD	13.2	8.9	11.4	8.6	2.9	2.0
Conduct disorder						
M	94.8	94.2	92.2	97.2	8.8	10.8
SD	10.7	10.3	8.7	13.2	1.4	2.8
Signs present						
No disorder						
M	94.4	92.3	90.6	95.7	8.7	8.9
SD	14.6	12.7	12.8	14.5	3.1	2.8
Conduct disorder						
M	87.8	84.6	80.9	91.6	6.8	8.6
SD	17.8	17.5	14.5	21.4	3.5	3.5

TABLE 4
CORRELATIONS BETWEEN VARIABLES EMPLOYED IN THE PATH ANALYSES

	GAS	Signs	PP	EA	Disadv	WAIS	WISC
GAS							
Signs	-.20*						
PP	-.25*	-.06					
EA	-.29**	-.04	-.09				
Disadv	-.26*	.01	.44***	-.21*			
Full-scale WAIS IQ	.33***	-.39***	.07	-.12	-.28**		
Full-scale WISC IQ	.30**	-.39***	.12	-.16	-.14	.68***	
Conduct/no disorder	-.93***	.17	.23*	.25*	.18	-.29**	-.26*

NOTE.—Bivariate correlations were computed with n s that ranged from 88 to 94. Signs = no of early signs, PP = parent psychopathology, EA = presence/absence of early aggression, Disadv = environmental disadvantage.

* $p < .05$

** $p < .01$

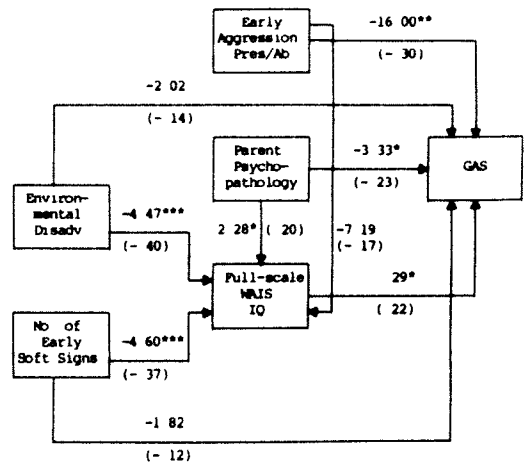
*** $p < .001$

6.29, $p < .05$, and Information, $F(1,90) = 5.25$, $p < .05$. A marginal effect was found for the age-4 Stanford-Binet, $F(1,83) = 2.70$, $p = .10$. The analyses revealed soft-signs main effects for full-scale WISC IQ, $F(1,89) = 8.10$, $p < .01$, verbal IQ, $F(1,90) = 9.60$, $p < .01$, and Block Design, $F(1,90) = 11.36$, $p < .001$. Marginal effects ($p < .10$) were found for the Stanford-Binet, $F(1,83) = 3.09$, and performance IQ, $F(1,89) = 3.06$. No significant interactions were obtained.

Path analyses—In order to investigate further the relation between conduct disorder and its possible antecedents, path-analytic procedures were introduced. The correlation matrix used in constructing the path models is presented in Table 4. A number of assumptions about the causal ordering of the variables needed to be made before conducting the path analyses. It was assumed that the constructs underlying full-scale WAIS IQ, environmental disadvantage, parent psychopathology, age-7 aggression, and soft signs took causal precedence over age-17 psychiatric status. This assumption was partly supported by the continuity of WAIS IQ, environmental disadvantage, and parental mental health with related archival measures, and the finding that excessive aggressive behavior was rare at age 7 (only seven subjects displayed measurable aggressive behavior during testing). Since soft signs were measured at age 7 and are in some sense reflective of central nervous system functioning, they were thought to be causally prior to the onset of the conduct disorder. The models made no assumptions about the causal order of soft signs, environmental disadvantage, parent psychopathology, and early aggression. Since one of the goals of this investigation was to assess the common-antecedents hypothesis, these

four variables were considered to be causally prior to both full-scale WAIS IQ and GAS.

In conducting the path analysis, the sample was limited only to those subjects with a conduct-disorder diagnosis ($n = 30$) or no diagnosis ($n = 64$). In the full path model, depicted in Figure 1, GAS was regressed (least squares) on five factors, including number of early soft signs, full-scale WAIS IQ, early aggression (absent/present), the environmental disadvantage scale (converted to standard scores), and the parent psychopathology scale (converted to standard scores), and IQ was regressed on the other four factors. GAS constituted a global index of severity of the conduct disorder.



* $p < .05$

** $p < .01$

*** $p < .001$

FIG 1—Full path model showing the effect of each exogenous variable on GAS and full-scale WAIS IQ.

In order to maximize power, means were substituted for missing values for the few subjects for whom scorable values were absent (no more than four subjects lacked values on any one predictor). Tests for systematic differences revealed no bias in the occurrence of missing values (Cohen & Cohen, 1983). It was thought that, if third variables explain the zero-order relation between cognitive functioning and conduct disturbance, the path from IQ to GAS would be nonsignificant when IQ and the other four potential causal factors were entered in the same regression equation. As can be seen in Figure 1, the path from IQ to GAS was significant, controlling for the other factors.

Beginning with the full model, causal links of negligible size were eliminated. A variable was retained when two criteria were met within a regression framework: (a) its path coefficient attained the 10 significance level when entered in the model last, and (b) the variable accounted for at least 2% of the variance in GAS or WAIS IQ when entered in the model last. Three predictors of GAS—early aggression, WAIS IQ, and parent psychopathology—met these criteria.

The trimmed path model is depicted in Figure 2. In this model, causal paths from environmental disadvantage and soft signs to GAS were eliminated. To facilitate understanding of the model, both unstandardized and standardized (in parentheses) path coefficients were included in the diagram. The un-

standardized coefficients may be interpreted to indicate the following relations to GAS: (a) adjusting for the effects of the other variables, the presence of early aggression was associated with a 14.47-point average decrease in GAS at age 17, (b) an adjusted increase of 1 SD in the parent psychopathology scale was associated with a 4.18-point average decrease in GAS, (c) an adjusted 1-point increase in IQ was associated with a 40-point average increase in GAS. Thus an adjusted 15-point (1 SD) increase in IQ was associated with a 6-point average increase in GAS. Similarly, a 15-point decrease in IQ was associated with a 6-point decline in GAS.

The unstandardized coefficients may be interpreted to indicate the following relations to WAIS IQ: (a) adjusting for the effects of the other factors, each additional soft sign was associated with a 4.60-point average decrease in WAIS IQ, (b) an adjusted increase of 1 SD in environmental disadvantage was associated with a 4.47-point average decrease in WAIS IQ, (c) an adjusted increase of 1 SD in parent psychopathology was associated with a 2.28-point average increase in WAIS IQ, (d) the presence of aggression at age 7 was associated with a 7.19-point average adjusted decrease in WAIS IQ.

The standardized path coefficients provide an index of the relative effects of each exogenous variable on GAS or IQ. The three variables (WAIS IQ, parent psychopathology, and early aggression) exerted approximately equal effects on GAS. The effects of environmental disadvantage and early signs on IQ were more sizable than the effects of early aggression and parent psychopathology. Disadvantage and early signs exerted about equal effects on WAIS IQ.

Another path analysis (see Fig. 3) of the factors affecting WAIS IQ was conducted. In contrast to the previous analysis, age-7 WISC IQ was introduced as a control variable. The previously described inclusion rules governed this analysis. When WISC IQ was introduced, the paths from early aggression and parent psychopathology became nonsignificant owing to variance shared with WISC IQ, however, the paths from environmental disadvantage and number of signs to WAIS IQ continued to meet causal criteria. The two variables exerted about equal direct effects on WAIS IQ. Number of signs also exerted indirect effects on WAIS IQ through WISC IQ. Although we did not construct an age-7 measure of environmental disadvantage, we expected early disadvantage to have an adverse effect on cognitive functioning. The Spear-

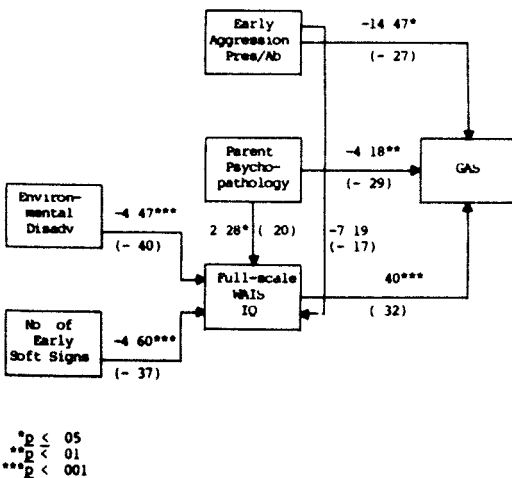


FIG. 2—Trimmed path model showing the effects on GAS and WAIS IQ of the exogenous variables meeting causal criteria. Paths from environmental disadvantage and early signs to GAS were dropped. The path from early aggression and WAIS IQ attained a p value of 10.

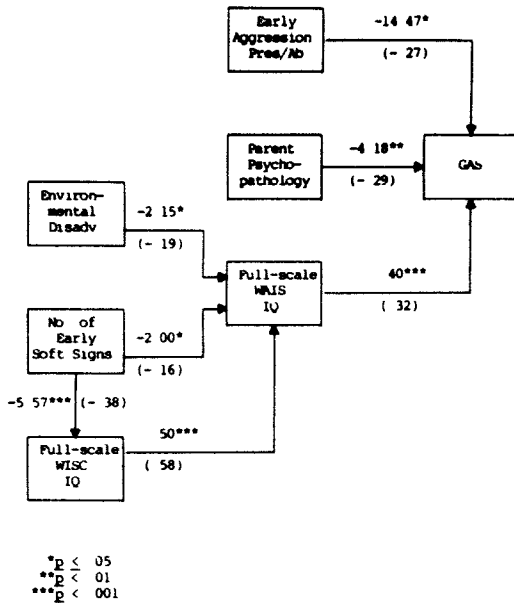


FIG 3—Path model in which age-7 WISC IQ was entered as a control variable. When WISC IQ was entered, the paths from early aggression and parent psychopathology to WAIS IQ no longer met causal criteria.

man correlation between age-7 family income and WISC IQ was $.33, p < .001$. The Spearman correlation between early income and number of signs was nonsignificant. The correlations were then recomputed using parametric statistics. The first-order partial correlation between early income and WISC IQ, controlling for signs, was $.27, p < .01$.

Other analyses relevant to the path models—We considered the possibility that we misspecified our path-analytic models. For example, adolescent psychopathology and WAIS IQ may influence each other bidirectionally, or some third factor may cause both WAIS IQ and GAS. In order to rule out these alternatives, we examined the WAIS IQ and GAS residuals using LISREL procedures (Joreskog & Sorbom, 1981). In each analysis the residuals were uncorrelated, a result that is incompatible with those two possibilities (Kenny, 1979).

Since WAIS IQ is more reliable than any other measure, it is possible that its relation to GAS was enhanced compared to that of the other predictors. In response to this possibility we recomputed the standardized path coefficients based on a correlation matrix in which the bivariate correlations were corrected for attenuation (see Kenny, 1979). The directions of the effects paralleled the findings obtained in the earlier analyses.

To clarify the relation between cognitive functioning and conduct problems still further, three additional path analyses were conducted. In each analysis, we substituted one of three alternative measures of cognitive functioning for WAIS IQ: the acquired knowledge scale, the spatial scale, or age-7 WISC IQ. When the acquired knowledge scale was substituted for WAIS IQ, the path coefficient from cognitive functioning to GAS was significant ($p < .01$), when the spatial scale was substituted, the path coefficient was nonsignificant, when WISC IQ was substituted, the path coefficient was marginally significant ($p < .06$).

One final test was conducted to rule out the possibility that change in IQ from age 7 to 17 (either increase or decrease) rather than trait IQ affected psychiatric functioning. The least-squares regression analyses were repeated with an IQ change score replacing full-scale WAIS IQ. The results of the analysis failed to reveal anything approaching an effect for IQ change.

Logistic regression analysis—Because path analysis calls for the use of continuous dependent variables, GAS was used as a dependent measure within a sample of youths with a conduct disorder or no disorder. GAS therefore served as a proxy for conduct disorder. In order to buttress the results of the path analyses, a logistic regression analysis was conducted in which a dichotomous measure—the presence of conduct disorder versus the absence of any disorder—was regressed on the same five predictors (Cleary & Angel, 1984). The results of the logistic regression indicate that early aggression, parent psychopathology, and WAIS IQ were the best predictors ($p = .05$) of conduct disorder when entered into the regression equation last. The other two factors, environmental disadvantage and signs, were unrelated to the disorder.

Logistic regression analysis was also used to compute the adjusted odds ratio for each of the three risk factors for conduct disorder, controlling for the other two factors (Kleinbaum, Kupper, & Morgenstern, 1982). The adjusted odds ratio for conduct disorder given a standard deviation (15-point) decrease in full-scale IQ was $2.65 (p < .01)$. If the acquired knowledge scale were substituted for full-scale IQ, the adjusted odds ratio given a standard deviation (9-point) decrease was $3.42 (p < .01)$. The adjusted odds ratio for the presence of early aggression was $2.34 (p < .07)$, and for a standard deviation increase in parent psychopathology, $1.89 (p < .05)$.

In another logistic analysis—the presence of any anxiety disorder ($n = 20$) versus the absence of all disorders ($n = 64$)—was regressed on the five predictors. The results indicated that WAIS IQ was unrelated to the presence of anxiety disorders. A parallel analysis indicated that WAIS IQ was unrelated to the presence of affective disorders ($n = 30$). Too few cases were available to examine substance abuse. Cautioning that the n 's are small, these additional analyses suggest that the relation of cognitive functioning to psychiatric status is specific to conduct disorders.

Discussion

The results of the path analyses are consistent with the view that three factors contribute to the development of conduct disorder at age 17: IQ (whether measured at ages 7 or 17), parent psychopathology, and early aggression. As reflected in the standardized path coefficients, each factor exerted almost equivalent effects on psychiatric functioning. Logistic regression analyses suggest that IQ is *specifically* related to conduct disorder, although tests involving larger sample sizes would be warranted in order to cross-validate this finding. Profiles of the mean IQ scores for conduct-disorder and disorder-free subjects taken at different times underline the consistency of the relation of conduct disorder to cognitive functioning. Least-squares and logistic regression analyses, as well as tests for mean differences, link conduct disorder to deficits in acquired knowledge but not spatial ability, suggesting that the origins of the IQ deficits affecting psychiatric status reside in the individual's learning environment. The findings involving change IQ and age-7 WISC IQ suggest that enduring deficits in cognitive functioning affect adolescent psychiatric status.

The acquired knowledge scale is thought to reflect crystallized intelligence, an intellectual ability believed to be highly dependent on past learning and acculturation. By contrast, the spatial ability scale is thought to reflect fluid intelligence, an intellectual ability believed to be highly related to biological/hereditary factors (Schonfeld, 1986). Conduct-disorder-related differences are more pronounced on the age-7 and -17 verbal IQ scales than on contemporary performance IQ scales. Horn (1982) noted that verbal IQ overlaps somewhat with crystallized ability, and performance IQ shares features with fluid ability. Differences found on two of the three age-17 achievement tests are consistent with the pattern of IQ findings. Thus, the results tend to

be consistent with the view that long-term deficits in cognitive functioning, particularly deficits related to acculturational knowledge, lead to conduct disturbance.

The possibility that the findings may be the result of methodological factors needs to be examined. One methodological factor concerns the relative reliabilities of the different measures. The internal consistency reliability of the WAIS IQ for 18- and 19-year-olds in the standardization sample was .97 (Wechsler, 1955), considerably greater than the reliabilities of the environmental disadvantage and parent psychopathology scales. Excessive error variance in the environmental disadvantage and parent psychopathology scales tends to bias the effects to be detected, and differences in reliability favor the detection of IQ-related effects (Kenny, 1979).

In response to the problem of differential reliabilities, the standardized path coefficients were recomputed using disattenuated correlations. The paths from parent psychopathology, early aggression, and WAIS IQ to GAS were strengthened, but their signs remained unchanged. In addition, when the acquired knowledge scale was substituted for full-scale IQ, the reliability of the cognitive functioning variable was weakened, the magnitude of the path coefficient from cognitive functioning to GAS, however, was increased.

Although the path analyses suggest that full-scale IQ has a direct effect on adolescent psychopathology, it may be argued that parent psychopathology, a factor related to adolescent conduct problems, mediates both, and therefore the relation between low IQ and adolescent psychopathology is spurious. A number of findings argue against this view. First, the signs of the path coefficient from parent psychopathology to WAIS IQ in the first two path diagrams indicate that mildly increased parent psychopathology levels are associated with higher, not lower, levels of IQ. Second, the sign of the path did not change when disattenuated correlations were employed. Third, the path from parent psychopathology to WAIS IQ became nonsignificant when WISC IQ was controlled.

The use of WAIS IQ constitutes another potential problem because of the possibility of bias in testing black adolescents. Bias connected to testing, however, is minimal for three reasons. First, the analyses were conducted within, not between, race. Second, the IQ test results are consistent with other sources of data on deviant behavior. That adolescents free of disorder performed better

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than adolescents with a conduct disorder is consistent with a body of literature involving very different samples (Hirschi & Hindelang, 1977). Third, the pattern of cross-age IQ correlations is highly consistent with research involving other samples (Bloom, 1964). On the other hand, because the sample consists exclusively of black males, the generalizability of the results to whites and females is limited. A longitudinal study involving whites and blacks as well as males and females would be of great utility in cross-validating the present findings.

One limitation of the present study consists of the lack of evidence on the distribution of moderate conduct problems in early life. The evidence on early aggressive behavior comes from the psychologists' ratings of the subjects' conduct during testing, hardly an ordinary sample of behavior. It was assumed that the subjects who manifested any aggressiveness during the age-7 psychological examination were likely to be excessively aggressive outside the testing situation since one-to-one psychological testing usually inhibits behavioral excess. Support for this expectation comes from the finding that five of the seven call-back subjects who had positive ratings for aggression at age 7 were found to have a conduct disorder at age 17. This result is consistent with findings based on other measures and other samples (Huesmann et al., 1984; Olweus, 1979).

The results are not consistent with the hypothesis that conduct problems lead to deficits in cognitive functioning. The third path analysis indicated that early aggression was unrelated to age-17 IQ when age-7 WISC IQ was controlled, although a stronger test could have been conducted if a more differentiated measure of early aggression were available. The sturdiest predictors of age-17 IQ outside of age-7 IQ were environmental disadvantage and soft signs, results that are consistent with the view that both biological and environmental handicaps adversely affect cognitive functioning. Parallel relations were mirrored in findings linking both age-7 disadvantage, as captured by family income, and soft signs to age-7 IQ.

The results of the path analyses are inconsistent with the common-antecedents hypothesis. Soft signs, a factor that shared a moderate but significant amount of variance with IQ, did not constitute a third variable that explained the IQ-conduct disturbance association. The other three candidate third factors, parent psychopathology, disadvantage, and early aggression, also did not explain the

IQ-conduct disturbance association. It is possible that some unmeasured variable constitutes the third factor that explains an association. In the present study, however, the four potential third variables were selected on the basis of their known links to IQ and conduct problems.

Before going into some detail on the relation of cognitive functioning to conduct problems, we briefly comment on the other two factors, early aggression and parent psychopathology, found to affect psychiatric status. In line with Olweus (1980), the measure of early aggression used here might be interpreted as reflecting early temperament. One weakness in Olweus's measure of early temperament, however, is that it was based on parent recall of behavior occurring about 7 years prior to interview. On the other hand, the measure pertained to a broad band of behaviors. One strength of the measure of early aggression employed here is that it was based on observations made during the period of interest. At the same time, the observations pertained to a narrow band of behaviors. Even so, both the Olweus measure and the present one were related to later conduct problems, suggesting that temperament might be implicated in the development of antisocial conduct.

Consistent with prior research, parent psychopathology was found to contribute to the development of conduct disorder in children (Olweus, 1980; Rutter et al., 1970). For want of power to test each factor individually, the parent psychopathology scale included items reflecting disturbance in either parent as well as in the marital relationship as a whole. Candidate mechanisms by which parents' psychopathology contributes to conduct problems in their offspring include deviant socialization and impaired child-management skills.

The question of how the deficits in cognitive functioning lead to conduct disorder remains. One explanation is that educational failure, a consequence of cognitive deficits, leads to low self-esteem and antagonism to school, paving the way for conduct disorder (Rutter et al., 1970; Rutter & Giller, 1983). This hypothesis, however, is not consistent with the distributions of conduct disorder and disorder-free subjects expressing dislike for school in response to an item on the age-17 adolescent interview (57% vs 47%, *N* S). It should, however, be noted that the results are based on responses to a single interview item and thus subject to instability.

To develop an alternative hypothesis, we draw upon studies that attempt to describe

how aggressive and rejected children process social information Dodge (Dodge, 1980, Dodge & Frame, 1982) found that aggressive boys tend to show bias in processing particular types of social information Compared to nonaggressive controls, aggressive boys tended to attribute hostility to peers issuing ambiguous social cues—especially cues directed toward themselves Vosk, Forehand, and Figueroa (1983) found that, compared to socially accepted children, rejected children tended to misinterpret affective states in others

It should be noted that characteristics of research on social cognition include the reliance on cross-sectional designs and the use of convenience samples Although not the purpose of the Dodge and Vosk studies, it is difficult, using cross-sectional designs, to test etiologic hypotheses concerning the relation between aggression and cognition (MacMahon & Pugh, 1970) Aggressive behavior may cause attributional biases, or attributional biases may be spuriously related to aggression because third factors, like pervasive developmental difficulties or deviant parental socialization practices, produce both The use of convenience samples makes it difficult to describe the characteristics of the population of aggressive boys (Kleinbaum et al., 1982) With these caveats in mind, the social cognition literature is helpful in developing alternative hypotheses concerning the relation between cognitive functioning and conduct problems

Damon (1981) summarized a line of thought which holds that there should be no distinction between the processes involved in acquiring social and other types of knowledge because "all cognition is intrinsically social in origin" (p. 162) Since IQ constitutes an omnibus measure of cognitive functioning, low test scores are likely to be related to cognitive differences that appear in a variety of circumstances, including situations that call for the processing of social information Consistent with the data presented here, the sources of differences in cognitive functioning, as reflected in IQ test performance, include biological and environmental adversities It is unlikely that the aggressive children studied by Dodge, the rejected children studied by Vosk et al., and the conduct-disorder children studied here (samples which should be overlapping) show only differences in some circumscribed set of cognitive behaviors pertinent to interpreting social cues It seems equally, if not more, plausible that differences in understanding social cues emerge out of the unfavorable learning contexts that give

rise to global deficits in cognitive functioning This explanation is consistent with the findings revealing greater conduct-disorder-related differences on IQ subtests reflecting acculturation (e.g., Information, the acquired knowledge scale) than on subtests assessing a more native spatial ability (e.g., Block Design, the spatial ability scale)

Future research with representative samples of young people can examine a variety of cognitive factors that potentially increase the risk for conduct problems Longitudinal designs could include, at two or more points in time, omnibus cognitive measures, like the Wechsler scales, as well as more fine-grained cognitive measures, like Dodge's and Vosk's Such research would be very useful in testing etiologic hypotheses concerning which specific cognitive differences contribute to conduct problems

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