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A META-ANALYTIC REVIEW OF THE RELATION BETWEEN ANTISOCIAL BEHAVIOR AND NEUROPSYCHOLOGICAL MEASURES OF EXECUTIVE FUNCTION

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ABSTRACT. *Previous narrative reviews of the relation between antisocial behavior (ASB) and neuropsychological tests of executive functioning (EF) have raised numerous methodological concerns and produced equivocal conclusions. By using meta-analytic procedures, this study attempts to remedy many of these concerns and quantifies the relation between ASB and performance on six reasonably well validated measures of EF. Thirty-nine studies yielding a total of 4,589 participants were included in the analysis. Overall, antisocial groups performed .62 standard deviations worse on EF tests than comparison groups; this effect size is in the medium to large range. Significant variation within this effect size estimate was found, some of which was accounted for by differences in the operationalizations of ASB (e.g., psychopathy vs. criminality) and measures of EF. Evidence for the specificity of EF deficits relative to deficits on other neuropsychological tasks was inconsistent. Unresolved conceptual problems regarding the association between ASB and EF tests, including the problem of localizing EF tests to specific brain regions, are discussed.* © *2000 Elsevier Science Ltd*

THE PAST DECADE has witnessed a heightened appreciation of the role of biological influences on antisocial behavior (ASB) (Lykken, 1995; Raine, 1993). Among the biological factors that have been found to be associated with ASB are genetic influences, pre- and perinatal complications (Raine, Brennan, & Mednick, 1994), psychophysiological abnormalities (Raine, 1997), and differences in neurotransmitter functioning (Berman, Kavoussi, & Coccaro, 1997). One major issue that has received heightened attention in recent years is the relation between ASB and both intellectual and neuropsychological functioning. Antisocial groups score approximately 8 points lower on intelligence tests than nonantisocial groups (Heilbrun, 1979; Heilbrun & Heilbrun, 1985; Henry & Moffitt, 1997), although the reasons for this difference are unclear. In

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addition, numerous authors (e.g., Elliott, 1978; Gorenstein, 1982; Raine, 1997) have conjectured that deficits in the brain's executive functions (EFs) are an important correlate or risk factor for ASB. Nevertheless, the relation between ASB and EF remains controversial, largely because previous studies of this association have typically yielded contradictory results (see Kandel & Freed, 1982; Lilienfeld, 1992, for reviews).

The purpose of this article is to quantify the relation between ASB and EF using meta-analytic methods. Specifically, we combine effect sizes from individual studies into a mean estimate of the relation between ASB and performance on EF tests. Although the ultimate goal of research in this area may be to establish causal factors for ASB, that is not the primary purpose of this article. In all of the studies we reviewed, ASB was already present at the time of assessment. Consequently, it is not possible to determine whether EF is a precursor of ASB, a sequela of ASB, or a correlate that is associated with ASB via unidentified third variables.

THE CONSTRUCT OF EF

What is the F in EF?

EF is an umbrella term that refers to the cognitive processes that allow for future, goal-oriented behavior. It is broadly defined as comprising the abilities needed to achieve and maintain a problem-solving set, and includes such processes as planning, organizational skills, selective attention and inhibitory control, and optimal cognitive-set maintenance. Beginning anterior to the central sulcus, the frontal lobes encompass up to 33% of the human cortex. The frontal lobes are responsible for such behaviors as monitoring and coordination of gross muscle movements, speech and language production and processing, and integration of sensations, perceptions, consciousness, and memory into organized and planned behaviors (Fuster, 1980; Stuss & Benson, 1984).

EFs are considered necessary for socially appropriate adult conduct. They allow an individual to be self-sustaining and self-reliant (Lezak, 1995), and include self-awareness, language comprehension and expression, and the regulation of motoric behavior via verbal instructions (Luria, 1973). In addition, the frontal lobes are related to personality dimensions and emotional regulation. When the frontal lobes are damaged, many patients exhibit distinct personality changes. Some become inert, apathetic, and indifferent, whereas others become euphoric, restless, and impulsive (Stuss & Benson, 1984).

Blumer and Benson (1975) characterized two classes of personality changes that often follow frontal damage: the *pseudodepressed* and *pseudopsychopathic* personality constellations. Pseudodepressed personality, which is marked by apathy, lack of motivation, depressed cognitions, and an inability to plan, is associated with damage to the dorsolateral area of the frontal lobes. Pseudopsychopathic personality, which is characterized by jocular attitude, disinhibition, extreme self-indulgence, poor judgment, and inappropriate sexual humor, is associated with damage to the orbitomedial cortex. Individuals with the latter personality constellation also typically exhibit perseverative responding, an inability to appreciate one's impact on others, and a tendency toward immediate gratification (Gorenstein, 1982). Damasio, Tranel, and Damasio (1990) labeled this set of personality changes produced by frontal damage "acquired sociopathy." It should be noted, however, that patients with frontal lobe damage typically resemble "partial" psychopaths (Elliott, 1978; Hare, 1984) and do not possess su-

perficial charm, poise, and some of the other traditional personality traits of psychopathic personality (Cleckley, 1941/1982).

Because the frontal lobes are intimately associated with EFs, some researchers tend to reduce the definition of EFs to what the frontal lobes do. Nevertheless, this definition is problematic and somewhat tautological. Because of the complexity of the connections between the frontal lobes and other brain regions, it is difficult to ascertain whether EFs are produced by the frontal cortex, the neuronal tracts that connect it to other areas, or both. In light of the lack of clear evidence as to the specifics of EF localization, perhaps the most that can be said is that the frontal lobes are clearly involved in EFs. With this caveat in mind, Pennington and Ozonoff (1996) used the term *frontal metaphor* to make clear that the precise nature of the relation between EF and the frontal lobes is unknown.

Moreover, without direct knowledge of neuropathology (through neural imaging or direct anatomical investigation), executive dysfunction can only be inferred. Similar problems exist when performance on neuropsychological tests derived from individuals with developmental or personality disorders (including disorders characterized by chronic ASB) is compared with that of individuals who possess neuropathology acquired later in life. Although the behavioral manifestations of these causes may appear similar, their anatomical etiologies may be quite different (Luria, 1973).

Neuropsychological Tests of EF

EFs are difficult to operationalize because they can be observed only as changes in lower-level cognitive functions (Borkowski & Burke, 1996). As such, there are no unambiguous or direct indicators of executive dysfunction. EFs encompass four distinct cognitive domains: volition, planning, purposive action, and effective performance (Lezak, 1995). Therefore, valid tests of EF must incorporate some or all of these domains. Furthermore, valid measures of EF should be able to differentiate patients with frontal lobe damage from patients with other forms of brain damage. Therefore, in this meta-analysis we only included tests that fulfilled the following criteria: (a) the test incorporated at least one of the aforementioned theoretical domains of EF (e.g., planning) and either (b) or (c) (or both): (b) the test has been found in at least several studies to differentiate patients with focal frontal lobe lesions from either patients with diffuse brain damage or patients with focal lesions in other areas, and/or (c) the test has been found in brain imaging research to preferentially activate the frontal cortex.

Based on these criteria, results from six reasonably well validated tests of EF were included in this meta-analysis. Specifically, the following tests were examined: (a) the Category Test of the Halstead-Reitan Neuropsychological Battery (HRNB), (b) the Qualitative (Q) score on the Porteus Mazes Test, (c) the Stroop Interference Test, (d) Part B of the Trail Making Test (TMT), (e) the perseverative error score on the Wisconsin Card Sorting Test (WCST), and (f) Verbal fluency Tests (see "EF Measures" for a brief description of each task and the evidence for its validity as a marker of frontal dysfunction). Other tests that are sometimes conjectured to assess EF, such as the Sequential Matching Memory Test (SMMT), Necker cube reversals (see Hare, 1984, for criticisms of these two tests), and the Motor Scale of the Luria-Nebraska Neuropsychological Battery (see Malloy, Webster, & Russell, 1985; McKay & Golden, 1979), were not included because they did not unambiguously meet the aforementioned criteria. It should be borne in mind, however, that none of the six tests of EF used in this meta-

analysis is entirely specific to frontal damage, and that performance on these measures can be adversely affected by damage to other brain areas (see "EF Measures"). Thus, each of these six measures should be viewed as useful, although probabilistic, markers of executive dysfunction.

Furthermore, it is likely that different tests of EF assess functions subserved by different frontal lobe regions. Data from positron emission tomography (PET) research, for example, indicate that the dorsolateral region of the frontal cortex exhibits increased activity during the WCST (Rezai et al., 1993). Nevertheless, most EF tasks have not been consistently localized to specific frontal regions. Therefore, this meta-analysis will not attempt to differentiate effect sizes by specific regions of the frontal lobes.

OPERATIONALIZATION AND ASSESSMENT OF ASB

ASB has generally been operationalized in two major ways. First, ASB has been operationalized in terms of categorical clinical syndromes, including diagnoses of antisocial personality disorder (ASPD) and conduct disorder (CD), and the personality constellation of psychopathy. These syndromes are characterized by chronic irresponsible behavior, disregard for the rights of others, poor behavioral controls, and an inability to conform to social norms. CD is related to ASPD in two additional ways. First, in order to receive a diagnosis of ASPD, the individual must meet criteria for CD prior to age 15 (American Psychiatric Association, 1994). Second, Robins and Regier (1991) found that 25 to 50% of children with CD are later diagnosed with ASPD. Individuals receiving the diagnosis of either ASPD or CD will be included in this meta-analysis.

Psychopathic personality (psychopathy) is a constellation of personality traits first described in detail by Cleckley (1941/1982). It includes such features as lack of remorse or sincerity, dishonesty, egocentricity, and impoverished affective reactions. Several findings indicate that psychopathy is characterized by learning deficits that distinguish it from other forms of ASB. For example, Lykken (1957) and Newman and his colleagues (e.g., Newman & Kosson, 1986), demonstrated that psychopaths, compared with other antisocial individuals, are more prone than other individuals with ASB to poor passive avoidance learning. Psychopathy has most commonly been assessed by means of three instruments: the Psychopathic Deviate scale of the Minnesota Multiphasic Personality Inventory (MMPI Pd; Hathaway & McKinley, 1942), the Socialization scale of the California Psychological Inventory (CPI So; Gough, 1969), which is scored in reverse as a measure of psychopathy, and the Psychopathy Checklist and Psychopathy Checklist-Revised (PCL and PCL-R; Hare, 1991). Studies that used any of these assessment methods will be used in the meta-analysis; however, because of the small number of studies that assessed psychopathy, a separate analysis of each assessment method will not be performed.

The second way in which ASB has been examined is by means of the legal concepts of criminality and delinquency, which comprise behaviors that are unlawful and often lead to incarceration. Because criminality and delinquency are significantly correlated with the three clinical syndromes already discussed (Abram, 1989; Moffitt, 1988), studies examining criminality and delinquency will be included in the meta-analysis. Criminality and delinquency were assessed through the use of self-report measures, criminal records, and (in the case of some studies examining delinquency) legal adjudication.

Two questions arise when considering these operationalizations. The first is their concurrent validity with other putative indicators of ASB. The PCL-R correlates with

the *Diagnostic and Statistical Manual of Mental Disorders*, third edition-revised (*DSM-III-R*; American Psychiatric Association, 1987) criteria for ASPD at $r = .55$, and $r = .30–.35$ with the MMPI-Pd and CPI-So scales (Hare, 1991). Moreover, Frick, O'Brien, Wootton, and McBurnett (1994) found that a childhood measure of psychopathy correlated with the *DSM-III-R* criteria for CD at $r = .35–.50$. Although it is difficult to determine the amount of overlap between the clinical syndromes and legal status, the prevalences of ASPD and psychopathy within prison samples are estimated to be 50% and 15–20%, respectively (Harpur, Hare, & Hakstian, 1989). Finally, the prevalence of CD in delinquent populations has been estimated at 63 to 87% (Eppright, Kashani, Robinson, & Reid, 1993; Vitelli, 1996).

The second question that arises is whether violence is related to higher rates of EF deficits among individuals with ASB. Some authors have suggested that biological influences may be most relevant to forms of ASB characterized by physical aggression (Brennan & Raine, 1997). Because most researchers have not explicitly differentiated violent from nonviolent individuals in their studies, however, violent behavior was not examined as a moderator in this meta-analysis.

GOALS OF THE PRESENT REVIEW

Although there has been much speculation as to possible executive dysfunction in ASB, the research evidence has been equivocal. Qualitative reviews using the box score or "voting" method (Schmidt, 1992) have yielded inconclusive results. For example, in their narrative literature review, Kandel and Freed (1982) concluded that "[t]he trends in the data indicate that frontal-lobe dysfunction cannot be ruled out in relation to any type of crime . . ." (p. 411) (see Lilienfeld, 1992, for similar equivocal conclusions). Furthermore, they criticized this body of literature on many grounds, including use of different operationalizations of ASB, failure to use validated measures of frontal lobe dysfunction, and a lack of control for the effects of nuisance variables (e.g., education level, socioeconomic status, age).

Other authors have investigated group differences in tests of EF using relatively specific operationalizations of ASB. For example, Pennington and Ozonoff (1996) found no relation between CD and EF in a quantitative review. However, they included only published studies in their analysis and did not limit their review to well-validated measures of EF. To date, a meta-analysis of this literature comparing different operationalizations of ASB (e.g., psychopathy, CD) has not been performed.

This meta-analysis attempted to clarify the relation between ASB and EF by examining the differences in effect sizes between antisocial and comparison groups. Compared with Kandel and Freed's (1982) review, a larger number of studies were examined using a broader range of operationalizations of ASB. Furthermore, several of the nuisance variables discussed by Kandel and Freed were examined as potential moderators of the relation between ASB and EF. This meta-analysis focuses exclusively on neuropsychological indices of EF deficits. Although the results of several recent functional brain imaging studies (e.g., Goyer & Semple, 1996; Raine et al., 1994) suggest the possibility of hypofrontality among certain individuals with ASB (e.g., murderers), these studies are too few in number to permit a systematic quantitative analysis.

Important questions remain, however, concerning the specificity of EF deficits for individuals with ASB. Do antisocial individuals tend to perform poorly on all neuropsychological tests or on EF tests only? To begin to address this question, three non-

EF tests, Part A of the TMT, the number of categories achieved on the WCST, and the mental test age (TA) of the Porteus Mazes Test, were analyzed as "control" measures. These three tests were selected for two reasons. First, they are hypothesized to be less sensitive markers of EF than the other six measures used in this meta-analysis (Lezak, 1995) and are often used to compare performance on the parts of these tests that are used to assess EF. Second, in the studies that assessed EF, these three tests were the most frequently used non-EF indices.

METHOD

Search Strategy

The search for studies to be included in the meta-analysis began by examining several computerized data bases (PsycINFO, MEDLINE, ERIC, and Dissertation Abstracts International) with 22 keywords relevant to ASB and EF (e.g., "antisocial," "psychopathy," "sociopathy," "frontal," "executive"). In addition, the reference sections of literature reviews and research studies located through this method were scrutinized for articles that might have been overlooked in the computer search. More recent articles were obtained by searching the indices of journals in the fields of abnormal psychology, psychiatry, and neuropsychology.

Inclusion Criteria

To be included in this meta-analysis, a study had to satisfy *all* of the following criteria:

1. The dependent measures had to include one or more of the six aforementioned measures of EF (i.e., Category Test of the HRNB, the Q score of the Porteus Mazes Test, Stroop Interference Test, Part B of the TMT, the perseverative error score of the WCST, the HRNB Category Test, and Verbal Fluency Tests). Studies that combined two or more of these measures into a battery, but which did not report data on separate EF measures (e.g., Skoff & Libon, 1987), were not included in the meta-analysis.
2. The study used ASB as the independent variable and included one or more of the following groups: psychopathic personalities, individuals with either ASPD or CD, criminals, delinquents, psychiatric comparison participants, or normal comparison participants. Studies that compared antisocial individuals with brain damaged individuals (e.g., Krynicki, 1978) were excluded. In addition, a study (Burgess, 1992) that combined ASPD patients with patients with other personality disorders (e.g., borderline personality disorder) was not included because it did not permit separate analysis of EF deficits among ASPD patients.
3. Either of these two conditions was satisfied: (a) the results were presented in such a way that the effect sizes could be calculated with a reasonable degree of accuracy (cf. Hoffman, Hall, & Bartsch, 1987), or (b) the authors provided information to the first author permitting the calculation of effect sizes.

EF Measures

Each of the six tests used in this meta-analysis, along with the evidence for its specificity to frontal lobe damage, is discussed briefly below. To permit an examination of

whether EF measure moderated the magnitude of effect sizes, separate analyses were conducted by each test.

1. *Category Test of the HRNB.* Each subset of the Category Test consists of a series of forms characterized by a unifying principle (e.g., shape, the proportion of the forms in solid lines). Each form is displayed, one at a time, to the participant, who attempts to ascertain the underlying grouping principle in each subset. The participant receives one guess per turn and receives feedback after each guess. The Category Test was reported by Halstead (1947) to distinguish patients with frontal damage from patients with lesions in other areas. Others (e.g., Wang, 1987) have replicated these findings, although there is evidence that performance on this task is affected by damage to other brain areas as well (Reitan & Wolfson, 1995).
2. *The Porteus Mazes Q score.* The Porteus Mazes consist of mazes of increasing difficulty. Participants must trace each maze without entering blind alleys. The Porteus Mazes yield two scores: the test age (TA), that is, the maze with the highest level of difficulty that participants complete; and the Q score, that is, the number of errors ostensibly reflecting impulsivity (e.g., crossed lines, pencil lifts, changed directions) made by the participant. The Q score has been found to differentiate patients with frontal lesions from patients with lesions in other areas (Levinson, Meadow, Atwell, Robey, & Bellis, 1953; Milner, 1964; see Stuss & Benson, 1984). Although the studies using mazes in this meta-analysis were based almost exclusively on the Porteus Mazes, one study (Moffitt & Henry, 1989) used the Mazes subtest from the Wechsler Intelligence Scale for Children-III (Wechsler (1991).
3. *Stroop Interference Test.* In this test, the participant is shown the names of colors printed in conflicting ink colors (e.g., the word "red" printed in blue ink) and is asked to name the color of ink in which the word is printed. The Stroop Interference Test has been reported to distinguish patients with frontal damage from patients with lesions in other areas (Milner, 1963, 1964), and frontal patients often read the words instead of naming the ink color (Spreen & Strauss, 1991). In addition, performance on the Stroop Interference Test has been found to be accompanied by right frontal activation as measured by PET (Bench, Frith, Grasby, & Friston, 1993). Blenner (1993), however, found that the Stroop Interference Test failed to distinguish frontal lesion patients from temporal lesion patients.
4. *Part B of the TMT.* This test requires participants to connect a series of circles with a pen. In Part A, the circles are numbered from 1 to 25, and participants must connect them in order. Part B contains circles numbered from 1 to 13 and circles lettered from A to L, and participants must connect the circles in order by alternating from numbers to letters (i.e., 1-A-2-B-3-C, etc.). The scores for both Part A and B are the total amounts of time required to complete the sequence. Part B has been found to be sensitive to frontal lobe damage (e.g., Boll, 1981), although the specificity of this test to the frontal regions requires clarification (Lezak, 1995; Reitan & Wolfson, 1995).
5. *Perseverative error score of the WCST.* The WCST requires participants to sort a deck of 64 cards according to the dimensions of color, form, and number. Because participants are not told about these sorting dimensions, they must educe their existence on the basis of examiner feedback. Following each card placement,

the examiner informs participants whether their responses are correct or incorrect, but provides no additional information. After a certain number of consecutive correct responses have been made, the sorting criterion is suddenly altered without the participant's knowledge. Perseverative errors reflect the participant's failure to shift to the new sorting criterion. The WCST perseverative errors score was reported by Milner (1963, 1964) and Drewe (1974) to differentiate patients with frontal lesions from patients with nonfrontal lesions, although the evidence for its specificity to the frontal cortex has not been entirely inconsistent (Robinson, Heaton, Lehman, & Stilson, 1980).

6. *Verbal fluency tests.* These measures assess individuals' capacity to produce spontaneous speech, and typically require participants to name as many words as possible beginning with a given letter (e.g., F) within a specified time interval (usually 60 seconds). Milner (1963, 1964) reported that patients with frontal damage produced significantly fewer responses on verbal fluency tests compared with patients with lesions in other brain areas. These findings have been replicated by other authors (e.g., Benton, 1968; Perret, 1974). Moreover, data from PET (Frith, Friston, Liddle, & Fracknowiak, 1991) and functional magnetic resonance imaging (fMRI) scans (Phelps, Hyder, Blamire, & Shulman, 1997) indicate that the left frontal cortex is activated during verbal fluency tasks (Phelps et al., 1997).

Non-EF Tasks

As noted earlier, an analysis was conducted on tests that are hypothesized not to rely heavily on EFs in order to examine the specificity of the relation between ASB and tests of EF. Specifically, completion time for Part A of the TMT, the number of categories achieved on the WCST, and the TA score from the Porteus Mazes Test were examined as "control" measures for the corresponding scores on these tests that appear to assess EFs (see "EF Measures").

ASB Measures

The construct of psychopathy was operationalized in almost all cases by means of 3 commonly used indices: the PCL/PCL-R, the MMPI Pd scale, and the CPI So scale. The construct validity of each of these methods has been supported by an extensive body of evidence (see Hare, 1991, for a review of the construct validity of the PCL and PCL-R, and (Duckworth & Anderson, 1995) and Gough, 1994, for reviews of the construct validity of the Pd and So scales, respectively), although both the Pd and So scales have been criticized for their inadequate coverage of the core personality features of psychopathy (Lilienfeld, 1994, 1998). The scoring of the CPI So scale was reversed in analyses to permit comparison with other measures of psychopathy and ASB. In the case of the one study (Lilienfeld, Hess, & Rowland, 1996) in which both the CPI So scale and measures of psychopathy other than these three indices were used, the CPI So scale was selected to permit comparability with other studies in the meta-analysis. One study included in the meta-analysis (Hare, 1984) used a global rating based on the Cleckley (1941/1982) criteria, which was the precursor of the PCL. In addition, one other study (Schalling & Rosen, 1968) used a similar global rating of Cleckley psychopathy.

The focal groups were classified into one or more of the following categories of

ASB: ASPD, CD, psychopathy, criminality, and delinquency. ASPD and CD were used as grouping variables only if the criteria specified in the *Diagnostic and Statistical Manual of Mental Disorders*, third edition (*DSM-III*; American Psychiatric Association, 1980) or *DSM-III-R* (American Psychiatric Association, 1987) were used to classify participants (none of the studies reported here used *DSM-IV* [American Psychiatric Association, 1994] criteria). In studies in which the categories overlapped (e.g., when inmates were divided into psychopaths and nonpsychopaths), both descriptors were recorded, but the analyses were conducted using the most specific category (e.g., psychopathy/nonpsychopathy). These five operationalizations of ASB were examined as moderators of the relation between ASB and EF.

Calculation of Effect Sizes

Effect sizes were calculated using the computerized *Meta-Analysis Programs* (Schwarzer, 1994). The formulas used for the calculations were taken from Hedges and Olkin (1985). An effect size is the magnitude of the difference between two groups in standardized terms, and is free of the original measurement unit (Cohen, 1988). The effect size is calculated by dividing the difference between the means of the groups by the standard deviation (Glass, 1976). Because the pooled within-group standard deviation has about half of the sampling error of the control group standard deviation, this statistic was used in the calculation of effect sizes. The formula for the effect size used in this study has a small-sample bias that was taken into account and adjusted for in the computation of effect sizes (see Hedges & Olkin, 1985, for the formula for the unbiased estimator, d). The results of studies that reported only the Pearson product moment coefficient, r, were converted to d (see Cohen, 1988).

A potential problem arises when multiple studies are published by the same research team. Although different participants were used in many of these studies, the similarity in methodology could lead to spuriously similar effect size values. Because there is no straightforward way to avoid this problem, the analyses were conducted using multiple studies from the same research teams, but studies that used the same samples over multiple occasions were not all included. Instead, the one study out of the set that provided the most data on EF was used.

Mean effect sizes were calculated as both unweighted and weighted estimates. The averaged effect sizes from the studies were combined into a simple grand mean estimate of the degree of association of EF and ASB. Greater weight was accorded to larger studies by weighting each study effect by its respective sample size (Hedges & Olkin, 1985).

Tests of Homogeneity of Effect Sizes

The assumption in pooling effect sizes is that all of these effect sizes derive from a single population. This assumption can be tested by calculating the amount of variation within the observed effect sizes. To determine whether the studies could be described as sharing a common effect size, a test of homogeneity was performed using the Q statistic, which is distributed as χ^2 (Hedges & Olkin, 1985). When the value Q equaled or exceeded the critical value associated with an a priori alpha level (in this case, $p = .05$), the samples were examined for possible moderator variables in an effort to reduce heterogeneity (see Hedges & Olkin, 1985).

The File Drawer Problem

One of the major criticisms of meta-analyses is that they often rely on published studies, which may not reflect the total population of studies conducted (Rosenthal, 1991; Sharpe, 1997). Two commonly suggested solutions to this problem were used in this meta-analysis. First, we compared the effect sizes from published versus unpublished studies (Sharpe, 1997). Second, for both the overall effect size and the effect size within each EF measure and operationalization of ASB, we calculated the "fail-safe *N*," which is an estimate of the number of studies with no relation between variables (d = 0.00) that would be needed to reduce the mean effect size to a defined critical level (see Orwin, 1983, for the formula for the fail-safe *N*).

Potential Moderator Variables

If a relation between EF and ASB is found, it may be influenced by other factors (e.g., moderator variables) that are partly responsible for the observed effects of the variables of interest. In addition to EF measure and operationalization of ASB, (a) age, (b) sex, (c) ethnicity, and (d) intelligence were examined as moderators in exploratory analyses. Effect sizes were correlated with each of these four variables both *within* and *between* ASB and comparison groups. For within-group analyses, effect sizes were correlated with each of four potential moderators within both ASB and comparison groups. For between-groups analyses, effect sizes were correlated with the difference between ASB and comparison groups on each of four potential moderators. Intelligence was of particular interest in this study in view of findings that ASB and intelligence are negatively correlated (Wilson & Herrnstein, 1985; Lynam, Moffitt, & Stouthammer-Loeber, 1993). Moreover, there is a significant negative correlation between intelligence and at least one manifestation of executive dysfunction, viz., impulsivity (White et al., 1994). As a consequence, a spurious relation between ASB and EF might result if the differences between ASB and comparison groups were not taken into account.

The age of the participants was coded as a continuous variable, using the mean age of the ASB and comparison groups. For studies that reported only the age range of the groups, the midpoint of the range was used. The differences in age between ASB and comparison groups were also recorded. Appropriate information regarding age was available in 33 studies. Wherever available (n = 36), the proportion of female participants in each group was recorded as a continuous variable, and the difference in the proportions of females between groups was calculated. Ethnicity was coded in a manner similar to that described for sex. Wherever available (n = 16), the proportions of non-White participants were recorded for each group, and the difference in the proportions of ethnic minorities between groups was calculated. Finally, to examine the potential moderating role of intelligence, scores from any well-validated measure of general intelligence, including full scale or subtest scores from versions of the Wechsler Adult Intelligence Scale, Wechsler Intelligence Scale for Children (Wechsler, 1991), and Shipley Institute of Living Scale (Shipley, 1940), were used whenever available.

RESULTS

Analysis of EF Tests

Thirty-nine studies yielding a total of 4589 participants were included in the meta-analysis. Table 1 presents a summary of the studies and effect sizes for the EF mea-

sures. The effect sizes of each measure within a study were averaged to produce a single overall effect size. As a guide for the interpretation of effect sizes, Cohen (1988) considered effect sizes of .2 standard deviations to be small, effect sizes of about .5 standard deviations to be medium, and effect sizes of .8 standard deviations or higher to be large. A stem-and-leaf plot of the distribution of these effect sizes (see Rosenthal, 1991) is displayed in Figure 1. These combined effect sizes were averaged, yielding a grand mean effect size of .57 standard deviations difference between the ASB and comparison groups. Furthermore, 79% of the effect sizes were positive. The effect sizes were then weighted by their respective sample sizes and averaged. The grand mean effect size based on the weighted effect sizes was a .62 standard deviations difference between the ASB and comparison groups. This effect size is significantly different from zero ($z = 18.60$; $p < .001$) and is in the medium to large range.

A test of homogeneity was performed using the weighted effect sizes. The results were statistically significant ($Q = 303.21$, $p < .001$) and indicate that the sample of effect sizes is heterogeneous. Therefore, the common weighted effect size does not appear to derive from a single underlying population.

In an effort to reduce heterogeneity, effect sizes from each study were grouped by separate operationalizations of ASB and separate tests of EF, and weighted mean effect sizes and tests of homogeneity were computed for each operationalization. Table 2 displays the mean effect size, value of Q, and reduction in the value of Q associated with each operationalization and EF measure.

As seen in Table 2, the weighted mean effect sizes across operationalization of ASB ranged from .08 to .94, and all were significantly different from 0 (all ps $< .001$). When operationalization of ASB was taken into account, there was a significant reduction in heterogeneity for each group. Nevertheless, the effect sizes were still significantly heterogeneous for all operationalizations except ASPD and CD. Table 2 reveals that the mean effect sizes for criminality and delinquency were in the large range, whereas the effect sizes for CD and psychopathy were in the small to medium range. The effect sizes for ASPD, although statistically significant, were negligible. Because the number of studies in each group was too small after grouping by operationalization, further subgrouping was not conducted.

Across EF measures, the unweighted mean effect sizes ranged from .24 to .80 standard deviations difference between groups, and the weighted average effect sizes ranged from .24 to .74 standard deviation difference between groups (see Table 2). All of the weighted average effect sizes were significantly different from zero (all ps $<$ .001). The largest effect size was found for Porteus Mazes Q score, which was in the large range, whereas the effect sizes for the other EF tasks were in the small to medium range. Nevertheless, all tests of homogeneity were significant, suggesting that all studies within each grouping do not share a common population effect size. Further subgrouping of the effect sizes for each measure could not be conducted because the number of effect sizes for each group was too small.

Analysis of Non-EF Tests

As displayed in Table 2, Trails A produced an unweighted average effect size of .39 standard deviations difference between ASB and comparison groups, which was almost identical to the amount of difference for Trails B (i.e., .40). Moreover, Trails A was associated with a weighted effect size of .34 standard deviations difference ($z = 5.26$, $p < .001$), which was almost identical to the weighted effect size for Trails B

TABLE 1. Effect Sizes for Studies Included in Meta-Analysis

Reference	Operationalization	Assessment Method	Age	SD	Females (*n*)	Minorities (*n*)	*n*	IQ	*SD*	Measure	Effect Size
Appellof (1985)	Delinquent	Criminal records	15.4			10	30			WCST-Cat	0.466
	Normal controls		16.3			10	30			WCST-Per	0.4723
										Porteus-Q errs	0.449
										Word Fleuncy	0.5569
Aronowitz et al. (1994)	CD	*DSM-III-R*	15	1.65	6		14			WCST-Errors	0.3915
	Clinical controls		15	1.65	2		6			TMT-A	1.2204
										TMT-B	1.035
										WCST-Cat	0.6198
Berman & Siegel (1976)	Delinquent	Legal adjudication	16.1	0.75	0	14	45	87.49	11.84	Category test	0.7358
	Nondelinquent		16.1	0.75	0	14	45	101.78	11.47	TMT-A	0.9931
										TMT-B	0.6943
Bihrle (1995)	Criminal	Criminal records	19.5		0	0	81			Word Fleuncy	−0.2217
	Noncriminal		19.5		0	0	108			Booklet Category test	0.1415
Deckel, Hesselbrock, & Bauer (1996)	Delinquent	*DSM-III-R*	23.3	1.8	0	0	34			Porteus TA score	−0.3628
	Nondelinquent		22.8	1.5	0	0	57				
Devonshire, Howard, & Sellars (1988)	Psychopathically disordered	1959 Mental Hygiene Act (G.B.)					22			Nelson WCST-Per	−0.1991
	Psychiatric controls						27			Nelson WCST-Cat	0.4266
Doctor & Winder (1954)	Delinquents	Criminal records	15.33		0	0	60	94		Porteus-Q errs	0.9424
	Normal controls		15.33				60	94			
Fooks & Thomas (1957)	Delinquent/psychopath	Criminal records and clinical judgment	14.85		25		50	95.9		Porteus-Q errs	1.0579
	Normal controls		14.95		25		50	93.65			
Giancola, Mezzich, & Tarter (1997)	CD[a]	*DSM-III-R*	15.8	1.5	40	20	40	8.58	2.7	Porteus-Q errs	0.3383
	CP/PSUD[a]	Criminal records	16.1	1.3	119	34	119	8.76	2.82	Stroop	0.3711
	Normal controls		15.7	1.3	90	21	90	9.91	2.52	Go/No-go	0.3948
Gibbens (1958)	Delinquents	Criminal records	15	1.65			191			Porteus-Q errs	1.0375
	Normal controls						52				
Gillen & Hessebrock (1992)	ASPD, alcoholic	*DSM-III*	23.39	1.69			34	106.53	11.29	TMT-A	0.098
	Non-ASPD, alcoholic		22.82	1.45	0	0	57	108.18	11.51	TMT-B	0.0646
										Porteus-Q errs	0.6554
										COWAT	0.0153
										WCST-Per	0.0623
Gillstrom (1994)	Psychopathy, inmates	PCL-R and criminal records	32.24	9.18	0		17	102.56	12.65	Category test	0.3649
	Nonpsychopathy, inmates		31.25	8.4			28	105.39	10.76		

Gorenstein (1982)	Psychopath	CPI-So and RDC-ASPD	26.5	7.5			20			WCST-Per	1.2544
	Nonpsychopath[b]		29.3	8.8			23			Stroop comp time	0.6729
	Normal controls[b]		19				18				
Hare (1984)	High psychopathy, inmates	Hare RSAP	30.2	8.1	0	0	14	103.4	12.2	WCST-Per	0.0733
	Medium psychopathy,[b] inmates		30.3	7.8	0	0	16	100.8	11.1	WCST-Cat	0
	Low psychopathy,[b] inmates		34.7	9.9	0	0	16	103.6	9.4		
Hart, Forth, & Hare (1990)	High psychopathy, inmates	PCL-R	30.8	8.6			22	9	2.2	COWAT	−0.0444
	Medium psychopathy, inmates[b]						41	9.1	2.5	TMT-A	−0.1127
	Low psychopathy, inmates[b]						27	9.1	2.1	TMT-B	0.4789
	High psychopathy, inmates	PCL-R	29.8	7.2			32			COWAT	0.1
	Medium psychopathy, inmates[b]						95			TMT-A	0.0152
	Low psychopathy, inmates[b]						40			TMT-B	−0.0308
Hurt & Naglieri(1992)	Delinquents	Criminal records	15.67	0.58	0	0	30			Modified Stroop	0.8134
	Normal controls		15.5	0.67	0	0	30				
Langley (1989)	Delinquent	Court referred	15.6	1.2	0	118	155	74.01	15.66	TMT-A	0.4176
	Normal controls		16.1	1.1	0	24	103	108.8	17.54	TMT-B	0.426
										Word Fluency-Raw	0.9023
LaPierre, Braun, & Hodgins (1995)	High psychopathy, inmates	PCL	33.47	8.53	0		30			Porteus Q errs	1.417
	Low psychopathy, inmates		33.47	8.59	0		30			WCST-Per	0.486
										WCST-Cat	
Lilienfeld, Hess, & Rowland (1996)	Psychopathy	CPI-So	18.89	2.58	61	38	101			Porteus-Q errs	0.199
										Porteus TA score	
Lueger & Gill (1990)	CD	*DSM-III*	15	1.22	0		21			WCST-Per	0.8301
	Normal controls		16.2	1.05	0		20			WCST-Cat	0.9798
										TMT-B	0.348
										TMT-A	0.3778
Malloy, Noel, Rogers, Longabaugh, & Beattie (1989)	ASPD, alcoholic	*DSM-III*	32.1	7.3			36	77.1	12.6	Category test	0.0069
	Clinical controls		40.2	12.7			146	78.1	12.3	TMT-B	0
Moffitt & Henry (1989)	Delinquent, non-ADD[a]	Self-report and adult nomination	13		44		89			Mazes-Q score	0.2338
	ADD, nondelinquent[b]		13		1		14			TMT-B	0.2153
	Normal controls[b]		13		276		549			WCST-Per	0.2449
	ADD, delinquent[a]		13		2		21				
O'Keefe (1975)	Delinquents	Criminal records and court ordered	11.92	1.17			40			Porteus-Q errs	−0.6179
	Normal controls										

(*continued*)

TABLE 1. Continued

Reference	Operationalization	Assessment Method	Age	SD	Females (*n*)	Minorities (*n*)	*n*	IQ	*SD*	Measure	Effect Size
Porteus (1942)	Delinquents	Criminal records and clinic referred	14+		100		100			Porteus-Q errs	1.4001
	Normal controls		14+		100		100				
	Delinquents	Criminal records	14+		0		50			Porteus-Q errs	1.8981
	Normal controls	Clinic referred	14+		0		31				
	Delinquents	Criminal records	14+		0		100			Porteus-Q errs	1.373
	Normal controls	Clinic referred	14+		0		100				
	Inmates	Criminal records	14+		0		100			Porteus-Q errs	1.8751
	Normal controls	Clinic referred	14+		0		100				
Porteus (1945)	Delinquents	Criminal records	15	1.65			50			Porteus-Q errs	1.1698
	Normal controls						25				
	Inmates	Criminal records	15	1.65			100			Porteus-Q errs	1.4087
	Normal controls						50				
Rirle (1993)	High psychopathy	PCL-R	34.48	7.61	0	26	43			TMT-B	−0.0263
	Medium psychopathy[b]				0	23	36			TMT-A	0.1032
	Low psychopathy[b]				0	23	46				
Schalling & Rosen (1968)	High psychopathy	Global ratings of Cleckley psychopathy	31.42	6.77	0	0	60	106.63	10.23	Porteus-no. of trials	0.6877
	Inmates									Porteus-Q errs	0.6377
	Low psychopathy		27.65	5.81	0		23	109.35	13.02		
	Inmates										
Smith, Arnett, & Newman (1992)	High psychopathy	PCL-R and criminal records	25.88	4.2	0	0	37	96.73	11.56	COWAT	0.3912
	Inmates										
	Low psychopathy		26.03	4.38	0	0	32	97.42	8.8	Stroop time	0.196
	Inmates									TMT-B	0.0452
										TMT-A	−0.0763
Sobotowicz, Evans, & Laughlin (1987)	Delinquents	Criminal records			0	25	50	86.62		Category Test	0.7885
	Nondelinquents				0	25	50	87.28			
Sullivan (1992)	CD	*DSM-III-R*	15			0	11	104.9	13.9	Stroop no. of colors read	−0.6141
	Clinical controls					0	12	110.4	11.8	WCST-Per	0.0276
	Normal controls						10	116.6	10.2	Book Category Test	0.3162
										COWAT	−0.0476
										WCST-Cat	−0.2766

Sutker, Moan, & Swanson (1972)	Psychopaths	MMPI	27.3		0	43	110		Porteus-Q errs	−0.5529
	Antisocial psychotics		26.6			27	103.6			
	Prison normals		27.6			24	109.9			
Sutker, Moan, & Allain (1983)	Psychopaths, inmates	MMPI	29.11	7.75	0	44	116.39	15.53	WCST-Per	−0.2266
	Nonpsychopaths, inmates		29	4.95	0	14	119.62	11.64		
Williams-Timo (1989)	CD	*DSM-III-R*	16			20			Category Test	−1.2691
	Clinical controls					20			Porteus-Q errs	0.6021
Wolff, Waber, Bauermeister, Cohen, & Ferber (1982)	Delinquents	Criminal records	15			56			Stroop	0.6172
	Normal controls				0	48	101.9			
					0	48	109.9		TMT-A	0.8527
Yeudall, Fromm-Auch, & Davies (1982)	Delinquent	Criminal records	14.8		35	99	95.04		TMT-B	1.3468
	Normal controls		14.5		18	46	118.98		Category test	0.6661
									Word Fluency	0.7113

[a]Groups were combined into one antisocial group for the analyses.
[b]Groups were combined into one comparison group for the analyses.
WCST-Cat: Wisconsin Card Sorting Test-Categories; WCST-Per: Wisconsin Card Sorting Test-Perservative Errors; Porteus-Q: Porteus Mazes Qualitative; CD: conduct disorder; *DSM-III*: *Diagnostic and Statistical Manual of Metnal Disorders*, third edition; TMT-A: Trail Making Test-Form A; TMT-B: Trail Making Test-Form B; Porteus TA: Porteus Mazes mental test age; ASPD: antisocial personality disorder; PCL-R: Psychopathology Checklist-Revised; CPI-So: California Psychological Inventory-Socialization scale; ADD: attention deficit disorder; MMPI: Million Multiphasic Personality Inventory.

(.33). The mean unweighted effect size for categories achieved on the WCST was .39 standard deviations difference between groups, and the mean weighted effect size was .37 standard deviation difference. Similar to the results for Trails A, this weighted effect size was significantly different from zero ($z = 3.23$, $p < .001$). Finally, contrary to the previous two tests (i.e., Trails A, categories achieved on the WCST), the mean unweighted effect size for the Porteus TA was .08 standard deviations. The mean weighted effect size was .02 standard deviations for both groups, which was not significantly different from zero ($z = .162$). This figure is in marked contrast to the weighted effect size for the Porteus Q score, which is in the large range (see Table 2).

The File Drawer Problem

A comparison of the results of published ($n = 33$) versus unpublished ($n = 5$) studies revealed no significant difference in effect size magnitude, $F(1, 37) = 2.69$ ($p = .11$). This analysis suggests a slight, but nonsignificant, tendency for published studies to yield larger effect sizes than unpublished studies. The number of studies with null results that would be needed to bring the grand mean effect size down to a value at or below .2 (i.e., a small effect size; Cohen, 1988), which was calculated using the fail-safe *N* statistic, was 72.

Analysis of Other Potential Moderators

Both within and between group analyses yielded no significant correlations between effect sizes and age, sex, ethnicity, or IQ. Thus, the magnitude of effects for EF measures could not be attributed to individual differences in any of these four variables.

DISCUSSION

The results of this meta-analysis indicate that there is a robust and statistically significant relation between ASB and EF deficits. There was an average weighted mean effect of .62 standard deviation difference between groups with ASB and comparison groups, which is medium to large in magnitude (Cohen, 1988). Nevertheless, because the sample of effect sizes was significantly heterogeneous, this mean effect size is not an adequate description of the data. The effect sizes were largest for the Porteus Mazes Q score, although the mean effect sizes for all other EF tests were positive and significant.

The large effect size for the Porteus Mazes can be interpreted in at least two ways. First, because the Porteus Mazes is a motor task, it could be argued that the principal difference between ASB and non-ASB groups is evident on tasks that assess motoric control and inhibition. This interpretation is consistent with findings that individuals with ASB perform more poorly on the Motor Scale of the LNNB than individuals without ASB (Kelly, 1982; Rogers, 1983; Voorhees, 1987). On the other hand, it should be noted that the Porteus TA score did not produce a significant difference between ASB and comparison groups, suggesting that the relation between the Porteus Q score and ASB may be attributable to more than this measure's motor component. Second, in contrast to most other measures of EF examined in this meta-analysis, almost all of the studies that used the Porteus Mazes compared ASB groups with normals rather than with clinical comparison groups. As a consequence, the larger effect size for the Por-

teus Mazes compared with other EF measures might be at least partly attributable to confounding variables that differ between ASB and normal comparison groups (e.g., generalized severity of psychopathology, effects of institutionalization, intake of psychotropic medications).

Although we limited our meta-analysis to neuropsychological measures that were reasonably well-validated markers of EF, questions have been raised concerning the specificity of several of these measures to frontal lobe damage. For example, in contrast to several previous investigators, Reitan and Wolfson (1995) reported that the Category Test and Part B of the TMT failed to distinguish patients with frontal lesions from patients with lesions in other cerebral areas. Further research using other and perhaps better validated tests of EF, such as the tower tests (e.g., Tower of Hanoi, Tower of London; see Lezak, 1995), will be necessary to ascertain the robustness of the association between ASB and executive deficits.

This meta-analysis yielded inconsistent findings regarding the specificity of ASB to EF deficits per se as opposed to generalized neuropsychological deficits. On two of three tests that are conjectured to be non-EF tests, there was a moderate difference between ASB and comparison groups. This association could have arisen because EF may overlap with a general factor, such as intelligence, that influences performance on many complex neuropsychological tasks. Nevertheless, it should be noted that IQ did not correlate significantly with effect size estimates either within or between groups. Moreover, the non-EF test that did not differ between ASB and comparison groups was the Porteus TA score, which correlates significantly with IQ (Riddle & Roberts, 1977). The present findings leave unresolved, however, the question of whether individuals with ASB are characterized by neuropsychological deficits in domains other than EF. Further research on the specificity of the neuropsychological deficits of ASB individuals should become a major focus among researchers in this area.

Conversely, it should be noted that EF deficits are not specific to ASB. For example, individuals with both schizophrenia and schizophrenia spectrum disorders exhibit hy-

-0	6	6				
-0	4					
-0	3					
-0	0	0	1			
0	0	0	1			
0	2	2	2	2	2	2
0	4	4	4			
0	6	6	6	7	7	
0	8	8	8	9	9	
1	0	0	0	1		
1	2					
1	4	4	4			
1	9	9				

FIGURE 1. Stem-and-leaf plot of combined effect sizes ($\bar{d}$) for each study.

TABLE 2. Mean Effect Sizes and Tests of Homogeneity for All Studies, Grouped by the Operationalizations of Antisocial Behavior and Measures of Executive and Nonexecutive Function

Operationalization	d	d_+	Q	df	ΔQ	Δdf
ASPD	.10	.08***	.43	1	302.78***	37
CD	.40	.36***	2.28	3	300.93***	35
Psychopathy	.29	.25***	52.68***	14	250.53***	24
All clinical syndromes	.27	.22***	62.56***	19	240.65***	19
Criminality	1.09	.94***	80.82***	2	222.39***	36
Delinquency	.86	.78***	119.03***	12	184.18***	26
All judicial status	.91	.81***	202.14***	15	101.07***	23
EF measure						
Category Tests	.24	.37***	37.70***	8	265.51***	30
Mazes	.80	.74***	254.07***	18	49.14***	20
Stroop Test	.35	.43***	12.78***	5	290.43***	33
Trails B	.40	.33***	46.45***	12	256.76***	26
WCST-Perseverative Errors	.28	.24***	19.82*	10	283.39***	28
Word Fluency	.26	.33***	44.45***	8	258.76***	30
Non-EF measure						
Porteus TA scores	.08	.02	10.40	2	292.81***	36
Trails A	.39	.34***	46.49***	13	256.72***	25
WCST-Categories Achieved	.39	.37***	9.11	6	294.10***	32

ASPD: antisocial personality disorder; CD: conduct disorder; EF: executive function; TA: Test Age score from Porteus Mazes; Trails A: Trail Making Test, Part A; Trails B: Trail Making Test, Part B; WCST: Wisconsin Card Sorting Test.
*$p < .05$.
***$p < .001$.

pofrontality on neuropsychological indices, such as the perseverative errors score on the WCST and on functional brain imaging techniques, such as PET (Williamson, 1987). Another group that appears to exhibit deficits on certain EF tasks, including the perseverative errors score on the WCST, are individuals with obsessive-compulsive disorder (OCD) (Gibbs, 1996). This finding presents a curious paradox, because individuals with OCD have been to exhibit *hyper*frontality on PET scan studies (Baxter et al., 1992), and because OCD and psychopathy are posited by Gray (1982) to lie on opposite ends of a single psychobiological and personality dimension, viz., the behavioral inhibition system. Consequently, the specificity of EF deficits to forms of psychopathology other than ASB merits examination.

It should also be borne in mind that our findings do not conclusively demonstrate that individuals with ASB possess either structural or functional frontal lobe dysfunction. Performance on neuropsychological measures is multiply determined, and may reflect personality variables associated with ASB (e.g., impulsivity, motivational deficits) rather than neuropsychological deficits (Lilienfeld, 1992). Nevertheless, because some of these personality variables may themselves be subserved by the frontal lobes, this distinction between personality and neuropsychological variables may be somewhat artificial.

We found that operationalization was a significant moderator of the association between ASB and EF, and that effect sizes were greater for indices of criminality and de-

linquency than for indices of ASPD, CD, and psychopathy. Because studies of criminality and delinquency were more likely than studies of ASPD, CD, and psychopathy to use normal (rather than psychiatric or inmate) comparison participants, however, this difference may reflect the composition of the comparison groups rather than operationalization per se. Our results suggest a positive association between EF measures and CD, although, as noted earlier, the specificity of this finding to executive dysfunction as opposed to generalized neuropsychological deficits requires clarification. This finding runs counter to that of Pennington and Ozonoff (1996), who found no relation between EF tests and CD. It may be relevant, however, that Pennington and Ozonoff (1996) did not limit their analysis to well-validated tests of EF. For example, they included tests (e.g., SMMT and Necker Cube reversals) for which the theoretical and empirical links to EF are unclear (see Hare, 1984). The inclusion of measures with low validity could have obscured any genuine association between EF and CD.

Although age, sex, ethnicity, and intelligence were analyzed to determine if they influenced the relation between ASB and EF, none significantly moderated this association. Nevertheless, these results may be attenuated because many studies used comparison groups that were matched on these variables. In addition, these findings were based on a relatively small number of effect sizes. Two potential moderators that could not be examined in this meta-analysis are substance abuse/dependence and attention-deficit/hyperactivity disorder (ADHD). Fewer than 25% of the studies assessed participants' substance abuse/dependence history, and only one study (Moffitt & Henry, 1989) assessed the presence of ADHD. These omissions are problematic because substance abuse and ADHD are significantly correlated with ASB (Foley, Carlton, & Howell, 1996; Lilienfeld & Waldman, 1990). Moreover, ASPD is associated with alcohol abuse and dependence (Lewis, 1984). In addition, both ADHD and substance abuse are associated with frontal lobe dysfunction and other neurological deficits (Barkley, 1997; Pennington & Ozonoff, 1996). These factors might represent important unexamined confounds in this meta-analysis. Indeed, Hare (1984) has suggested that some positive findings regarding the presence of EF deficits among psychopaths (e.g., Gorenstein, 1982) are attributable to the high rates of substance abuse in this group. Thus, research needs to be directed toward differentiating the effects of substance abuse and ADHD from the association between ASB and EF.

Two final limitations of this meta-analysis should be noted. First, several of the measures of psychopathy examined may not adequately assess the core personality features of this syndrome as delineated by Cleckley (1941/1982). In particular, the MMPI Pd scale and CPI So scale correlate moderately with indices of generalized ASB, but only weakly or negligibly with the principal personality traits of psychopathy, such as guiltlessness and egocentricity (Harpur et al., 1989; Lilienfeld, 1994). In contrast, the PCL and PCL-R are based largely on the Cleckley conceptualization of psychopathy and have demonstrated excellent construct validity as measures of psychopathy (Hare, 1991). Nevertheless, because the number of studies examining the relation between psychopathy and EF was small, it was not possible to conduct separate analyses of this association by each psychopathy measure. Further research on EFs using the PCL, PCL-R, and other well-validated measures of Cleckley psychopathy is clearly warranted.

Second, as noted earlier, we were unable to subdivide EF measures in terms of their associations with different brain regions (e.g., dorsolateral, orbitomedial) because of the lack of knowledge concerning the neuroanatomical substrates of most EF tasks. Because several empirical and theoretical models posit that psychopathy and perhaps

other forms of ASB are associated primarily with damage to the orbitomedial area (Blumer & Benson, 1975; Damasio et al., 1990) rather than to the entire prefrontal cortex, the overall effect size reported here may underestimate the relation between orbitomedial dysfunction and ASB. Block (1994) argued that the best way to establish the relative health of a group of individuals with an inferred deficit on a diffuse indication of brain functioning (e.g., neuropsychological measures of EF) is to use anatomical or imaging techniques. Thus, to overcome the problem of inferred dysfunction in both clinical and nonclinical participants, tests of EF should be used in conjunction with such neuroimaging techniques as PET and functional magnetic resonance imaging (fMRI). Research using PET and fMRI is already taking place using normal participants (Rezai et al., 1993), but such work with antisocial groups has only recently been initiated (Hare, 1996; Raine et al., 1994; Raine, 1997).

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