NEUROPSYCHOLOGICAL MEASURES OF EXECUTIVE FUNCTION AND ANTISOCIAL BEHAVIOR: A META-ANALYSIS

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ABSTRACT

A meta-analysis was performed to quantify the association between antisocial behavior (ASB) and performance on neuropsychological executive functioning (EF) measures. The meta-analysis built on Morgan and Lilienfeld's (2000) meta-analysis of the same topic by including recently published studies and by examining a wider range of EF measures. A total of 126 studies involving 14,786 participants were included in analyses. Antisocial groups performed significantly worse on measures of EF compared to controls, with a grand mean effect size of $d = 0.44$. There was significant variation in the magnitude of effect sizes calculated across studies. The largest effect sizes were found for criminality ($d = 0.62$) and externalizing behavior disorder ($d = 0.54$) ASB groups, while the smallest effect sizes were found for antisocial personality disorder ($d = 0.19$). Larger differences in EF performance were observed across studies involving participants from correctional settings and with comorbid attention-deficit and hyperactivity problems. Overall, results indicated that there was a robust association between ASB and poor EF that held across studies with varied methodological approaches. Methodological issues in the research literature and implications of the meta-analysis results are discussed and directions for future research are proposed.

Keywords: antisocial behavior, executive function, meta-analysis

Running Head: Executive function and antisocial behavior

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Recent Publications:

Allard, T., Stewart, A., Chrzanowski, A., **Ogilvie, J.,** Birks, D., & Little, S. (2010). Police diversion of young offenders and Indigenous over-representation. *Trends & Issues, No 390*. Australian Institute of Criminology.

Ogilvie, J., M., & Stewart, A., L. (2010). The integration of rational choice and self-Efficacy theories: A situational analysis of student misconduct. *The Australian and New Zealand Journal of Criminology, 43*(1), 130-155.

Livingston, M., Stewart, A., Allard, T., & **Ogilvie, J.** (2008). Understanding juvenile offending trajectories. *The Australian and New Zealand Journal of Criminology, 41*(3), 345-363.

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Recent Publications:

Thompson, C. M., Dennison, S. M. & **Stewart, A. L**. (in press). Challenging conceptions of relational stalking as a male perpetrated phenomenon *Sex Roles: A Journal of Research*.

Ogilvie, J. M & **Stewart, A. L**. (2010). The integration of rational choice and self-efficacy theories: A situational analysis of student misconduct. *Australian and New Zealand Journal of Criminology*, 43, 130 – 155.

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Recent Publications:

Chan, R. C. K., Di, X., McAlonan, G. M., Gong, Q. (2010). Brain anatomical abnormalities in high risk individuals, first-episode and chronic schizophrenia: an activation likelihood estimation meta-analysis of illness progress. *Schizophrenia Bulletin, 37*(1), 177-188.

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Recent Publications:

Shum, D., Fleming, J., Gill, H., Gullo, M., Strong, J. (2011). A randomised controlled trial of prospective memory rehabilitation in adults with traumatic brain injury. *Journal of Rehabilitation Medicine.* DOI**:**10.2340/16501977-0647

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INTRODUCTION

The discipline of social neuroscience is emerging as an important research perspective when studying risk factors for the development of antisocial behavior (ASB). This research focuses on delineating the neural mechanisms associated with the cognitive and affective processes that regulate social behavior (Raine and Yang 2006). There is a growing body of research on risk factors associated with the development of ASB that recognizes the role of neuropsychological factors in the onset, persistence and desistance of ASB over the developmental lifespan (Moffitt 1990, 2006; Raine et al. 2005; Seguin 2004, 2008). This body of research has a crucial role in informing theoretical accounts of the development of ASB, and treatment and prevention interventions. It has been argued that neuropsychological impairments may be a key mechanism mediating the effects of genetic and psychosocial influences on ASB (Friedman et al. 2008; Raine and Yang 2006; Yang, Glenn, and Raine 2008). Impairments in the neuropsychological processes of executive functioning (EF), which include a collection of cognitive functions necessary for self-regulation and the regulation of socially appropriate behavior, have received considerable research attention in relation to ASB. EF impairments are hypothesized to increase the risk of engaging in ASB through decreasing behavioral inhibition, impairing the ability to anticipate behavioral consequences and assess punishment and reward, damaging the capability to generate socially appropriate behavior in challenging contexts (Giancola 1995; Ishikawa and Raine 2003; Seguin 2008). Impairments in EF have consistently been linked to various operationalisations of ASB, including criminality, delinquency, physical aggression, conduct disorder, psychopathy and antisocial personality disorder (Morgan and Lilienfeld 2000). However, there is inconsistency across studies about the nature of EF processes in various forms of antisocial behavior, primarily resulting

from methodological differences in the conceptualization and measurement of ASB and EF.

The aim of this paper is to summarize findings across studies on the association between EF and ASB using meta-analytic methods. This was completed in an attempt to reconcile inconsistencies across studies, identify methodological issues that may impact on findings and assist in specifying the nature of EF impairments that are associated with various conceptualizations of ASB. To provide a context for the present systematic review, a number of conceptual and practical issues will be highlighted. First, the neuropsychological construct of EF will be described, including issues related to the measurement of the construct. Second, the operationalisation of ASB will be explored, and conceptual and empirical knowledge of the relation between ASB and EF will be summarized.

CONCEPTUAL AND PRACTICAL ISSUES

EXECUTIVE FUNCTION

EF is an umbrella term encompassing a diverse range of cognitive processes and behavioral competencies to facilitate the initiation, planning, regulation, sequencing and achievement of complex goal-oriented behavior and thought (Royall et al. 2002; Shallice 1988; Stuss and Benson 1986; Stuss et al. 2002). EF abilities are often conceptualised as higher level cognitive processes that regulate lower level cognitive process in the performance of complex tasks (Friedman et al. 2008; Miyake et al. 2000). No overarching or widely accepted conceptual framework of EF has been developed and there continues to be disagreement about the processes thought to be involved in EF (Burgess 1997; Jurado and Rosselli 2007; Miyake et al. 2000; Royall et al. 2002; Salthouse 2005; Stuss and Knight 2002).

EF is best understood as a collection of multifaceted, related but separate set of cognitive abilities that are subserved by numerous neurological systems distributed throughout the brain (Collette et al. 2006; Collette and Van der Linden 2002). The concepts of EF and frontal lobe functioning have traditionally been closely related, although contemporary evidence indicates that these cognitive/behavioral and anatomical concepts are dissociable (Robbins 1998). While patients with frontal lobe dysfunction most commonly exhibit EF impairments, it must be noted that EF impairments are also evident among patients with damage to other brain regions. The frontal cortex, particularly the prefrontal cortex (PFC), plays a central role in mediating EF processes, although efforts to localize EF processes to discrete frontal areas have produced equivocal results (Ardila 2008; Collette et al. 2005; Duncan and Owen 2000; Stuss and Knight 2002; Tanji and Hoshi 2008). Current evidence indicates that optimal performance on EF tasks depends on the integrity of the whole brain (Collette et al. 2005; Funahashi 2001; Prabhakaran et al. 2000; Stuss and Alexander 2000).

Impairments in EF have been implicated in a range of developmental disorders, including Attention-Deficit/Hyperactivity Disorder (ADHD), Conduct Disorder (CD), Autism, and Tourette Syndrome (Pennington and Ozonoff 1996). EF impairments have also been implicated in a range of neuropsychiatric and medical disorders, including schizophrenia, major depression, alcoholism, structural brain disease, diabetes mellitus and normal aging (Royall et al. 2002). Recent evidence suggests that the level of general psychopathology rather than specific psychiatric diagnoses is more strongly associated with EF impairments (Stordal et al. 2005). It is probable that different disorders have distinct levels and/or profiles of specific EF

impairments. The challenge is for research to identify such specificity in EF impairments within and between disorders.

MEASUREMENT OF EXECUTIVE FUNCTION

There is no 'gold standard' of EF measurement against which to compare measures of the construct (Royall et al. 2002). Traditionally, the measurement of EF has used tasks purported to rely on the functions of the frontal lobe, with the validity of such tasks assessed on their sensitivity to frontal damage. As a consequence, the exact nature of EF abilities necessary for successful performance on these traditional measures is not fully specified (Miyake et al. 2000). Many measures of EF have uncertain validity since they involve complex, demanding and multi-faceted tasks that draw on both executive and non-executive processes (Chan et al. 2008). Multiple executive processes may be elicited by a single complex task, and single executive processes may be utilized across multiple tasks. As a result, it is difficult to isolate specific cognitive deficits from the results of EF measures (Anderson 2002). Performance on EF measures is likely to represent the pooled effect of several distinct EF processes, resulting in a significant level of `task impurity' for many EF tasks (Hughes and Graham 2002). The task impurity problem refers to the issue that EFs by definition are believed to operate on other cognitive processes, whereby any executive task will implicate both EFs and other cognitive processes not relevant to the target EF, producing difficulties in accurately measuring executive processes (Burgess 1997).

EF measures are generally designed to capture clinically significant performance in experimental settings (Burgess et al. 2006; Chan et al. 2008; Chaytor, Schmitter-Edgecombe, and Burr 2006). The demands placed on EF capacities in real-

life settings are complex, multifaceted and involve multiple sub-tasks, while experimental EF tasks are commonly de-contextualised and involve relatively simple responses to simple tasks. Individuals who do not display impairment on EF tasks in experimental settings may still encounter difficulties in everyday tasks that require executive control. This issue is relevant to the study of EF impairments in antisocial individuals. Deficits in EF experienced by a large proportion of antisocial individuals are likely to be sub-clinical and representative of individual differences rather than pathology in EF abilities. These individual differences in EF abilities associated with ASB may produce subtle impairments that impact on the regulation of everyday behavior. However, this is not to discount the existence of EF pathology in specific subgroups of antisocial individuals, including serious and persistent antisocial individuals who initiate offending at a young age (Moffitt 1993).

These measurement issues have likely contributed to the inconsistencies in findings across studies regarding the nature of EF impairments among antisocial individuals. EF processes are most commonly conceptualised as a broad range of cognitive abilities and assessed by a limited range of tests. Consequently, EFs are best assessed through the use of a battery of measures, since it is unlikely that a single measure will assess all components of EF. Examples of EF test batteries include the Behavioural Assessment of the Dysexecutive Syndrome (BADS; Wilson et al. 1996), the Cambridge Neuropsychological Test Automated Battery (CANTAB; Robbins et al. 1998), and the Delis-Kaplan Executive Function System (D-KEFS Delis, Kaplan, and Kramer 2001). The use of EF test batteries is rare, although there is a growing recognition of the need to their use (for example, see Broomhall 2005; Cauffman, Steinberg, and Piquero 2005).

ANTISOCIAL BEHAVIOR

Antisocial behavior is a complex construct that cannot be clearly conceptualised under a single theoretical framework, as it encompasses a diverse range of socially disapproved behaviors (Rutter 2003). Antisocial behavior may be broadly operationalised according to three major categories: clinical psychiatric diagnoses, the violation of legal or social norms and aggressive or violent behavior. Clinical diagnostic categories most frequently associated with ASB are CD, Oppositional Defiant Disorder (ODD), Antisocial Personality Disorder (ASPD) and psychopathy.

CD is diagnosed as a pattern of persistent behavior characterized by the violation of the rights of others or major age-appropriate norms and is usually diagnosed after the age of 9 years but not after 18 years (American Psychiatric Association 2000). Examples of these behaviors include aggression, property destruction and theft. ODD is a diagnosis associated with persistent patterns of negativistic, hostile, defiant, provocative, and disruptive behavior and is usually diagnosed after 9 years but not after 18 years (American Psychiatric Association 2000). ASPD is a diagnosis associated with a persistent pattern of behavior characterized by a disregard and violation of the rights of others. ASPD requires a diagnosis of CD before age 15 years and cannot be diagnosed before the age of 18 years (American Psychiatric Association 2000). Psychopathy is characterized by a lack of empathy or insight for the effect of one's behavior on others, callous, shallow and superficial traits, and behavioral characteristics including impulsiveness and poor behavioral control (Hare 1996). Although these disorders often involve engagement in deviant or criminal behavior, they are not synonymous with crime (Rutter, Giller, and

Hagell 1998). Studies operationalising ASB by the clinical syndromes of CD, ODD, ASPD and psychopathy will be included the current meta-analysis.

Legal operationalisations of ASB include criminality and delinquency, and relate to the violation of legal or social norms, and the commission of criminal acts as a juvenile. These operationalisations are most commonly measured by official records and/or self-reports of criminal activity. Studies using these legal operationalisations will also be included in the current meta-analysis.

The ASB operationalisation of physical aggression or violent behavior most commonly refers to engagement in behavioral aggression directed towards others, including bullying, initiating physical fights, using a weapon and causing serious physical harm. Studies examining physical aggression or violence will be included in the study.

These three categories of ASB (clinical, legal, and aggression) overlap to a significant degree. For example, ASPD criteria include the presence of criminality and CD, and a diagnosis of CD requires the criteria of aggression and delinquency. Furthermore, antisocial clinical syndromes are highly prevalent among incarcerated offenders (Abram et al. 2003; Fazel and Lubbe 2005). However, these operationalisations are not entirely synonymous. For example, not all youth diagnosed with CD will be later diagnosed with ASPD. It is reasonable to assume that these operationalisations overlap to a moderate degree, although they may differ subtly in terms of etiological origins.

ANTISOCIAL BEHAVIOR AND EXECUTIVE FUNCTION

EFs are believed to be central abilities necessary for self-regulation, including the regulation of emotion and socially appropriate adult conduct. Impairments in EF

often result in socially inappropriate behavior, an inability to plan and problem solve, distractibility, aggressiveness, impulsive behavior, poor judgment of behavioral consequences and poor memory (Fuster 2000; Mesulam 2002). The similarity of EF impairments to features of ASB suggests that EF processes are important in the etiology of ASB. However, it must be noted that current evidence linking ASB and EF does not clearly support the conclusion that EF underlies ASB in a causal manner. The observation of EF impairments among antisocial individuals does not explain how such impairments develop over time and may lead to ASB.

Morgan and Lilienfeld (2000) conducted a meta-analysis to quantify the association between ASB and EF. Results of the study indicated that there was a robust association between ASB and EF that held across varying study methodologies. This meta-analytic review remains as the only systematic quantitative review of studies examining the relationship between ASB and EF, with narrative reviews being more common (e.g., Brower and Price 2001; Hawkins and Trobst 2000; Ishikawa and Raine 2003; Seguin 2008; Teichner and Golden 2000). Morgan and Lilienfeld (2000) examined a total of 39 studies including 4,589 participants. To be included in the meta-analysis, a study must have employed at least one of six measures of EF with demonstrated sensitivity to frontal damage: the Category Test of the Halstead-Reitan Neuropsychological Battery, the Qualitative score on the Porteus Mazes Test, the Stroop Interference Test, the Wisconsin Card Sorting Test (WSCT) and verbal fluency tests. Additionally, the studies must have grouped individuals according to ASB and comparison groups. Individuals were classified in the groups of psychopathic personalities, individuals with either ASPD or CD, criminals, delinquents and psychiatric comparison participants or normal comparison participants.

Results of the meta-analysis indicated that the grand mean weighted effect size for all studies was 0.62 standard deviations difference between antisocial and comparison groups on all EF measures, with 79% of all study effect sizes being positive. These results indicated that antisocial individuals performed significantly worse on EF measures compared to comparison groups. The effect sizes were, however, heterogeneous across the studies, indicating that the grand mean effect size was not derived from a single population of studies. Effect sizes were found to vary according to the type of ASB, with the largest effects found for criminality (d = 1.09) and delinquency (d = 0.86), and small to medium effects found for CD (d = 0.40) and psychopathy (d = 0.29).

Effect sizes were also found to vary according to EF measures, with the largest effect found for the Porteus Mazes Q score (d = 0.80) and all other EF measures having effect sizes in the small to medium range. These results highlighted the need to examine EF impairments across differing groups of antisocial individuals using varied measures of EF. However, results further indicated that antisocial individuals were not specifically impaired in EF, as antisocial individuals were also found to have deficits on non-EF tests, including Trails A (d = 0.39) and categories achieved on the WCST (d = 0.39). However, the status of these measures as non-EF tests is questionable given that they may also tap EF processes.

The Morgan and Lilienfeld meta-analysis provided a valuable summary of the research base, indicating that there is a robust association between EF and ASB that holds across varying study methodologies. This methodological variation across studies may be viewed as an asset. Variability in how both EF and ASB are conceptualised and measured provides an opportunity to examine the robustness of

the relationship by examining how the relationship may vary across different ASB groups and EF measures.

Morgan and Lilienfeld (2000) argued that further research is needed to examine the specificity of EF impairments among antisocial individuals and resolve inconsistencies in findings across studies. Studies vary in the types and severity of EF impairments observed among antisocial participants, which suggests that EF impairments may be more important in the expression of particular antisocial syndromes. For example, there is inconsistency in the level and types of EF problems observed in psychopathic samples. While studies performed by Dolan and colleagues (Dolan and Anderson 2002; Dolan et al. 2002) indicate that psychopathic individuals perform poorly on a range of EF tests, other studies indicate that psychopathic individuals display minimal impairments in EF (e.g., Dvorak-Bertsch et al. 2007; Smith, Arnett, and Newman 1992). Such discrepancies in findings appear to relate to sampling differences, including the use of antisocial versus healthy comparison groups and how psychopathic individuals are categorized (e.g., high versus low anxious).

Since the publication of Morgan and Lilienfeld's meta-analysis, a number of studies have examined how EF impairments may be more prominent in particular groups and subgroups of antisocial individuals (e.g., psychopathy; Ishikawa et al. 2001; Pham et al. 2003) and particular forms of ASB (e.g., physical aggression; Seguin et al. 2004). Specifically, EF impairments appear to be more pronounced in groups characterized by severe and persistent behavioral problems. For example, Clark, Prior and Kinsella (2000) found EF impairments to be most pronounced in children with comorbid externalizing behavior disorders and ADHD compared to children with non-comorbid externalizing behavior problems. Additionally, Raine et

al. (2005) and Piquero (2001) found that individuals following lifecourse persistent pathways of antisocial behavior displayed greater impairments in EF compared to less severe antisocial comparison groups. Unfortunately, studies in large part have not been specific in the groups and subgroups of antisocial individuals that are included in analyses and how EF impairments may differ among these groups. Global categories of ASB may potentially conceal subgroups of antisocial individuals and the causal mechanisms associated with the development of specific ASB groups (Barker et al. 2007).

It is possible that the inconsistent findings are indicative of the heterogeneity of both EFs and antisocial individuals as a population, and also variation in how ASB is operationalised, the characteristics of samples, control groups and assessment measures employed (Raine and Scerbo 1991). There are inconsistencies across studies in the examination of factors that may impact on the association between ASB and EF, including the age of participants, the presence of ADHD symptoms, intelligence, substance misuse, and gender differences. It is important for studies to control for such factors when examining the association between ASB and EF, although efforts to do so have been inconsistent. For example, there is evidence to suggest that EF impairments are associated with both substance use disorders (Giancola, Shoal, and Mezzich 2001) and ADHD (Willcutt et al. 2005), which are both highly prevalent among antisocial individuals (Jacobson et al. 2008; Van Goozen et al. 2007). However, few studies have explicitly controlled or examined the mediating or moderating effects these factors may have on the association between ASB and EF.

GOALS OF THE PRESENT REVIEW

The primary aim of this study was to quantify the association between ASB and EF in an effort to summarize the state of the current research literature. Meta-analytic methods are used to expand on and address the limitations of the earlier meta-analysis performed by Morgan and Lilienfeld. Studies published up to September 2010 that used a wide range of EF measures were examined to summarize the findings of studies and characterize advancements and continuing methodological issues in the research field. Studies included in Morgan and Lilienfeld's meta-analysis were included to provide a more robust estimate of the association between EF and ASB, as well as increase the statistical power of analyses. The inclusion of a wider range of more contemporary EF measures improves on the earlier meta-analysis by not solely relying on measures validated by their sensitivity to frontal damage, since frontal functioning is not synonymous with EF.

Specificity in EF impairments was explored across groups of ASB and measures of EF. The ASB group of physical aggression and/or violence was added to aid in the identification of specificity in EF impairments. This operationalisation was not included in the earlier meta-analysis due in part to the lack of attention in the earlier research literature to issues of specificity. Similar to the earlier meta-analysis, a number of possible moderating variables were examined to assess their effects on the links between ASB and EF, including age, gender, correctional recruitment, and comorbid ADHD. This analysis expands on the earlier study by including age and ADHD as potential moderators of effect sizes.

METHOD

SEARCH STRATEGY AND INCLUSION CRITERIA

All studies included in the original meta-analysis were included in the present analysis. In addition, three search strategies were employed to identify subsequently published and unpublished studies. First, nine computerized databases were searched: Web of Science ISI, Scopus, Google Scholar, PsycINFO, MEDLINE, PubMed, ERIC, Cambridge Scientific Abstracts, and Dissertation Abstracts International. The keywords used to search the databases were relevant to ASB and EF: “antisocial”, “antisocial personality disorder”, “psychopathy”, “delinquency”, “criminal”, “conduct disorder”, “oppositional defiant disorder”, “externalizing disorder”, “aggression”, “violence”, “sex offender”, “executive function”, “executive control”, “cognitive control”, “frontal function”, “frontal lobe”, “working memory”, “attention”, “attentional control”, “impulsivity”, “inhibition”, “neuropsychological”, and “neurocognitive”. Second, the reference lists of published studies collected and narrative reviews of the topic (viz., Blair 2005; Brower and Price 2001; Golden et al. 1996; Hawkins and Trobst 2000; Moffitt 1990; Raine and Yang 2006; Seguin 2008; Teichner and Golden 2000) were scanned to locate further studies not found in the database searches. Third, five authors in the research area (viz., R.J.R Blair, P.R. Giancola, S.O. Lilienfeld, T.E. Moffitt, and J.R. Seguin) were contacted to request additional published and unpublished research that had either been overlooked using the previous search strategies or had not been published.

Studies were required to satisfy the following criteria to be included in the meta-analysis:

1. The independent variable of ASB included one or more of the following groups: incarcerated offenders, delinquents, expression of physical aggression

and/or violence, psychopathic personalities, individuals with Conduct Disorder (CD) and/or Oppositional Defiant Disorder (ODD), Antisocial Personality Disorder (ASPD), and psychiatric/institutionalized comparison groups, or normal comparison groups.

2. The neuropsychological functioning of study groups was assessed using test instruments purported to measure executive functioning abilities, as determined by consulting major neuropsychological assessment texts and resources (see below).
3. Studies including individuals comorbid with ADHD and ASB without separating groups by ADHD and ASB were excluded given that this is a disorder strongly associated with EF impairments (Willcutt et al. 2005). It is recognized this exclusion is dependent on whether the ADHD was assessed and reported in studies. It is likely that a high proportion of antisocial participants would meet criteria for ADHD. To examine the moderating effects of ADHD on ASB EF impairments, studies that separated groups by ASB and ASB comorbid with ADHD were included in subsidiary analyses.
4. The results presented were sufficient to calculate effect sizes (i.e., means and standard deviations, *t*-values, *F*-values, *p*-values, or *r*-values). When such information was not present, authors were contacted to obtain data.

ANTISOCIAL BEHAVIOR OPERATIONALIZATIONS

Study samples were grouped according to six ASB groups: 1) externalizing behavior disorders (CD/ODD), 2) physical aggression and/or violence, 3) delinquency, 4) criminality, 5) ASPD; and 6) psychopathy. The categories of CD and ODD were combined to represent antisocial/disruptive behavior disorders first

diagnosed in childhood. There is a high degree of overlap between CD and ODD, where ODD is a possible developmental precursor to CD and a large proportion of children diagnosed with CD will often meet criteria for ODD (Maughan et al. 2004). It is acknowledged that combining CD and ODD may mask potentially meaningful subgroups of antisocial individuals. However, studies often do not report additional information regarding specificity in externalizing behavior problems. The categories were combined in the present meta-analysis to retain statistical power in analyses. Studies were only included in the categories of disruptive behavior disorder (CD/ODD) and ASPD if clinical criteria using the Diagnostic and Statistical Manual of Mental Disorders (DSM-III, DSM-IV; American Psychiatric Association 1987, 2000) or the International Statistical Classification of Diseases and Related Health Problems (ICD-10; World Health Organization 1992) were used to classify participants. Both delinquency and criminality were classified using criminal records or self-reports. Psychopathy was classified using the *Psychopathic Personality Inventory* (PPI; Lilienfeld and Andrews 1996), the *Psychopathy Checklist-Revised* (PCL-R; Hare 1991), or derivatives of this checklist (e.g., Self-Report Psychopathy Scale, version three; Paulhus, Hemphill, and Hare In press). Physical aggression and/or violence groups were classified by collateral behavioral information, including criminal history records, teacher reports, and psychometric results.

It is recognized that classification of ASB is more likely to represent a researcher's orientation rather than a distinct group of antisocial individuals, and that there is significant overlap between categorizations. Overlapping ASB definitions (e.g., criminality and psychopathy) were present for a number of studies. Morgan and Lilienfeld (2000) defined ASB as a mutually exclusive specific group. That is, if a psychiatric diagnosis was applied to a criminal or delinquent group, the participants

were classified according to that psychiatric definition of ASB. However, for the present meta-analysis, an approach was taken whereby ASB categorizations were not mutually exclusive. For example, in a study examining incarcerated psychopaths, effect sizes derived from the study would be included in the analyses for both criminality and psychopathy ASB groups. This approach violates assumptions of independence in the calculation of effect sizes. However, for the purposes of between ASB group comparisons this approach provided a more accurate estimate of effect size magnitude given that there is a high degree of overlap in definitions of ASB (Lipsey and Wilson 2001). The six ASB groups were examined as potential moderators of the relationship between ASB and EF impairments. The classification of each study according to ASB category is listed in the supplemental table.

EXECUTIVE FUNCTION MEASURES

The present study expanded the meta-analysis performed by Morgan and Lilienfeld (2000) and included a wider range of more contemporary EF measures. Instead of only including those measures with demonstrated evidence for specificity to frontal damage (i.e., the criterion used by Morgan & Lilienfeld), measures were included if they were explicitly used to measure cognitive processes relevant to executive functioning. This rationale being that recent evidence indicates that the frontal lobes are only one aspect of an executive system that involves multiple cortical and subcortical structures (Alvarez and Emory 2006; Duffy and Campbell 2001; Robbins 1998; Stuss and Alexander 2000). There is considerable variation across studies in regard to the sensitivity and specificity of executive function measures to frontal lobe damage, even among those measures with the most reliable evidence for specificity to frontal damage (e.g., WCST, Verbal Fluency and the Stroop; Alvarez

and Emory 2006). This suggests that EF measures should not be regarded as purely frontal lobe functioning tests, but as measures that require the coordination of several neural circuits for successful performance (Alvarez and Emory 2006).

EF measures were included in the meta-analysis if at least one study meeting the inclusion criteria used the measure to assess EF processes. The utility of a measure in assessing EF processes was confirmed through reference to major neuropsychological assessment texts and resources (Alvarez and Emory 2006; Chan et al. 2008; Lezak 2004; Rabbitt 1997; Stuss and Knight 2002). To retain a larger sample of studies for analyses, studies that combined EF measures using factor analytic composite scores were included (e.g., Giancola, Mezzich, and Tarter 1998; Giancola, Shoal, and Mezzich 2001).

It must be noted that measures of both working memory and attentional control were included in the present meta-analysis as measures of EF. Working memory is argued to be central to executive control, and includes cognitive processes involved in the manipulation, integration and transformation of information to plan and guide behavior (D'Esposito and Postle 2002; Prabhakaran et al. 2000; Shimamura 2002; Wagner and Smith 2003). Attentional control has also been conceptualised as a central component of executive control through its role in the inhibition of task irrelevant information processing and switching between competing tasks, as well as being a major component of working memory capacity (Kane et al. 2001; Rossi et al. 2009).

DATA COLLECTION AND PREPARATION

One hundred and twenty six studies met inclusion criteria for the meta-analysis, including the 39 studies identified by Morgan & Lilienfeld and 87 newly

identified studies up to September 2010. Study details are provided in the supplemental table. Fifty studies employed more than one comparison group. Extreme group contrasts were used in such cases to simplify the calculation of effect sizes, where all effect sizes were derived from one group comparison per study. The extreme group method involved deriving effect sizes from the two groups that represented the extremes of the study participants. For example, if a study included low, medium and high psychopathy participants, the low and high group scores would be used to calculate effect sizes. This methodology may inflate effect sizes. Some studies with multiple comparison groups used clinical comparison groups based on psychopathological disorders (e.g., ADHD in Oosterlaan, Scheres, and Sergeant 2005). These comparison groups were not used to calculate effect sizes. The supplemental table lists the comparison groups within each study included in the meta-analysis, and highlights the group comparisons that effect sizes were derived from. One study (Herba et al. 2006) reported data separately by gender. In this case, the study was coded once for each gender separately. Three studies (Dvorak-Bertsch et al. 2007; Smith, Arnett, and Newman 1992; Vitale et al. 2007) divided psychopathic groups according to levels of anxiety. For these studies, effect sizes were calculated within levels of anxiety. Several studies included participants exhibiting comorbid ADHD and ASB characteristics (e.g., Albrecht et al. 2005; Schachar et al. 2000). Effect sizes for EF were calculated for these participant groups using the control groups in each study for comparisons.

EFFECT SIZE PROTOCOL

The following approach was adopted to calculate effect sizes:

1. Calculation of individual effect sizes (*d*) and corresponding variances for each EF measure in each study
2. Calculation of weighted mean effect size for each study
3. Calculation of weighted mean effect sizes for each EF measure across studies
4. Calculation of weighted mean effect sizes for ASB groups across studies
5. Calculation of 95% confidence intervals (CIs) surrounding weighted effect sizes
6. Calculation of *Q* and I^2 statistics to assess heterogeneity of effect sizes by EF measures, ASB groups and studies.

Cohen's *d* (Cohen 1988) standardized mean difference effect sizes using pooled standard deviations were used to determine the magnitude of EF impairments. Zakzanis (2001) proposed that Cohen's *d* is the most appropriate measure for neuropsychological research primarily due to its ability to explicitly account for the variability observed between neuropsychological patients. Impairments in EF by antisocial groups were represented by positive effect sizes. Cohen (1988) defines a small effect size as $d \geq .2$, a moderate effect as $d \geq .5$, and a large effect as $d \geq .8$. Zakzanis (2001) proposed that a Cohen's *d* of 3.0 is an appropriate marker of clinical significance in neuropsychological disorders. All Cohen's *d* statistics are expressed in standard deviation units.

Individual effect sizes were first calculated for every EF measure used by a study. In studies reporting means and standard deviations for EF scores, *d* (Eq. 1) was calculated by subtracting the ASB group mean score (X_1) from the control group mean score (X_2) and dividing the result by the pooled standard deviation (S_{pooled}) (Eq. 2). N_1 is the number of participants in the ASB group, N_2 is the number of participants

in the control group, SD_1 is the standard deviation of the mean score for the ASB group, and SD_2 is the standard deviation of the mean score for the control group.

$$d = \frac{\left(\overline{X}_2 - \overline{X}_1\right)}{S_{pooled}} \quad (1)$$

where

$$S_{pooled} = \sqrt{\frac{\left(N_1 - 1\right)SD_1^2 + \left(N_2 - 1\right)SD_2^2}{\left(N_1 - 1\right) + \left(N_2 - 1\right)}} \quad (2)$$

When means and standard deviations were not reported, *r* values and *t* and *F* statistics were converted to *d* using formulae provided by Zakzanis (2001) and Lipsey and Wilson (2001). All computed effect sizes were corrected for small sample bias (Hedges g) using the formula provided by Hedges (1981) and displayed in Eq. 3. *N* is the total number of participants and d' is the unbiased standardized mean difference.

$$d' = d\left[1 - \frac{3}{4N - 9}\right] \quad (3)$$

The variance for each individual effect size (v_d) was calculated using Eq. 4, with *N* being the sample sizes for each group. The inverse of the sampling variance ($w_i = 1/v_i$) was used to weigh each effect size for the fixed effect model of analysis.

$$v_d = \left[\frac{N_1 + N_2}{N_1 N_2} + \frac{\left(d'\right)^2}{2\left(N_1 + N_2\right)}\right] \quad (4)$$

After calculation of individual effect sizes, three classes of weighted mean effect sizes ($\overline{d}$) were calculated (steps two to four of the effect size protocol) for 1) studies; 2) EF measures; and 3) ASB categorizations. A mean effect size was calculated for each study by averaging all effect sizes and inverse variance weights within the study. Therefore, each study produced an average effect size and an average inverse variance weight. An average inverse variance weight was used for studies, as weights are a function of sample size and highly similar across effect sizes

within a study. Weighted mean effect sizes for EF measures and ASB categorizations were calculated from the individual effect sizes using the formula provided by Hedges and Olkin (1985). In Eq. 5, k is the number of effect sizes, $w_i = 1/v_i$ (inverse variance weight), and v_i is the variance of the individual effect size.

$$\bar{d} = \left[\frac{\sum_{i-1}^{k} w_i d_i}{\sum_{i-1}^{k} w_i} \right] \quad (5)$$

The variance of the weighted mean effect size was then calculated using Eq. 6, which was then used to calculate 95% confidence intervals for weighted mean effect sizes to aid in the determination of statistical significance (Eq. 7).

$$v_{\bar{d}} = \left[\frac{1}{\sum_{i-1}^{k} w_i} \right] \quad (6)$$

$$95\%CI = \bar{d} \pm 1.96\sqrt{v_{\bar{d}}} \quad (7)$$

Percentage overlap (%OL) scores were calculated using tables provided by Zakzanis (2001) for weighted mean effect sizes to estimate the extent to which scores from antisocial and comparison groups on EF measures overlapped. The %OL score is inversely related to effect size, where an effect size of $d = 0.00$ corresponds to a 100% overlap in scores between the criterion and comparison groups, and an effect size of $d = 4.00$ corresponds to an overlap of 2.3% between the two groups.

Tests of the homogeneity of the three classes of weighted mean effect sizes were performed to determine whether the effect sizes were derived from a single population. When the variation of effect sizes is greater than would be expected from sampling error alone, the distribution of effects sizes is deemed to be heterogeneous

and not derived from a single population (Lipsey and Wilson 2001). The *Q*-statistic was calculated as a homogeneity test (Eq. 8):

$$Q = \sum_{i=1}^{k} w_i \left(d_i - \bar{d} \right)^2 \quad (8)$$

where k is the number of effect sizes, w_i is the inverse variance weight of each individual effect size, d_i is the individual effect size, and $\overline{\mathrm{d}}$ is the weighted mean effect size. If the *Q*-statistic exceeds a critical value associated with a pre-determined alpha level (in the present study, $p < .05$) the sample of effect sizes are characterized as heterogeneous. The *I*-squared statistic (Higgins and Thompson 2002) was also calculated, and is a measure of heterogeneity expressed as a percentage (Eq. 8):

$$I^2 = 100 \left[\frac{Q - df}{Q} \right] \quad (8)$$

where Q is the *Q*-statistic and df is the number of effect size observations minus one. *I*-Squared values of 25%, 50% and 75% represent low, moderate and high levels of heterogeneity respectively. *I*-squared values greater than 50% indicate that variability in a group of effect sizes is large enough to suspect that they were not derived from the same population.

Both fixed- and random-effects models of the weighted mean effect sizes were estimated to analyze potential heterogeneity in effect size distributions. Fixed effect models assume that random error in effect size estimates results only from sampling error, while random effect models assume that variation in effect sizes stems from both sampling error and other systematic sources of variance (e.g., operationalisation of ASB). Random effect models provide a more conservative estimate of effect sizes in a population.

Potential moderator variables were examined to reduce possible heterogeneity among effect sizes. Weighted mean study effect sizes were used for moderator

analyses. Each study contributed one effect size to the analyses with the exception of studies reporting data separately for participants with comorbid ADHD and ASB, where such studies provided an effect size for antisocial and comorbid groups separately. Age, proportion of females, correctional recruitment, comparison group type and ADHD comorbidity were analyzed as potential moderators of effect sizes. These moderators were examined as between-study variables impacting on effect size magnitude. Average age was calculated for each study by averaging antisocial and control group ages. All categorical variables were dummy coded to allow for meta-analytic regression analyses. Correctional recruitment was examined to assess potential bias in effect sizes derived from in was expressed as a dichotomous variable with a study coded 1 if the sample was recruited from a correctional setting and 0 otherwise. Comparison group type was expressed as a dichotomous variable with a study coded 1 if the comparison was made with antisocial controls and 0 if the comparison was made with a normal control group. ADHD comorbidity was expressed as a dichotomous variable, with 1 representing comorbidity and 0 representing no comorbidity. IQ was not examined as a moderator of EF effect sizes, given that IQ test performance is dependent on a range of neuropsychological functions, including EF (Dennis et al. 2009). Effect sizes for group differences in IQ were calculated for each study reporting such data.

A meta-analytic regression random effects model (Hedges and Olkin 1985; Lipsey and Wilson 2001) was used to examine possible moderating effects of the continuous variables of age, and proportion of females, and the categorical variables of correctional recruitment, ADHD comorbidity and comparison group type on effect sizes. Duval and Tweedie's (2000) trim and fill method was used to explore publication bias. Finally, calculated effect sizes were checked using the

Comprehensive Meta-Analysis software package (Borenstein et al. 2005). All calculated effect sizes and related statistics were the same as those obtained using the software package.

RESULTS

PARTICIPANTS

A total of 126 studies involving 14,786 participants (5,847 antisocial and 6,904 controls) met inclusion criteria for the meta-analysis. The total number of participants exceeded the number of antisocial and control participants combined given that some study samples did not divide participants into groups. Studies included 391 antisocial participants who had comorbid ADHD characteristics. Antisocial participants had a mean age of 22.31 years old (*SD* = 10.50) and the controls had a mean age of 21.86 years old (*SD* = 10.14). Participants included 4,125 females and 1,388 reported minority participants. There were 73 studies that reported data relating to IQ (64 studies allowing calculation of effect sizes), 26 studies that used antisocial comparison groups, and 62 studies that recruited antisocial participants from correctional settings. Antisocial groups had significantly lower IQ scores (*M* = 97.08, *SD* = 13.46) compared to comparison groups (*M* = 103.27, *SD* = 13.64) using a paired samples *t*-test, $t(70) = -7.97$, $p < .001$.

WEIGHTED STUDY EFFECT SIZES

A total of 570 effect sizes for EF measures were initially calculated across all studies, with these effect sizes then used to calculate weighted mean effect sizes for each study. All calculated effect sizes for each EF measure are provided in the supplemental table. Eight studies produced negative weighted mean effect sizes, and 57 studies produced weighted mean effect sizes that were not significantly different from zero ($p > .05$). Weighted mean effect sizes ranged from -1.05 to 5.14 across studies. Both fixed and random effects models for the summary grand mean effect size estimate where produced across studies, with effect sizes (mean, standard error,

variance, %OL, 95% CIs) and homogeneity statistics displayed in table 1. According to the fixed effect model, the weighted grand mean effect size was d = 0.44 (95% CIs from 0.41 to 0.47), indicating that across studies the average difference between antisocial and comparison groups on EF scores was 0.44 standard deviations. This effect was significantly different from zero (z = 30.60, p <.0005) and was in the medium range compared to the medium to large grand mean effect size found by Morgan and Lilienfeld (d = 0.62) . According to the random effects model, the average difference between antisocial and comparison groups was d = 0.53 (95% CIs from 0.45 to 0.61) standard deviations on EF scores.

For the fixed effect model, the test of homogeneity was statistically significant (Q = 767.61, p <.0005) and I-squared was greater than 50% at 83.72%, indicating that the sample of effect sizes was heterogeneous, with not all effect sizes derived from a single population. Overall, these results indicated that there was a robust association between ASB and EF, although there was significant variation across studies in the magnitude of effect sizes.To address the significant variation in effect sizes across studies, effect sizes were first grouped according to ASB operationalisations.

*** INSERT TABLE 1 ABOUT HERE ***

ANTISOCIAL BEHAVIOUR CATEGORIES

Table 1 also displays weighted effect sizes (mean, standard error, variance, %OL, 95% CI's) and homogeneity statistics for a fixed effect model of effect sizes grouped by ASB categories. The magnitude of mean effect sizes varied considerably across antisocial groups. Mean weighted effect sizes ranged from 0.19 to 0.62 across ASB groups, and were all significantly different from zero (p <.05). The largest mean effect size was for criminality (d = 0.62), and the lowest effect size was for ASPD (d

= 0.19). Heterogeneity in effect sizes decreased when effect sizes were grouped by ASB operationalisations, although effect sizes remained significantly heterogeneous across all ASB categories except for ASPD (Q_W = 1156.18, p <.0001 and Q_B = 53.44, p <.0001). Figure 1 displays a forest plot of weighted mean effect sizes for ASB groups, where the mean effect size is represented by the marker, and the upper and lower 95% confidence intervals for the estimate are represented by the horizontal lines connected to the marker.

*** INSERT FIGURE 1 ABOUT HERE ***

MEASURES OF EXECUTIVE FUNCTION

Effect sizes were grouped according to EF measures (table 2), with effect sizes ranging from -0.13 to 3.05. The largest effect size for EF measures with more than two studies contributing to the estimate was the Self-Ordered Pointing task (d = 0.83). Measures with medium to large effect sizes included the Porteus Maze Test (d = 0.71) the Delayed Matching to Sample Task (d = 0.59), the Go/No-Go Task (d = 0.56), EF composites (d = 0.55) and the Spatial Working Memory Task (d = 0.54). Heterogeneity in effect sizes reduced when effect sizes were grouped by EF measures, although remained significant (Q_W = 1238.04, p <.0001 and Q_B = 402.04, p <.0001).

*** INSERT TABLE 2 ABOUT HERE ***

Further analyses were conducted to explore whether the smaller grand mean effect size for the present analysis in comparison to Morgan and Lilienfeld's analysis was due to the use of a more inclusive approach to EF measures. The weighted mean effect size was re-calculated using only those EF measures included in Morgan and Lilienfeld's meta-analysis (i.e., Porteus Mazes, Stroop Interference Test, Part B of the

Trail Making Test, the Wisconsin Card Sort Test, and Verbal Fluency Tests). These EF measures produced a fixed model mean weighted effect size of d = 0.34 (95% CIs from 0.31 to 0.37, Q = 741.46, p <.0005), which was lower than both the total mean effect size for the present study and Morgan and Lilienfeld's meta-analysis. EF measures not included in the Morgan and Lilienfeld analysis produced a fixed model mean weighted effect size of d = 0.43 (95% CIs from 0.40 to 0.46, Q = 827.51, p <.0005).

Effect sizes for IQ ranged from -0.52 to 6.96 across the 64 studies reporting means and standard deviations for intelligence scores. The average weighted effect size for group differences in IQ across studies using a fixed effect model was d = 0.48 (95% CIs from 0.42 to 0.53, Q = 102.09, p <.0001) and d = 0.57 (95% CIs from 0.43 to 0.71) using a random effect model. The average IQ effect size was in the same range as the average effect size for EF. Using meta-analytic regression, mean IQ effect sizes were significantly associated with mean effect sizes for EF across studies (Q_{Model} = 11.98, df = 1, p <.0005; $Q_{Residual}$ = 118.55, df = 69; R^2 = .09), indicating that larger effect sizes for IQ differences were associated with larger effect sizes for EF differences.

ANALYSIS OF MODERATORS

Meta-analytic regression was performed on studies reporting data for the moderators of correctional recruitment, comparison group type, comorbid ADHD, age and proportion of females, with mean effect size as the dependent variable and the inverse variance of effect sizes used as the weighting variable (Table 3). The Q_{Model} was significant, (Q_{model} = 25.35, df = 5, p <.0005), indicating that the moderator variables accounted for a significant level of variability in effect sizes. The variables

of correctional recruitment, comparison group type and comorbid ADHD emerged as significant predictors of mean study effect sizes. These results indicated that larger effect sizes were associated with studies that recruited participants from correctional settings, used non-antisocial comparison groups and included participants comorbid with ADHD and ASB features.

*** INSERT TABLE 3 ABOUT HERE ***

PUBLICATION BIAS

Using the meta-analytic analog to AVOVA, there was no significant difference in weighted mean study effect sizes between unpublished (N = 11) and published (N = 115) studies ($Q_{Between}$ = .21, df = 1, p > .05). The trim and fill method (Duval and Tweedie 2000) was used to assess publication bias. Inspection of the observed funnel plot of mean study effect sizes and the standard error of effect sizes in Figure 2 indicated an asymmetry around the overall weighted mean effect size suggestive of publication bias. Using the trim and fill method it was estimated that 26 studies were missing to the left of the mean study effect size due to publication bias (see figure 2). The trim and fill adjusted mean study effect size estimate was d = 0.33 (95% CIs from 0.31 to 0.36) using a fixed effects model and d = 0.34 (95% CIs from 0.25 to 0.43) using a random effects model.

*** INSERT FIGURE 2 ABOUT HERE ***

DISCUSSION

Consistent with the results of Morgan and Lilienfeld's (2000) meta-analysis, the results of the present meta-analysis confirm that there is a robust and statistically significant association between ASB and poorer executive functioning. This effect hold across varying study methodologies, including different antisocial groups and EF measures. An average weighted grand mean effect size of 0.44 standard deviations difference between antisocial and comparison groups was found across studies. This effect size was in the medium range, compared to the medium to large 0.62 average weighted mean effect size produced by Morgan and Lilienfeld. Given the current state of the literature in terms of the variation across studies in methodologies, caution should be exercised in interpreting grand mean effect sizes. We argue that the true value of effect sizes cannot be estimated at the present time due to this methodological variation. Rather, the results of Morgan and Lilienfeld's and present meta-analysis at best indicate that there is a robust association between ASB and EF impairments, and highlight the need for future studies to examine specificity in impairments. The results of the present meta-analysis were likely to be a more accurate reflection of the association between EF and ASB compared to Morgan and Lilienfeld's meta-analysis given the inclusion of a wider range of studies and increased statistical power with the inclusion of a larger number of studies.

It should be pointed out that the grand mean effect size was based on a highly heterogeneous sample of effect sizes, indicating that effect sizes were not derived from a single population. In large part, this heterogeneity is likely a direct reflection of the variability within antisocial individuals as a group. In general, more contemporary EF measures produced some of the largest effect sizes, including the Self-Ordered Pointing task (d = 0.83), Risky Choice Task (d = 0.63), the Delayed

Matching to Sample task (d = 0.59), and the Spatial Working Memory task (d = 0.54). It is possible that more recently developed measures of EF are better able to differentiate between antisocial and comparison groups. EF measures are likely to vary significantly according to their usefulness in discriminating between ASB and comparison groups performance.

Effect sizes for EF impairment varied across ASB categorizations. Effect sizes for EF measures were found to be largest for the categories of criminality (d = 0.61), ODD/CD (d = 0.54) and psychopathy (d = 0.42). However, caution must be observed when interpreting differences in EF between ASB categorizations given the substantial overlap between categories. ODD/CD has been characterized as a precursor to the development of later persistent ASB (Loeber and Farrington 2000). Theoretical models of the development of antisocial behavior may be more useful in classifying antisocial individuals, including Moffitt's (1993) developmental taxonomy. Moffitt (1993) proposed that neurocognitive deficits present from an early age are a key mechanism underlying the expression of serious and persistent antisocial behavior that emerges in childhood and continues throughout the lifecourse. Based on the theory, only those most serious and persistently antisocial individuals will display neurocognitive impairments. Such theories are necessary to form testable hypotheses regarding the role and nature of EF impairments in antisocial behavior, as opposed to atheoretical overlapping categories focused on describing rather than explaining antisocial behaviors.

The use of developmental data in examining the links between EF and ASB is rare, which has impeded efforts in identifying the etiological role of neurocognitive impairments in the expression of ASB over the lifecourse across different groups of antisocial individuals. It is possible that different forms of ASB develop along

separate but related developmental trajectories. There is some support for Moffitt's (1993) assertion that early onset neurocognitive deficits are associated with lifecourse persistent ASB and not ASB limited to adolescence (Moffitt 2006; Piquero 2001; Raine et al. 2005). There is also developmental evidence indicating that neurocognitive functioning tests, including measures of EF, fail to differentiate between lifecourse persistent and adolescence limited antisocial groups (Barker et al. 2007; White, Bates, and Buyske 2001). Such evidence suggests that there is likely to be heterogeneity in EF impairments both within and between current conceptualizations of antisocial groups.

There is emerging support for the hypothesis of variation in EF impairments across subtypes of antisocial behavior. Barker et al (2007) found that there was heterogeneity in EF impairments within the ASB category of CD. Their results indicated that EF impairments were associated with physical aggression but not theft. Furthermore, the results of Barker et al. (2011) confirmed that EF impairments were associated with physical aggression but not theft, even after controlling for ADHD. Such results highlight the importance of examining how the etiological role of EF impairments may differ across subtypes of ASB.

Compared to Morgan and Lilienfeld's study, there were a number of significant differences in effect sizes produced for ASB categories. These included effect sizes for psychopathy (0.42 vs. 0.29), criminality (0.61 vs 1.09), delinquency (0.41 vs 0.86) and ODD/CD (0.54 vs 0.40) for the current and Morgan and Lilienfeld's meta-analysis respectively. It is probable that these differences in part reflect changing sampling methods. For example, in most studies published after the original meta-analysis psychopathy studies used the PCL-R or derivatives for operationalisation, compared to the use of other personality measures (e.g., California

Psychological Inventory and the Minnesota Multiphasic Personality Inventory) by studies in Morgan and Lilienfeld's meta-analysis. Furthermore, in the current meta-analysis a large number of studies adopted more specific operationalisations of ASB, including psychopathy and ASPD as opposed to operationalisations based solely on legal status (i.e., criminality and delinquency). Rather than viewing this situation as a limitation, we believe that this reflects the evolving nature of research examining EF and ASB. Research studies are moving toward identifying specificity in EF impairments among specific populations of antisocial individuals as opposed to relying on generalized operationalisations based on criminal history factors alone.

The association between ODD/CD and EF has been questioned due to findings suggesting that the association can be explained by comorbidity of these externalizing disorders with ADHD, which has been associated with EF impairments (Pennington and Ozonoff 1996). However, more recent findings suggest that ODD/CD is significantly associated with EF impairment after controlling for ADHD (Barkley et al. 2001; Clark, Prior, and Kinsella 2000; Van Goozen et al. 2004). Results from the meta-analysis indicated that ASB comorbid with ADHD was associated with poorer EF performance compared to non-comorbid antisocial groups. ADHD is highly comorbid with ASB and increases the risk of adult criminality, although ADHD alone is not a sufficient risk factor for later ASB (Loeber and Farrington 2000; Nigg 2003; Satterfield et al. 2007). Current evidence suggests that the effects of ADHD are likely to additive in increasing EF difficulties among antisocial individuals (Déry et al. 1999; Van Goozen et al. 2004). However, further research is needed to examine how ADHD may impact on the association between ASB and EF impairments.

The association between ASB and EF impairment varied across EF measures, which suggests that particular EF processes may be more strongly associated with

ASB. Measures of working memory, spatial working memory and attention were found to have some of the largest effect sizes, including Spatial Working Memory, Self-Ordered Pointing and the Go/No-Go tasks. Compared to Morgan and Lilienfeld's meta-analysis, EF measure effect sizes produced by the present analysis were largely similar. These included effect sizes for Trail Making Test Part B (.38 vs. .40), Porteus Mazes (.71 vs. .80), Stroop (.35 vs. .35) and Verbal Fluency (.36 vs. .26) for the present and Morgan and Lilienfeld's meta-analysis respectively. Interestingly, some of the more common or traditional measures of EF (viz., the Tower of London, Trail Making Task Part B, Wisconsin Card Sort, and verbal fluency) produced small to medium effect sizes. These findings suggest that there may be variability across EF measures in their abilities to differentiate between antisocial and control groups, and is suggestive of specificity in EF impairments for antisocial individuals.

A further issue with the study of ASB and EF relates to the measures of EF used to assess performance. Most EF measures are designed to be sensitive to clinical impairments in functioning. While useful in clinical settings, these measures may not be sensitive enough to capture sub-clinical EF problems displayed by ASB individuals or individual differences in EF that may impact on everyday functioning. It is possible that the problems in EF associated with some groups of antisocial individuals result from such sub-clinical or individual differences in EF. This highlights a need for further research to construct and validate EF measures relevant to everyday functioning that are sensitive to sub-clinical problems and individual differences in EF.

Rather than regard ASB as specific to EF impairments, a more accurate view may be that ASB is associated with a broader syndrome of more generalized neurocognitive impairments that include EF impairment. Antisocial groups had

significantly lower IQ scores compared to comparison groups, suggesting poorer general intellectual functioning. The average effect size for IQ was similar in magnitude to effect size for EF, with larger differences in IQ associated with larger differences in EF between ASB and control groups. This was not unexpected, given that EF correlates highly with general intelligence (Ardila, Pineda, and Rosselli 2000).

It must also be noted that EF impairments are not unique to ASB and have been linked to a range of clinical disorders, including ADHD, substance use disorders, autism spectrum disorders, schizophrenia and bipolar disorder (Giancola, Shoal, and Mezzich 2001; Giancola and Tarter 1999; Martinez-Aran et al. 2002; Pennington and Ozonoff 1996; Schug and Raine 2009; Willcutt et al. 2005). Furthermore, ASB generally does not present in isolation, but is a component of a larger constellation of psychological, emotional and behavioral problems, including physical and mental health issues, substance use, and poor academic functioning (Abram et al. 2003; Farrington 2005; Kenny and Nelson 2008; Loeber and Farrington 2000; Odgers et al. 2007). It is possible that EF impairments are not specific to ASB, but are associated with these psychological, emotional, and behavioral problems in general.

The current findings do not provide conclusive support for the assertion that antisocial individuals have structural or functional PFC impairments. Further research is needed to explore this issue, including neuroimaging studies. Recent meta-analysis findings indicate that antisocial and violent behavior is associated with structural and functional abnormalities in the PFC (Yang and Raine 2009). Specifically, structural and functional impairments are consistently observed in the right orbito-frontal cortex, left dorso-lateral PFC, and right anterior cingulated cortex (Yang and Raine 2009). Structural and functional impairments in other brain regions have been implicated in the development of ASB, including the amygdala, ventro-medial PFC

and the hippocampus (Crowe and Blair 2008). Further research is needed to explore how structural and functional brain impairments in antisocial individuals translate to an increased risk of engaging in ASB.

Analyses indicated that the variables of correctional recruitment, antisocial comparison groups and ADHD comorbidity moderated the association between EF and ASB. These results highlight the need for studies to consider sampling methods in examining the links between ASB and EF, as they have the potential to attenuate the magnitude of findings. Age and sex did not significantly moderate the association between EF and ASB. However, it is not possible to rule out any of these variables as having an effect on the association between EF and ASB, given that variation in these variables was likely to have been limited in the sample of studies included in the meta-analysis. For example, there is limited research that has examined the role of age in the association between EF and ASB. Developmental research examining a wider age range is needed to examine how EF relates to ASB at different points of the life course. However, even longitudinal designs have limitations in establishing causal relations, given the retrospective nature of analyses.

A major drawback to the current study was the inability to fully examine the effects of substance abuse/dependence and ADHD comorbidity, since these variables are significantly associated with both ASB and EF impairment. Too few studies consistently reported data for these variables. Despite this limitation being highlighted by Morgan and Lilienfeld (2000), few studies have made a concerted effort to examine the potentially moderating effects of the variables. While it is known that both ADHD and substance abuse are associated with EF and ASB, further research is needed to examine the mechanisms through which ADHD and substance abuse/dependence are associated with EF and ASB. For example, current evidence

suggests that the relationship between EF and substance use is highly complex and reciprocal, where poor EF may both increase the risk of engaging in substance use and be the result of prolonged substance use (Blume and Marlatt 2009; Clark, Thatcher, and Tapert 2008; Verdejo-Garcia et al. 2005; Verdejo-Garcia et al. 2004). Furthermore, poorer EF has been associated with greater difficulties in successfully treating substance use problems (Aharonovich, Nunes, and Hasin 2003; Blume and Marlatt 2009).

Substance use has the potential to temporarily and developmentally impair neurocognitive functioning, although the nature and extent of impairment appears to vary by the type of substance consumed. Substances that have been linked to neurocognitive impairments include alcohol (Bates, Bowden, and Barry 2002), cannabis (Verdejo-Garcia et al. 2005), cocaine (Aharonovich, Nunes, and Hasin 2003), methamphetamine (Kalechstein, Newton, and Freen 2003) and 3,4-methylenedioxymethamphetamine (MDMA; Bhattachary and Powell 2001; Verdejo-Garcia et al. 2005). Substance induced neurocognitive impairments may increase the risk of engaging in antisocial behavior. This situation appears plausible given that a large proportion of individuals entering the justice system test positive to substance use (Gaffney et al. 2009). In an Australian study examining the links between drug use and offending using a representative sample of detained offenders, results indicated that 44% of adult detainees reported taking drugs prior to committing their offence (Gaffney et al. 2009). However, based on current knowledge it is not possible to determine the extent to which the links between substance use and ASB may be explained by other factors, including lifestyle.

There continues to be a range of methodological problems in the research literature, including poorly specified operationalisations of ASB and EF, small

samples, a lack of prospective data, poor comparison group selection, and a failure to adequately control for confounding factors. However, despite considerable methodological variation across studies, a consistent and robust association between antisocial behavior and performance on EF measures was still observed.

The causal relationship between EF impairments and ASB remains unclear due to the predominant use of cross-sectional samples, which impedes efforts to determine whether EF impairments increase the likelihood of ASB or whether an antisocial lifestyle produces EF impairments or a combination of the two. The presence of EF impairments does not explain whether or how such impairments relate to ASB at the time such behavior is performed. The challenge for researchers is to explore how EF impairments translate to an increased tendency for ASB, and how these impairments interact with environmental characteristics.

Emerging research suggests that neurocognitive impairments may best be understood as a mechanism mediating the link between genetic risks and externalizing behavior problems (DeYoung et al. 2006; Langley et al. 2010). Langley et al. (2010) examined how the high activity COMT (catechol O-methyltransferase) valine/valine genotype in ADHD may moderate the links between cognitive functioning and ASB. Results indicated the high activity COMT genotype in ADHD was associated with impaired social understanding, which was in turn associated with increased ASB. Furthermore, the genotype was also associated with impaired EF, although this did no increase the risk of ASB. In a separate study DeYoung et al. (2006) found that the 7-repeat allele of the dopamine D4 receptor gene (DRD4-7) moderated the link between externalizing behavior and cognitive functioning, where the presence of DRD4-7 attenuated the negative association between externalizing behavior and general

cognitive functioning. Such studies are important in understanding the role of neurocognitive functioning in risk pathways for the development of ASB.

The robust association between ASB and EF impairments has implications for the treatment of ASB. EF abilities may be targeted to improve treatment effectiveness and reduce the likelihood of future ASB. There is evidence to suggest that poorer EF is associated with a range of negative treatment outcomes among offenders, including increased treatment drop out and increased disruptive behavior during treatment (Fishbein and Sheppard 2006). Treatment programs that aim to improve EF abilities may be useful in reducing the chances of engagement in ASB, particularly when such treatment is directed toward younger children. There is evidence to suggest that cognitive enhancement programs can be effective in improving the development of EF abilities among preschool children (Diamond et al. 2007). Research is needed to examine the effectiveness of cognitive enhancement programs in preventing the development of ASB.

CONCLUSION

The present meta-analysis expanded on the analysis conducted by Morgan and Lilienfeld (2000) by examining a wider range of EF measures and more recent studies. A robust association between ASB and performance on EF measures was observed that varied according to ASB groups and measures of EF. The findings highlighted the continued methodological variation across studies that contributed to the significant heterogeneity in computed effect sizes. The robustness of the link between EF and ASB was confirmed by the association holding across different methodological approaches. Further research is needed to examine specificity in impairments across types of antisocial individuals and measures of EF, factors that

may moderate the association (e.g., ADHD and substance abuse), and the role of EF development in the expression of ASB. A concerted effort from researchers is needed in examining EF and ASB from theoretical frameworks to better specify the constructs.

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Table 1. Weighted Effect Sizes for Fixed and Random Effect Grand Mean Models, and Mean Fixed Effect Estimates by Antisocial Category

Group		Effect Size					95% Confidence Interval		Homogeneity Test		
	Number of Studies	Point Estimate	Standard Error	Variance	*z*-value	%OL	Lower	Upper	*Q*-value	*df*(*Q*)	I-squared
Grand Mean											
Fixed	126	0.44†	0.01	0.00	30.60	70.10	0.41	0.47	767.61†	125	83.72
Random	126	0.53†	0.04	0.00	13.42	65.50	0.45	0.61			
ASB Group											
ASPD	11	0.19**	0.06	0.00	2.97	85.60	0.06	0.31	17.68	10	43.45
Criminality	48	0.62†	0.04	0.00	17.17	61.30	0.55	0.69	402.57†	47	88.33
Delinquency	27	0.41†	0.02	0.00	19.28	73.30	0.37	0.45	278.22†	26	90.66
ODD/CD	35	0.54†	0.04	0.00	15.33	65.50	0.47	0.61	94.53†	34	64.03
Physical aggression	25	0.41†	0.03	0.00	14.83	73.30	0.36	0.46	101.42†	24	76.34
Psychopathy	29	0.42†	0.04	0.00	10.36	71.70	0.34	0.50	272.64†	28	89.73
Within group									1167.06†	169	
Between group									50.62†	5	

* p <.05, ** p <.01, *** p <.001, † p <.0001

Figure 1. Forest Plot of Weighted Mean Effect Sizes for Antisocial Categories.

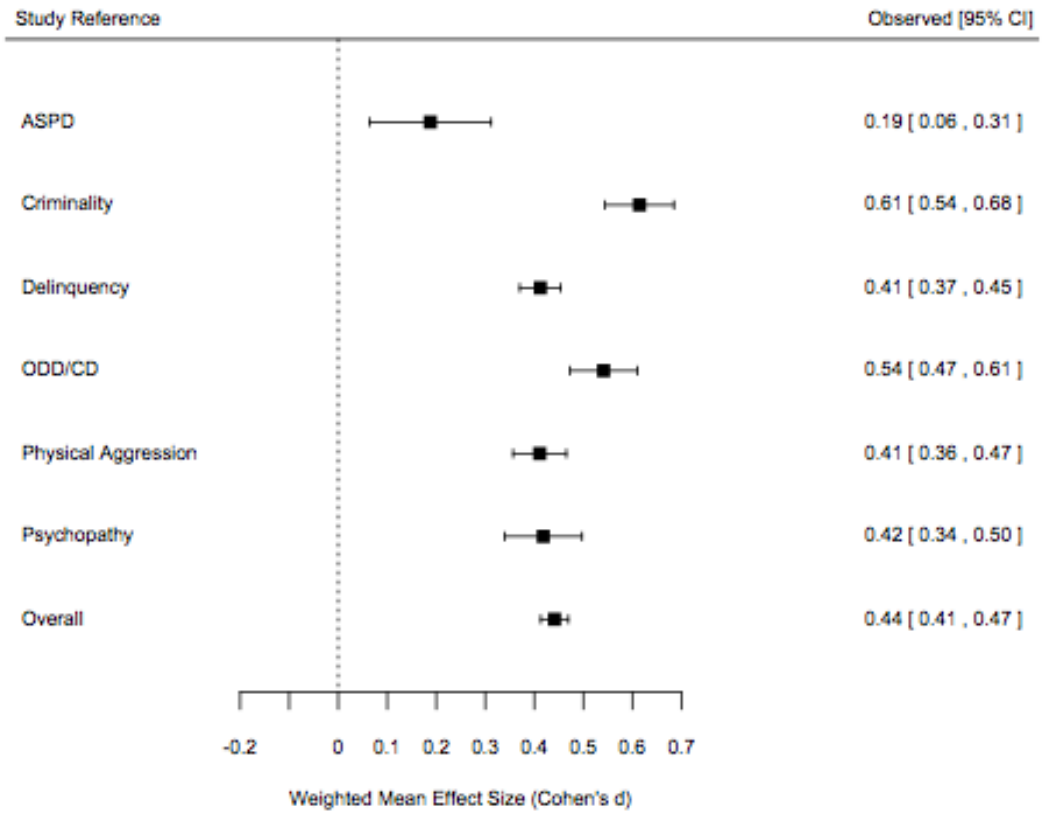

Table 2. Weighted Mean Effect Sizes for Executive Function Measures

EF Measure	Effect Size					95% Confidence Interval		Homogeneity Test	
	Number of Effect Sizes	Point Estimate	Standard Error	z-test	%OL	Lower	Upper	*Q*	*df(Q)*
OAT	1	3.05†	0.42	7.30	6.80	2.23	3.86	-	-
RFFT	1	1.04†	0.25	4.24	43.60	0.56	1.53	-	-
BADS	2	0.94†	0.18	5.14	46.70	0.58	1.30	0.79	1
IRT	1	0.85*	0.40	2.11	50.00	0.59	1.64	-	-
SOP	4	0.83†	0.09	8.97	51.20	0.65	1.01	15.74	3
CAT	1	0.74†	0.11	6.60	54.80	0.52	0.96	-	-
PMT	22	0.71†	0.03	18.96	56.00	0.63	0.78	142.89	21
NRT	1	0.70†	0.11	6.30	57.30	0.48	0.92	-	-
SMM	3	0.69†	0.17	4.06	57.30	0.36	1.02	6.04	2
RCT	2	0.63†	0.10	6.65	59.90	0.45	0.82	0.02	1
DOT	2	0.63**	0.22	2.80	59.90	0.20	1.06	1.16	1
SET	1	0.61*	0.26	2.36	61.30	0.10	1.12	-	-
Vigilance Task	1	0.59†	0.17	3.52	62.70	0.26	0.93	-	-
DMS	3	0.59***	0.18	3.37	62.70	0.25	0.94	1.23	2
GNG	12	0.56†	0.06	8.83	64.10	0.44	0.69	76.51	11
EF Composite	18	0.55†	0.03	19.50	64.10	0.49	0.60	228.88	17
SWM	6	0.54†	0.09	5.71	65.50	0.35	0.72	12.80	5
DKEFS	1	0.53	0.36	1.47	65.50	-0.18	1.24	-	-
PAL	2	0.53†	0.11	5.00	65.50	0.32	0.74	2.48	1
BCT	7	0.47†	0.08	5.74	68.50	0.31	0.64	17.30	6
Shape School	1	0.46***	0.13	3.40	68.50	0.19	0.72	-	-
D2	2	0.45*	0.19	2.40	70.10	0.08	0.82	1.83	1
ROCFT	7	0.45†	0.07	6.01	70.10	0.30	0.60	36.36	6
SST	12	0.42†	0.08	5.37	71.70	0.26	0.57	34.35	11
Flanker Test	1	0.38†	0.10	3.68	73.30	0.18	0.59	-	-
TMT	24	0.38†	0.03	10.39	73.30	0.31	0.45	72.64	23
ID/ED	6	0.38†	0.09	4.11	73.30	0.20	0.56	39.22	5
COWAT	26	0.36†	0.03	10.53	74.90	0.29	0.43	114.97	25
Day-Night Task	1	0.36**	0.13	2.67	74.90	0.10	0.62	-	-
SCWT	27	0.35†	0.03	12.49	74.90	0.30	0.41	120.29	26
VWM	1	0.33	0.30	1.12	76.60	-0.25	0.92	-	-
DGT	4	0.32†	0.09	3.71	76.60	0.15	0.49	1.63	3
Card	6	0.32†	0.08	4.19	76.60	0.17	0.47	18.02	5
CPT	10	0.29†	0.08	3.86	80.10	0.14	0.44	19.82	9
IGT	9	0.25†	0.07	3.67	81.90	0.11	0.38	44.77	8
AVLT	2	0.18	0.13	1.42	87.50	-0.07	0.43	1.26	1
Digit Span	13	0.17†	0.05	3.77	87.50	0.08	0.27	24.92	12
WCST	36	0.17†	0.02	7.13	87.50	0.12	0.22	169.56	35
ToL	9	0.11	0.07	1.64	91.50	-0.02	0.25	28.63	8
OCT	1	0.08	0.13	0.60	93.60	-0.18	0.34	-	-
EGT	1	0.05	0.33	0.15	95.70	-0.59	0.69	-	-
Simon Task	1	0.03	0.24	0.14	97.80	-0.44	0.51	-	-
CVLT	1	0.00	0.13	0.02	100	-0.26	0.26	-	-
PASAT	1	-0.08	0.13	-0.60	93.60	-0.34	0.18	-	-
Two-Back Test	1	-0.13	0.25	-0.52	89.50	-0.62	0.36	-	-
Within group								1238.04†	250
Between group								402.04†	44

* p <.05, ** p <.01, *** p <.001, † p <.0001

Note. AVLT = Auditory Verbal Learning Test; BADS = Behavioral Assessment of the Dysexecutive Syndrome; BCT = Booklet Category Test; Card = Card Playing task; CAT = Conditional Association Task; CPT = Continuous Performance Task; COWAT = Controlled Oral Word Association Test/Verbal Fluency; CVLT = California Verbal Learning Test; D2 = D2 Test of Attention; DGT = Delay of Gratification Task; DKEFS = Delis-Kaplan Executive Function System (Design Fluency);

DMS = Delayed Matching to Sample task; DOT = Door Opening Task; EGT = Executive Golf Task; GNG = Go/No-Go task; ID/ED = Intradimensional/Extradimensional Shift test; IGT = Iowa Gambling Task; IRT = Inhibitory Reach Task; NRT = Number Randomization Task; OAT = Object Alternation Task; OCT = Object Classification Task for Children; PAL = Paired Associates Learning task; PASAT = Paced Auditory Serial Addition Test; PMT = Porteus Maze Task; RCT = Risky Choice Task; RFFT = Ruff Figural Fluency Test; ROCFT = Rey-Osterreith Complex Figure Test; SCWT = Stroop Color-Word Test; SET = Six Elements Task; SOP = Self-Ordered Pointing task; SMM = Sequential Matching to Memory Task; SST = Stop Signal Task; SWM = Spatial Working Memory task; TMT = Trail Making Test Part B; ToL = Tower of London; VWM = Verbal Working Memory; WCST = Wisconsin Card Sort Test.

Table 3. Meta-Analytic Regression Random Effects Model Results for Moderator Variables

Variable	B	SE	z	95% Confidence Interval Lower	Upper
Constant	.20	.12	1.71	-.03	.43
Correctional recruitment	.41†	.10	4.13	.22	.61
Antisocial Contrast	-.44***	.12	-3.67	-.67	-.20
Comorbid ADHD	.30*	.14	2.13	.02	.58
Age	.01	.00	1.23	-.00	.02
Proportion of females	.04	.13	0.28	-.22	.29
			R^2 = 0.10		
			Q_{Model} = 25.35†, *df* = 5		
			$Q_{Residual}$ = 222.32†, *df* = 116		

* p <.05, ** p <.01, *** p <.001, † p <.0001

Figure 2. Trim and Fill Analysis Funnel Plots for Publication Bias in Mean Study Effect Size