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Executive–cognitive functioning in the development of antisocial personality disorder

Michael C. Stevens, Richard F. Kaplan*, Victor M. Hesselbrock

Department of Psychiatry, MC2103, University of Connecticut Health Center, Farmington, CT 06030, USA

Abstract

The present study examined the association of cognitive–executive abilities to two risk factors for alcoholism, i.e., antisocial behaviors and a family history (FH+) of alcohol dependence. A sample of 91 right-handed, non-substance-dependent, young male adults recruited from the community were classified into three groups: (1) a control group of $n=32$ men with no history of DSM-III-R childhood conduct disorder (CD) or antisocial personality disorder (ASPD); (2) $n=25$ men who met criteria for a DSM-III-R childhood CD diagnosis, but did not meet diagnostic criteria for ASPD (i.e., CD/ASPD−); and (3) $n=34$ men who met DSM-III-R criteria for ASPD. They were further divided into those with and without a positive family history of alcoholism. A two-way (Antisocial Profile (3)×Family History of Alcoholism (2)) ANOVA was used to compare several neuropsychological measures of executive–cognitive functioning (ECF) ability. Verbal abstraction ability was found to be significantly lower in ASPD subjects compared with controls and CD-only subjects, inversely related to antisocial behavior severity (as measured by symptom count). CD-only and control subjects' abstraction ability were statistically indistinguishable. FH+ was associated with increased errors in planning performance on the Porteus Maze Test and diminished performance on Luria's simple alternate-tapping motor tasks. The effect was more pronounced when inhibition of prepotent motor planning was required. Results are consistent with previous work examining ECF ability in antisocial samples that find subtle differences in ECF ability compared to controls. The findings suggest that normal versus abnormal behavioral outcome for children with conduct problems may be influenced by cognitive ability profile, perhaps because of varying maturational processes.

Keywords: Antisocial personality disorder; Conduct disorder; Executive functioning; Cognitive ability; Family history

* Corresponding author. Tel.: +1-860-679-3497.
E-mail address: kaplan@psychiatry.uchc.edu (R.F. Kaplan).

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1. Introduction

The consequences of childhood conduct disorder (CD) and its continuation into adulthood as antisocial personality disorder (ASPD) can represent tremendous societal and medical costs. CD and ASPD have been linked to various behavioral, social, and health problems, including aggression, criminal behavior, and substance disorder (Jordan, Schlenger, Fairbank, & Caddell, 1996; Lewis, Cloninger & Prais, 1983; Moeller, Doughtery, Lane, Steinberg, & Cherek, 1998; Patrick & Zempolich, 1998). Previous work highlights the role of CD and ASPD as a risk factor for substance dependence (Hesselbrock, Meyer & Hesselbrock, 1992), showing that adults with ASPD have a three- to fourfold greater likelihood of developing substance dependence compared to adults without ASPD. The widely noted association between alcoholism and ASPD has led to several theories proposing an ''antisocial alcoholic'' subtype (Babor et al., 1992; Cloninger et al., 1981; Jellinek, 1960), possibly with different etiological and developmental mechanisms than other subtypes.

Antisocial behavior shows heterogeneity of onset and expression across childhood and adolescence (Feehan, McGee, & Williams, 1994; Hinshaw, Lahey, & Hart, 1993; Lahey & Loeber, 1994; Lahey et al., 1995; Lahey et al., 1998; Loeber, 1991; Moffitt, 1993). These differences have complicated investigators' search to identify those factors that operate during maturation, which may be associated with ASPD. Longitudinal studies estimate at least one in four CD youth continues to display antisocial behaviors in adulthood (Robins, 1966). This raises the question of what factors operate to result in adult ASPD, or act to prevent the majority of problem-behavior youth from developing ASPD. A related question is whether the same factors also predispose youth to substance abuse and later alcohol dependence.

While environmental factors unquestionably play a role in the development of ASPD (Farrington, 1986), some researchers have examined neuropsychological test performance in an attempt to determine whether various neurobehavioral deficits predispose youth to ASPD. One approach has been to examine the relationship between executive–cognitive functioning (ECF) abilities and ASPD. ECF is thought to represent higher-order abilities involved in planning, initiation, and regulation of goal-directed behavior (Luria, 1980). ECF deficits in alcoholics include abstract reasoning, set shifting, set persistence, attention, verbal/categorical fluency, concept generation, persistence, temporal organization, sequencing, supervisory motor control, hypothesis formation testing, and cognitive flexibility (Giancola & Moss, 1998). The unifying element of these multifaceted abilities is that they are believed to have the majority of their primary neurological substrates in the prefrontal cortex (PFC) and its connections to other cortical and subcortical areas (Fuster, 1997; Luria, 1980).

Research has begun to address whether ECF ability in CD youth (Moffitt, 1993; Pennington & Ozonoff, 1996) or ASPD adults (Kandel & Freed, 1989; Gorenstein, 1987; Gorton, Swirsky-Sacchetti, Sobel, Samuel, & Goron, 1999) differs from healthy controls. In general, results favorably support the idea that deficits on neuropsychological tests of ECF can be found in these groups, as shown in a recent meta-analysis (Morgan & Lilienfeld, 2000). However, it remains unresolved if these deficits are solely attributable to executive function, or if impairments of other neuropsychological abilities are implicated. Despite the encouraging results, no published studies to date have examined whether ECF ability varies