The goal of this report is to critically review research on executive functioning (EF) and working memory in individuals with fetal alcohol spectrum disorder (FASD). Individuals with FASD exhibit EF deficits in the areas of cognitive flexibility, planning and strategy use, verbal reasoning, some aspects of inhibition, set shifting, fluency, working memory, and, recently, on tests of emotion-related or hot EF. Some researchers have linked prenatal alcohol exposure to abnormalities in the development of the frontal cortex of affected individuals or animals. One common finding is that these EF deficits persist regardless of whether the individual has facial dysmorphology. Furthermore, EF deficits are not simply due to a low IQ in these individuals. More research with larger sample sizes, smaller age ranges, and consistent measurement tools is needed in this area to ameliorate some inconsistencies in the literature. Furthermore, researchers should now focus on studying the pattern of weakness in EF in individuals with FASD as well as relations among working memory and EF, which will help to identify specific areas of weakness, to enhance diagnosis, and to improve treatment. There is limited research on the development of EF in individuals with FASD, which can have important implications for understanding of how these deficits unfold from childhood through adulthood.

Key Words: Executive Functioning, Fetal Alcohol Spectrum Disorder, Working Memory.

FETAL ALCOHOL SYNDROME (FAS) was first identified in 1973 by Jones and Smith. Recognition was based on case studies (Jones et al., 1973) in which researchers noted a similar pattern of malformations among infants born to alcoholic mothers. The worldwide incidence of FAS in the general population has been estimated at 0.97 in 1000 births (Abel, 1995), although estimates may be highly variable across different countries. In a population-based Seattle study, Sampson et al. (1997) estimated the incidence of FAS to be three in 1000 births, with the combined occurrence of FAS and alcohol-related neurodevelopmental disorder (ARND) to be one in 100 births. FAS is the most common known cause of mental retardation and occurs more often than the two most common birth defects (Down syndrome and spina bifida) combined (National Institute of Alcohol Abuse and Alcoholism, 1990). Yet, FAS is one of the only causes of mental retardation that is clearly preventable. FAS puts an enormous cost on society, with a devastating economic impact (Abel and Sokol, 1991).

Prenatal alcohol exposure can lead to a range of outcomes including FAS, partial FAS, and ARND. These diagnostic outcomes fall under the umbrella term fetal alcohol spectrum disorder (FASD). The term FASD is not intended for use as a clinical diagnosis, but it is an umbrella term used to describe the full range of outcomes observed among individuals with prenatal alcohol exposure. The cognitive deficits of FASD are not fully understood, and exploring these deficits is invaluable for enhancing our knowledge of the neuropsychological sequelae of these individuals and, ultimately, for improving diagnosis and treatment. Executive function (EF) and working memory are cognitive variables that have recently been implicated in FASD. The focus of this report is to critically review the research on EF and working memory in children, adolescents, and adults with FASD. We review all of the published papers (to our knowledge) on this topic that were found through various article searches. The goal is to highlight the common findings in this area, to discuss discrepancies among studies, and to critique the methodology to promote a more comprehensive understanding of EF and working memory in individuals with FASD. First, however, issues related to diagnosis and classification of alcohol-exposed individuals are discussed. Understanding the different terms is necessary for understanding the different subsets of FASD individuals used in various studies.

DIAGNOSIS AND CLASSIFICATION

Three criteria have been identified to diagnose FAS: 1) growth deficiency in weight and or height; 2) facial features that may include short palpebral fissures, smooth philtrum,
thin upper lip, flat midface, and short nose; and 3) damage to the central nervous system (CNS) (Clarren and Smith, 1978; Sokol and Clarren, 1989). CNS dysfunction may include microcephaly, cognitive deficits, learning problems, attentional difficulties, hyperactivity, and motor problems (Streissguth, 1997). A history of prenatal alcohol exposure was also required.

The term fetal alcohol effect (FAE) has been used to describe children who do not have all the characteristics of FAS (namely absence of some or all facial features and/or lack of growth deficiency) but still have prenatal alcohol exposure and some CNS dysfunction (Clarren and Smith, 1978). The Institute of Medicine (1996) differentiated five different types of prenatal alcohol effects: FAS with and without confirmed alcohol exposure, as well as partial FAS, ARND, and alcohol-related birth defect. Partial FAS includes those with confirmed maternal alcohol exposure, evidence of some facial characteristics, and either growth, CNS deficits, or a complex pattern of behavioral or cognitive abnormalities. Alcohol-related birth defect refers to individuals with some congenital physical abnormalities and a history or maternal alcohol exposure. Last, ARND includes those with CNS deficits or a complex pattern of behavioral or cognitive abnormalities as well as a history of maternal alcohol exposure. The Fetal Alcohol Syndrome Diagnostic and Prevention Network (FAS DPN) four-digit coding system was developed at the University of Washington (Astley and Clarren, 1999). This system ranks diagnostic information in the areas of growth deficiency, facial phenotype, brain dysfunction, and alcohol use. The magnitude of expression of each diagnostic feature is ranked independently on a four-point Likert scale, with one reflecting complete absence of the FAS feature and four reflecting a strong “classic” presence of the FAS feature. In Canada, Chudley et al. (2005) recommend a multidisciplinary approach to diagnosis, harmonizing the Institute of Medicine classifications and the four-digit Diagnostic Code approaches.

It has been noted that few children prenatally exposed to alcohol actually show all the facial features and growth deficiency required to diagnose FAS. This can result in many false negatives, in that affected individuals with prenatal alcohol exposure but who do not have the facial characteristics may not be identified (Sampson et al., 2000). Further, Streissguth and O’Malley (2000) noted that face-based diagnoses may be problematic, because it is not the face that needs services. In fact, the FAS face is the result of alcohol exposure during a very short period of vulnerability during pregnancy. In mice, there is a highly specific window in gestation (day seven) during which alcohol exposure must occur to produce facial features of FAS, a period that corresponds roughly to early during the first trimester in humans (Sulik et al., 1981). Streissguth and O’Malley (2000) suggested that the FAS face is not a good marker for children exposed to alcohol outside this period in gestation, nor is it a good marker for adolescents adults, because these facial characteristics can diminish with age. Further, they note that and unlike the face, the brain may be vulnerable to the effects of alcohol throughout the entire pregnancy. Ultimately, Chudley et al. (2005) suggested that “in the wide array of FASDs, facial dysmorphology is often absent and, in the final analysis, has little importance compared with the impact of prenatal alcohol exposure on brain function” (p. 56).

Mattson et al. (1998) compared children with FAS with those prenatally exposed to alcohol without full FAS and found similar neuropsychological deficits among both groups, regardless of whether the children had the physical features of FAS. Individuals with FAS and FAE also do not show meaningful differences on tests of cognitive abilities, secondary disabilities, and behavioral problems (Sampson et al., 2000). In fact, Conner and Streissguth (1996) suggested that CNS deficits in FAE may be as severe as or even worse than in individuals with FAS.

Thus, it is evident that diagnosis should focus more on CNS deficits, which has strong implications for future research because there is an increased need to identify the unique neuropsychological profile of these individuals or among subsets of individuals to determine precise diagnostic criteria. Children with FASD also may exhibit structural and functional brain damage (Streissguth, 1997) and many primary neuropsychological deficits as well as secondary disabilities. Secondary disabilities include mental health problems, trouble with law, confinement, alcohol and drug abuse, and dropping out of school (Streissguth, 1997). Neuropsychological impairments include deficits in memory, attention, visual-spatial abilities, declarative learning, planning, cognitive flexibility, processing speed (Carmichael Olson et al., 1998), lower IQ, achievement deficits, and learning problems (Streissguth et al., 1994a), as well as language and motor delays (for a review, see Mattson and Riley, 1998). The term FASD is used in this review to refer to individuals with prenatal alcohol exposure and related CNS damage. In some previous studies, however, children with FAS and FAE were differentiated and compared, so in these situations, the terms FAS and FAE are used.

EXECUTIVE FUNCTION AND FASD

Executive function has been defined as higher-order psychological processes involved in goal-oriented behavior under conscious control (Zalazo and Muller, 2002). EF is an umbrella term for many cognitive processes including planning, organized search, inhibition, working memory, set shifting, flexible thinking, strategy employment, and fluency (Welsh and Pennington, 1988; Welsh et al., 1991). EF is thought to be mediated by the frontal lobe of the brain (Welsh and Pennington, 1988; Stuss and Knight, 2002).

Executive function emerges around one year of age, with successful performance on object search, the A-not-B task, and measures of self-control (for a review, see Welsh and Pennington, 1988). Important developments in EF occur
between two and five years, and adult-level abilities are reached on some EF tasks by 12 years, whereas other EF abilities develop until adulthood (for a review, see Zalazo and Muller, 2002). In a normative developmental study, Welsh et al. (1991) found that measures of visual search reached adult performance at age six years, and performance on a measure of impulse control, hypothesis testing, inhibition, and set shifting (Wisconsin Card Sorting Task, WCST) reached adult level at age 10. Finally, measures of verbal fluency, motor sequencing, and planning (Tower test) continued to develop into adulthood. For comprehensive reviews of the development of EF in typical and atypical development, see Pennington (1997), Welsh (2002), and Zalazo and Muller (2002). Factor analytic studies have generally yielded three main EF factors: set shifting, updating working memory, and inhibition (Lehto et al., 2003; Miyake et al., 2000).

Executive Function in Children and Adolescents with FASD

Mattson et al. (1999) evaluated EF in 18 children (aged eight to 15 years) with FAS or with prenatal alcohol exposure but without physical features of FA, as well as nonexposed control children. Children were tested on a trail-making test to assess cognitive flexibility, a Stroop test to assess response inhibition, a tower test to measure planning ability, and a word context test to assess concept formation and verbal reasoning. Relative to the control group of typically developing children, both groups of alcohol-exposed children showed impairments on all EF measures. Furthermore, on all measures except for the switching condition of the Stroop test, the group with FAS did not differ from those with only prenatal alcohol exposure. Hence, cognitive functioning, and specifically EF, is affected even in those children who have prenatal alcohol exposure but no facial dysmorphism.

Schonfeld et al. (2001) compared 18 alcohol-exposed children (aged eight to 15 years) both with and without full FAS with control children on verbal and nonverbal fluency measures of EF with 10 nonexposed control children. On the verbal fluency measures, the child had to list as many words as possible within one minute of hearing a given letter (e.g., F) and a category (e.g., animals). The children were also required to list as many words as possible when switching between two categories (e.g., fruit and furniture). On nonverbal (design) fluency, the child had to make as many different four-line designs within one minute by connecting empty dots, filled dots, and switching between empty and filled dots. Both groups of alcohol-exposed children performed lower than the control group on nonverbal and verbal fluency tests, and, as in Mattson et al. (1999), no difference was found between the alcohol-exposed children with and without physical features of FAS. It was concluded that children prenatally exposed to alcohol appear to have a global fluency deficit in both verbal and nonverbal domains.

Other researchers have found similar results indicating that children with FASD (aged nine to 18 years, n = 10) have difficulty on tests of EF (Kodituwakku et al., 1995). As compared with control children, children with FASD showed marked difficulty on measures of planning (a tower test) and strategy use, attention, and letter but not category fluency. Children were also impaired on the WCST, which requires inhibition, set shifting, and use of feedback to modify behavior. The FASD group, however, did not differ from control children on other measures of regulation of behavior (delayed-response tests), inhibition, and rule learning. Carmichael Olson, et al. (1998) found that relative to control children, adolescents with FAS (n = 9) were impaired on the WCST, as well as tests of visual memory and attention. Similarly, Coles et al. (1997) found that seven-year-old children with FASD (n = 25) performed lower than control children on the WCST. Kodituwakku, et al. (2001a) found that children and adolescents with FASD (n = 20) performed much lower than control children on the WCST and the Children’s Executive Functioning Scale, a behavioral measure of social appropriateness, inhibition, problem solving, initiative, and motor planning. The WCST accounted for a large proportion of the variance on the Children’s Executive Functioning Scale.

The bulk of the EF research has been conducted with school-aged children and adolescents with FASD. Recently, however, Noland et al. (2003) examined EF in more than 300 four-year-olds prenatally exposed to alcohol, cocaine, or marijuana in comparison to unexposed to control groups. The children were tested on a tapping inhibition task, a category fluency task, and a motor planning task, in which the child was to touch their thumb to their fingers using different sequences. On the tapping inhibition task, the child was required to tap once if the experimenter tapped twice and to tap twice if the experimenter taped once. After mastering the rules, test trials were administered. The authors found a significant negative relation between alcohol exposure and performance on the inhibition tapping task, even when they partialled out verbal IQ, prenatal drug exposure, and postnatal environmental factors. Cocaine and marijuana were not related to any of the EF measures, and no relation was found between alcohol exposure and the other two EF tasks. These results may be compromised by the fact that on the tapping inhibition test, less than 30% of the sample mastered the rules, so analyses were limited to performance on training trials. Thus, it is not clear whether alcohol exposure negatively affected inhibition or rule learning. The authors suggested that the other two measures of EF may not be sufficiently developed at four years to ascertain differences.

Executive Function in Adults

Other researchers have found that adults with prenatal alcohol exposure (n = 30) have deficits on many measures of EF (Connor et al., 2000). In fact, Conner et al. found that performance on the Stroop task, trail making, WCST,
design fluency, and working memory were even lower than would be predicted on the basis of the participants’ IQ scores. Thus, the authors concluded that for these measures, alcohol appears to have a direct effect on EF, whereas for other measures (verbal fluency, cognitive estimation, Stroop and trails error scores, and memory and design fluency preservation scores), alcohol may have an indirect effect that is mediated by IQ. Therefore, the authors concluded that EF abilities that are more directly related to alcohol damage involve shifting tasks, attention, visuospatial processing, and holding and manipulating information in working memory. In contrast, EF abilities not directly related to alcohol damage and that tend to be mediated by IQ generally involve error scores. Again, there was no relation between facial dysmorphology and EF deficits.

Kerns et al. (1997) found that adults with FAS (n = 16) performed below the norm on EF tests of letter and design fluency. Participants were divided into an average IQ group and a borderline-low IQ group, with the latter group generally performing lower than the former. For both groups, the authors compared how many standard deviations the two groups were below the mean on IQ and the EF measures and found that both groups performed lower on EF than predicted on the basis of their IQ.

Hot and Cool Executive Function

In their review, Zalazo and Muller (2002) differentiated between cool and hot EF. Cool EF is thought to be mediated by the dorsolateral prefrontal cortex area and represents cognitive EF, on which most research has been conducted. In contrast, hot EF is associated with the orbitofrontal cortex and is involved in regulation of motivated and emotional behavior, as well as in processing affective and nonaffective stimuli. Kodituwakku, et al. (2001b) also distinguished cognition-based EF and emotion-related EF. Kodituwakku et al. reviewed research suggesting that cognition-based EF is mediated by the lateral prefrontal cortex and is involved in problem solving, planning, working memory, conceptual set shifting, nonverbal and verbal fluency, and fluid intelligence. In contrast, emotion-related EF is thought to be mediated by the orbitofrontal cortex and is involved in responses to reward and punishment stimuli. Evidence for this fractionation of the prefrontal cortex comes from research indicating that damage to the orbitofrontal cortex results in disrupted performance on emotion-related EF but not on cognition-based EF tasks, and damage to the lateral prefrontal context generally affects performance on cognition-based EF tasks more than emotional-based tasks (see Kodituwakku et al., 2001b for a review).

Very few researchers have examined emotion-related or hot EF in FASD. Kodituwakku et al. (2001a), however, compared children and adolescents (aged seven to 19) with prenatal alcohol exposure with and without FAS (n = 20) with control children on an emotional-related learning task (visual discrimination reversal task) and a conceptual set-shifting task (WCST). On the visual discrimination reversal task, participants were shown two images, one of which was previously randomly chosen to be rewarding (emotional aspect). The participant had to figure out which was the rewarding and nonrewarding image by receiving feedback and gaining points for correct responses. After the participant reached the learning criterion, the reinforcement contingencies changed. An extinction condition was included in which both images were nonrewarding. Alcohol-exposed children were slower to reach the learning criterion, completed fewer reversals, and had more variability in extinction as compared with the control group. Group differences on reversal learning were still significant after controlling for performance on the WCST (conceptual set-shifting) and intelligence. Thus, Kodituwakku et al. (2001b) concluded that conceptual and emotional set-shifting may be independent functions in FASD, although the children display deficits in both functions. In concordance with previous research, there was no relation between cognitive functioning and facial dysmorphology in that both the alcohol-exposed group and the group with FAS performed equally poorly.

Frontal Cortex and FASD

Children and adolescents with prenatal alcohol exposure show a smaller brain size and abnormal brain shape, specifically in the frontal lobe and left hemisphere (Sowell et al., 2002). Wass et al. (2001) measured the size of the frontal cortex in the fetuses of 167 pregnant women between 12 and 42 weeks’ gestation. Seventy of the mothers drank moderate to large amounts of alcohol and the rest abstained. Alcohol exposure was related to a decrease in size of the frontal cortex, but not with other brain areas. Furthermore, 23% of fetuses with heavy alcohol exposure had a frontal cortex that was below the 10th percentile in size, versus only 4% of the control children. Mihalick et al. (2001) examined behavior as well as neuron numbers (after the animal was killed) in the medial prefrontal cortex in rats prenatally exposed to alcohol. The authors found that the rats were impaired on a test of reversal learning and showed difficulty with inhibition and transfer of learning. The rats also had a significant decrease in the number of neurons in the medial prefrontal cortex. Furthermore, Rikonen et al. (1999) found mild hyperperfusion (increased blood supply) to the right frontal region of the brain of individuals with FAS. The authors noted that this finding of asymmetry in perfusion to the frontal lobe has also been noted in children with attention deficit hyperactivity disorder, a disorder in which EF deficits have been well documented and is now being referred to as a disorder of EF (Brown, 2002). Thus it appears that prenatal alcohol exposure has a unique negative effect on the development of the frontal cortex; however, more research is needed in this
area to substantiate these claims and to better understand the mechanisms involved.

**WORKING MEMORY AND FASD**

One aspect of EF that appears to be particularly important is working memory. In one model, working memory is defined as a three-component system used for short-term storage and manipulation of information required for diverse cognitive tasks (Baddeley, 1992; Baddeley and Hitch, 1974). The visuospatial sketchpad is for holding and manipulating visual-spatial information, and the phonological loop is for maintaining and rehearsing verbal information (Baddeley, 1992). The central executive, an attentional controlling system, is involved in planning, selective attention, set shifting, and inhibition (Baddeley, 1996). The former two components are involved mainly in temporary storage of information, whereas the central executive involves storage of information but also other core aspects of EF such as inhibition, shifting, planning, and attention. Baddeley’s three-component model of working memory has been supported by many studies with adults and more recently in a large-scale study in children aged six to 15 years (Gathercole et al., 2004).

According to some views (Pennington et al., 1996), EF comprises two main processes, working memory and inhibition, and working memory is critical for successful performance on many EF tasks. In addition, strong relations exist between working memory and EF performance in children with brain injury (McDowell et al., 1997; Proctor et al., 2004). In this review, working memory is referred to as one EF process, but it also may be involved in many EF tasks.

There has been ample research indicating that individuals with FASD have memory deficits (Carmichael Olson et al., 1998; Mattson et al., 1996) and, more specifically, deficits in spatial memory (Kaemingk and Halverson, 2000; Uecker and Nadel, 1996). Despite these documented memory difficulties, researchers have not extensively evaluated the working memory abilities of children with FASD, although some researchers (Kodituwakku et al., 1995) have suggested that working memory is strongly implicated in FASD. Working memory measures have been included among other cognitive measures in many research studies on FASD, however. The most frequently used working memory measure is the digit span from the Weschler Intelligence Scales for Children (WISC-R or WISC-III). By aggregating results across these studies, we can learn about the working memory abilities in FASD and draw conclusions that will aid in our understanding of cognitive development in FASD.

**Working Memory in Children**

In a longitudinal prospective study, Streissguth et al. (1990) examined the relation between maternal alcohol use during pregnancy and cognitive functioning of offspring at seven years of age (n = 482). Using a partial least-squares analysis, they found that of all the WISC-III subtests, digit span (used to measure phonological working memory) was one of two subtests (the other being arithmetic) most related to prenatal alcohol consumption. Similarly, Jacobson et al. (1998) found that among children moderately exposed to alcohol, there were no significant relations between alcohol and IQ scores on the WISC-III, yet specific deficits were found on the digit span subtest. Carmichael Olson et al. (1998) found that adolescents with FAS (n = 9) recalled fewer digits on the forward and backward digit span tasks than control subjects of similar IQ levels. This deficit was even more pronounced on the backward digit, which is thought to be a measure of the central executive component of working memory and is thought to involve EF.

As mentioned earlier, Kodituwakku et al. (1995) found that children with FASD performed poorly on many but not all measures of EF. On examination of task demands, the authors concluded that the tasks that were particularly difficult for the FASD sample (those that differentiated the FASD sample from the control subjects) had a high working memory demand, particularly the central executive component of working memory. Thus, because of the high correlations among these tasks that that appear to have a strong working memory component, Kodituwakku et al. concluded that working memory might be the core deficit in FASD. In a review paper, O’Malley and Nanson (2002) also suggested that working memory deficits are common in FASD.

**Working Memory in Adults**

Adults with prenatal alcohol exposure also show working memory deficits. Kerns et al. (1997) found that adults with FAS (n = 16) performed below the norm on the consonant trigrams test. In this task, the participant hears three consonants and has to recall them after zero, nine, and 18 seconds, during which they must count backward to prohibit rehearsal. This task is thought to be a measure of frontal lobe functioning and, specifically, working memory (Stuss et al., 1982). The participants showed no deficits in performance when they did not have the distractor task of counting backward. However, they showed clear deficits (as compared with the norm) when the distractor task was added, indicating that the distractor task added extra demand on the central executive component of working memory. In a related view, Connor et al. (2000) found that adults with FASD (n = 30) performed lower than control subjects on the digit span and the consonant trigrams tests, and the latter test was found to be directly related to alcohol exposure in a path analysis.

**CONCLUSIONS**

Individuals with FASD exhibit deficits on many aspects of EF. These deficits have been documented in the areas of cognitive flexibility (trails task), inhibition (Stroop, WCST,
and possibly the tapping inhibition task), planning and strategy use (Tower tasks), verbal reasoning (word context task), set shifting (WCST), working memory measures, and recently, on tests of emotion-related or hot EF. Individuals with prenatal alcohol exposure are also found to be impaired on design fluency tasks and verbal fluency tasks, although the latter has had mixed findings in that all reports indicate deficits in letter fluency and only some indicate deficits in category fluency.

One important common finding is that in all studies in which researchers compared performance of individuals with full FAS with those with FAE or prenatal alcohol exposure (Connor et al., 2000; Kodituwakku et al., 2001b; Mattson et al., 1999; Schonfeld et al., 2001), EF deficits generally occurred to the same degree, regardless of whether the participant had facial characteristics of FAS. This finding indicates that alcohol damage on some cognitive functions may be just as severe for those with FAE as those with FAS, and it highlights the importance of understanding the CNS deficits that these individuals display. It is also important to note that in some studies (Connor et al., 2000; Kerns et al., 1997), the EF deficits were lower than what would be predicted on the basis of the participant’s IQ, and in other studies (Carmichael Olson et al., 1998; Kodituwakku et al., 2001b; Noland et al., 2003; Schonfeld et al., 2001) EF deficits were still evident after controlling for IQ. Thus, the EF deficits found in FASD cannot be solely attributed to a low IQ.

Still, it is important that more research be conducted on EF in FASD to fully understand the EF profile of these individuals, as the past research has been subject to some flaws. One common problem in much of the research presented is the small sample size (between nine and 20 participants in the FASD group in most EF studies), which can limit the generalizability of the findings and constrains the examination individual differences. Another potential problem is that in most child or adolescent studies, a very large age range is used (usually from about eight to 18 years), with very few participants at each age level. Thus no age-related comparisons can be made, and not much is learned about the development of EF in FASD. This problem, which is less evident in studies with adults, only allows for gross comparisons between exposed and control groups collapsing across age, and thus masks any potentially interesting relations that may exist between EF and age. Studies with a large sample at each age level that would allow for age-related comparisons are imperative in light of evidence that EF undergoes important developments from preschool to adolescence (Zalazo and Muller, 2002). The important changes that occur in the preschool years emphasize the need for more research on EF development of preschool children with FASD. Furthermore, power was not reported in many of the studies, and different studies used different methods for diagnosing FASD, which may compromise comparisons between studies. Thus, it is important that clinicians use similar diagnostic approaches when diagnosing those with prenatal alcohol exposure so more direct comparisons can be made between studies.

Another problem in EF research is the multiplicity of definitions and measures of EF, with the consequence that EF has become a very broad umbrella and somewhat ambiguous term for a multitude of cognitive abilities. Because of this variability, there appears to be a lack of a theoretical framework of EF development to guide the research. (For a review of issues related to defining and measuring EF, see Eslinger, 1996). It is important that researchers construct a common and accurate definition of EF, which will probably lead to a more theory-driven research in this area. Because there are so many different ways to measure EF, this can lead to contrasting results across studies and even within studies. For instance, individuals with FASD have been found to have difficulties on tests involving inhibition (WCST and Stroop tasks), but Kodituwakku et al. (1995) did not find evidence for inhibition deficits using other measures of inhibition (Go-No-Go). Furthermore, it is not clear whether the children in the Noland et al. (2003) study actually showed inhibition deficits or difficulty learning the task rules. Thus, because many tasks are thought to measure inhibition, there are some contrasting results, which could reflect real differences in inhibition across age or simply differences related to the other demands of the selected tasks.

Even within one task (e.g., WCST), there are many different scores that can be used to measure performance. In one study (Connor et al., 2000), for example, nine EF tests were administered and 58 different scores were calculated and analyzed, thus possibly increasing the type I error rate. It was found that some scores of a particular test were directly related to alcohol exposure, whereas other scores from the same test were not directly related. Thus, it is important that researchers come to a common ground on a definition and theory of EF, measures of EF, and the use of specific scores from EF measures to enhance the reliability and validity of research in this area.

Based on the small amount of research available, it is evident that individuals with FASD display deficits in working memory. In fact, Jacobson and Jacobson (1999) suggested that working memory deficits are characteristic of children with prenatal alcohol exposure. Deficits on forward and backward digit span have been documented in studies with children and adults with FASD, and adults have been impaired on the consonant trigrams test. However, the few studies conducted on working memory in FASD have only been with three measures of working memory. The forward digit span is used to measure the phonological loop, and the backward digit span and consonant trigrams test are used to measure the central executive. No research has been conducted with any measures of visual-spatial working memory in FASD. Examining performance of individuals with FASD on measures of visual-spatial working memory is important, considering evidence that individuals with FASD have specific deficits in spatial
memory (Kaemingk and Halverson, 2000; Uecker and Nadel, 1996). It may be that like spatial memory, visual-spatial working memory may be particularly difficult for individuals with FASD. Thus, to draw more reliable conclusions, future research on working memory in FASD is important due to the sparse existing literature and research on visual-spatial working memory in FASD is imperative. Furthermore, to fully understand the working memory profile of individuals with FASD, research using a number of measures of each component of working memory would be valuable.

Although they did not measure working memory directly, Kodituwakku et al. (1995) concluded that deficits in working memory might be the underlying cognitive mechanism resulting in poor performance on EF tests. Working memory appears to be particularly important in FASD and might be critical for successful EF. Thus, research on working memory in relation to other measures of EF is important. If working memory is found to be a critical aspect of the EF deficits common in FASD, this finding would have strong implications for the selection of assessment tools to diagnose FASD and for remediation.

CONCLUSIONS

In conclusion, it is evident that individuals with FASD display many deficits in the area of EF. These EF deficits can lead to life-long difficulties adapting to and functioning in society. Specifically, EF difficulties can result in problems with planning, organizing, and learning from past mistakes (Moore and Green, 2005). Lack of sufficient inhibitory control combined with not understanding the consequences of their actions (cause and effect reasoning) could lead to devastating problems with the law. In fact, in one study of individuals with FASD, it was found that 60% of the sample had been in trouble with the law and 50% had been confined (see Streissguth, 1997). Furthermore, in a Canadian study it was found that 23% of youth remanded for a psychiatric inpatient assessment had FASD (Conry et al., 1999).

Despite the prevalent EF deficits in individuals with FASD and the potentially devastating life-long consequences they face functioning in society, there has been little if any research examining how to deal with EF deficits in terms of improvements, treatment, and upbringing. Watson and Westby (2003) wisely note that EF deficits are common in many disorders including attention deficit hyperactivity disorder, autism, learning disabilities, traumatic brain injury, as well as children prenatally exposed to other drugs. Thus, much can be learned if the intervention research with other populations can be applied to individuals with FASD. Using Barkley’s model of EF, Watson and Westby (2003) suggest various strategies for addressing EF deficits in children prenatally exposed to alcohol and other drugs. For example, for difficulties with nonverbal working memory (remembering events and information, behaviors, and so forth) they suggest using visualization techniques including self-awareness training, consistent and structured environments and routines, as well as visual aids and checklists. For difficulties with self-directed speech and verbal working memory, they suggest language intervention, cognitive-behavioral intervention, and linking visual cues to verbal prompts. The authors further recommend social skills training, role-playing, social stories, and teaching the vocabulary of emotions, to deal with problems with self-regulation. Finally, for difficulties with problem solving, they suggest cognitive modeling, coaching, and self-determination curricula.

There are some strategies suggested for EF deficits in children with FASD; still, some strategies can be taken from other areas of research, and yet some strategies simply intuitively make sense as appropriate for those with EF deficits (e.g., structure, limiting information, and so forth). However, the literature is lacking in research evaluating the effectiveness of these EF strategies, the efficacy of intervention programs for individuals with FASD, as well as research on whether EF skills can actually be improved in those with FASD. Research in this area is essential to improve the overall functioning of individuals with FASD as well as to minimize trouble with the law.

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