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Verbal and Nonverbal Memory in Adults Prenatally Exposed to Alcohol

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Abstract

Background—Neurocognitive effects of prenatal alcohol exposure in adulthood are not well documented. Questions persist regarding the extent to which there are specific, measurable effects beyond those associated with global ability deficits, whether individuals without the full fetal alcohol syndrome (FAS) demonstrate alcohol-related cognitive impairments and whether observed memory effects are specific to a particular modality, that is, verbal versus visual/spatial domains.

Methods—In this study, verbal and nonverbal selective reminding paradigms were used to assess memory function in 234 young adults (M age: 22.78, SD: 1.79). Alcohol-exposure was quantified prenatally. Alcohol groups included: Individuals with physical effects of alcohol exposure (Dysmorphic Group, n=47); Exposed individuals without such effects (n=74). Contrast groups included: Controls (n=59) matched for ethnicity, socioeconomic status and hospital of birth; Special Education contrast group (n=54) included to control for disability status. Memory outcomes entailed total recall, delayed recall, and measures of encoding and retrieval and learning over trials as indexed by slope.

Results—Results indicated that Dysmorphic individuals were significantly less efficient in memory performance than Controls on all of the outcomes measured but they did not differ from those in the Special Education contrast group. The non-dysmorphic, alcohol-exposed group was intermediate in their performance, suggesting a continuum of effects of prenatal exposure. Evaluation of the encoding and retrieval aspects of memory performance indicated that learning rather than forgetting accounted for the deficits associated with prenatal alcohol exposure. Finally, no interaction was found between modality of presentation (verbal and nonverbal) and effects of alcohol exposure on memory performance.

Conclusion—These findings indicate that prenatal alcohol exposure is associated with persistent and specific effects on memory performance and that these problems result from less efficient encoding of information across both verbal and nonverbal modalities. Education and training efforts with this clinical group should take these characteristics into account.

Fetal alcohol syndrome (FAS) and the spectrum of associated disorders that result from maternal alcohol use during gestation (i.e., fetal alcohol spectrum disorders, FASD) are among the most common developmental disorders (Centers for Disease Control and

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Prevention, 2009). FASD affects physical, cognitive, psychological, and social development of individuals across the lifespan (see Jacobson & Jacobson, 2002; Kable & Coles, 2004; Mattson & Riley, 1998 for reviews). Despite the frequency of this disorder and its widespread impact on affected individuals, information about the nature of associated cognitive deficits in adults is not extensive. Young adults should demonstrate the most efficient performance in cognition and our understanding of functioning at this time point would provide a basis for interpretation of any future cognitive declines in this group. In this study, we investigate the effects of prenatal exposure on aspects of memory functioning. Memory is a sensitive indicator of cognitive efficiency and this basic aspect of cognition is frequently affected in individuals who have neurological or neurodevelopmental deficits. Although there has been some previous work on memory function in alcohol-exposed individuals, many of these studies have been conducted with children, and these are often children drawn from clinical samples with control groups that are not equivalent and in some cases, not used (e.g., Pei, Rinaldi, Rasmussen, Massey, & Massey, 2008). Thus, a number of questions remain regarding adult memory function.

Memory deficits associated with prenatal alcohol exposure have been observed in animal models (e.g., Becker, Randall, Salo, Saulnier, & Weathersby, 1994; Driscoll, Streissguth, & Riley, 1990), in studies drawn from clinical samples (e.g., Mattson, et al, 1998; Pei, Rinaldi, Rasmussen, Massey, & Massey, 2008; Willoughby, et al., 2008), and in exposure cohorts (e.g., Coles, et al., 1997 Richardson, et al., 2002; Streissguth, et al., 1989) However, there remain some questions about the basis for these deficits. A possibility that is always raised is that memory problems result from the global performance deficit that is characteristic of alcohol-affected individuals. In clinical studies, memory performance has been associated with IQ in prenatally exposed children (Kaemingk & Halverson, 2000; Kaemingk, Mulvaney, & Halverson, 2003; Mattson et al., 1998), but IQ differences alone have not explained observed impairments in memory and learning (Roebuck-Spencer & Mattson, 2004). In one of the only studies to assess memory functioning in adults with FAS, Kerns, et al. (1997) compared alcohol-affected adults who had either average or below average ability level (i.e., IQ scores). Results indicated that, in comparison to normative samples (no control group was used), adults in both groups showed deficits in initial and final recall, although those with below average IQ scores showed more marked impairments. However, the lack of a contrast group makes it difficult to establish a strong case for specific cognitive and memory effects given that the results may be mediated by the IQ differences.

A related issue is whether cognitive, and specifically memory deficits, are similar in alcohol-exposed individuals with and without the characteristic physical effects, that is to say, whether there is a spectrum of effects on memory or whether significant effects are seen only in the most severely affected. Many clinically-based studies suggest that individuals with alcohol-related neurodevelopmental disorders (ARND) and behavior disorders attributed to alcohol will show similar levels of cognitive impairment to those meeting criteria for FAS (e.g., Green, et al., 2009; Mattson, et al., 1996; Rasmussen & Bisanz, 2009). In a sample with only moderate exposure, Willford et al. (2004) found memory effects among adolescents, a result that argues that these effects can be observed in those not meeting criteria for FAS. Finally, there is a question about the degree to which performance deficits in alcohol-exposed individuals are unique or similar to those in other disability groups. If unique, patterns of deficit could be used diagnostically; if similar to deficits in other groups, then the goal of a behavioral phenotype associated with prenatal alcohol exposure (see, Kodituwakku, 2007; Rasmussen & Bisanz, 2009) is more difficult to achieve.

Some studies have evaluated whether observed memory deficits can be attributed to the initial learning of information (that is, encoding) or to a problem in retrieval of previously learned information (that is, long term recall), a characteristic often reported clinically in

FASD. The preponderance of evidence suggests that the deficit occurs during encoding. In the Kerns et al. (1997) study, both groups of alcohol affected adults showed impaired recognition performance, which indicated that alcohol exposure was associated with encoding deficits rather than problems with retrieval. This study is consistent with several others carried out with children that attributed recall performance to encoding deficits, or problems learning new information, rather than problems retrieving information already learned (Coles et al., 1997a; Mattson, et al., 1996; Mattson et al., 1998). In support of the encoding explanation, it is reported that both children and adults prenatally exposed to alcohol show differences from controls in the frequency and type of strategies used to encode information. (Kerns et al., 1997; Roebuck-Spencer & Mattson, 2004). In addition, differences in learning slopes have been reported, suggesting that non-exposed children learned information at a faster rate compared to those prenatally exposed to alcohol (e.g., Mattson & Roebuck, 2002). In their study, learning slope differences were task-dependent, and slower rates were observed on verbal but not nonverbal tasks.

Finally, it is unclear whether alcohol-related deficits are specific to a particular modality or functional area. Some authors suggest that nonverbal (e.g., visual-spatial) memory is more significantly affected than verbal memory; however, results of investigations of this question have been mixed. One study showed that once IQ was statistically controlled, adolescents with FAS showed poorer performance on measures of visual-spatial but not verbal recall (Olson, et al., 1998) and another found that children with FAS showed recall deficits for the location of objects but not the objects themselves (Uecker & Nadel, 1998). However, several studies have failed to find this pattern of performance differences between domains (Mattson & Roebuck, 2002; Pei et al., 2008) and verbal learning and memory deficits have been verified using a variety of tasks (Kaemingk, Mulvaney, & Halverson, 2003; Korkman, Kettunen, & Autti-Ramo, 2003; Streissguth et al., 1989; Willford, et al., 2004.) Thus the relationship between alcohol-exposure and modality-specific memory deficits requires further examination.

In the present paper, we investigated a number of these questions about the impact of alcohol exposure on memory performance in a young adult cohort. We examined the effect of ability level (IQ) and physical dysmorphia by comparing performance in groups of young adults who are similar in ability level but who vary in the degree to which they demonstrate alcohol-related dysmorphology. In doing so, we measured support for the assumption that, when prenatal alcohol exposure has occurred, both those who demonstrate physical dysmorphia and those who do not have a similar level of cognitive dysfunction. We hypothesized that alcohol-exposed adults demonstrating more physical effects of alcohol (dysmorphic features) would perform more poorly than those without such physical features and that the nondysmorphic, in turn, would perform more poorly than non-exposed controls. By also including a group identified as learning disabled (Special Education), we investigated whether there are deficits in learning and recall specific to the effects of alcohol exposure or whether both groups of individuals with disabilities perform similarly.

In addition, due to the characteristics of the memory tasks chosen, we were able to address several questions about the nature of the encoding, learning and retrieval deficits in alcohol-affected individuals. Learning and memory were assessed using the Verbal Selective Reminding Memory Test (VSRT) (Buschke and Fuld, 1974) and its nonverbal counterpart, the Nonverbal Selective Reminding Memory Test (NVSRT) (Fletcher, 1985). As discussed by Trahan and Larrabee (1993), Selective Reminding tasks assess encoding and learning rate and also measure forgetting by taking into account the theoretical differences between short (STM) and long term memory (LTM). On each trial of these list-learning tasks, the subject is reminded only of the items that were not recalled during the previous trial. By recording not only the items recalled during the preceding trial but noting those that are recalled

consistently without reminding, it is possible to discriminate long term storage (LTS) and Consistent Long Term Recall (CLTR) from more immediate memory (which is assessed in Total Recall, TR, which includes both STM and LTM). It is also possible to compare these measures of encoding efficiency to a Delayed Recall (DR) trial (after 30 minutes), allowing assessment of the degree to which forgetting is occurring of material that was effectively encoded. In this paper, by examining different types of memory outcomes (i.e., total recall, delayed recall, learning slope) we investigated which characteristics of memory performance (encoding or retrieval) are affected by prenatal exposure. We also compared learning in two different modalities, verbal and nonverbal, to determine whether there is a relationship between alcohol exposure and facility in a particular learning modality. It was hypothesized that the basis for memory deficits would be problems with encoding rather than with retrieval of information that was encoded into long-term storage. A final hypothesis was that nonverbal memory would be more affected by alcohol exposure than verbal memory.

Method

Participants

The sample includes 234 young adults participating in a longitudinal study of effects of prenatal alcohol exposure on development. Of the 234, 180 were born to mothers recruited between 1980 and 1986 from an urban hospital serving a predominantly African-American, low income population. Mothers were interviewed prenatally using a measure of quantity/frequency of alcohol use during pregnancy. Those who reported consuming at least one ounce of absolute alcohol per week (equivalent to two drinks) and those who reported consuming no alcohol while pregnant were invited to participate in the initial study. Amount of alcohol consumed by those who met criteria for inclusion as drinkers ranged from 1 to 75 ounces of absolute alcohol per week (oz/AA/wk) with an average of 10.3 oz/AA/wk (SD: 11.88), that is, more than 20 drinks a week. The mothers who drank during pregnancy were advised that it could have negative health consequences for the baby and that they should stop. Those who agreed were provided with referrals to treatment programs. Infants were evaluated after birth and have been evaluated periodically since that time. At the postnatal examination, infants were assessed for growth patterns and presence of dysmorphic physical features related to prenatal alcohol exposure (Coles, et al., 1985). In evaluations completed at seven years of age, at mid-adolescence, and as part of the protocol for the current young adult follow-up, participants were assessed for physical effects of alcohol exposure using the Dysmorphia Checklist (Coles, et al. 1997b). This Checklist, which was developed for use in the initial infancy study, is similar to that used by other investigators (Jones, et al. 2006; Hoyme et al, 2005). It was administered without knowledge of the participants' alcohol exposure status by a pediatric geneticist or a nurse trained by this geneticist and included a weighted list of 30 physical characteristics associated with prenatal alcohol exposure. Those that are considered to be sentinel features of FAS (e.g., absent/indistinct philtrum; short palpebral fissures) are weighted as "3" while other characteristics that are observed in FASD may be weighted as 2 (e.g., ptosis, hypoplastic mandible) or 1 (e.g., clinodactyly). Weighted scores are then summed to yield a total score. Validity of this measure has been measured by correlation with alcohol use levels reported by mothers (Coles, et al. 1997) and reliability by test-retest assessments in a clinical setting (Blackston, et al., 2004). This measure has been used consistently throughout this longitudinal study. At each time point, participants also completed tests of intellectual ability (Wechsler, 1991; Wechsler, 1999). In young adulthood, participants completed a neuropsychological evaluation including the verbal and spatial memory tasks analyzed here.

In early adolescence, a contrast group of 84 participants with similar demographic characteristics was recruited from Special Education programs to serve as a control for the effects of disability status on social and academic outcomes. Thus, the sample in this paper

includes four groups of participants: 1) Control (n=59): Participants whose mothers did not consume alcohol during pregnancy; 2) Alcohol Exposed (EtOH, n = 74): Participants who were exposed to alcohol prenatally, but whose dysmorphia scores, collected on three occasions before this adult assessment, were less than one standard deviation above the mean of the whole sample; (3) Dysmorphic (DYSM; n =47): Participants who were exposed to alcohol prenatally and received a dysmorphia score at least one standard deviation above the mean of the sample; and 4) Special Education (SpecED, n=54): Participants received special education services in one of three public school systems in the Atlanta, Georgia Metropolitan area. Demographic and academic information about this group has been reported previously (please see, Howell, et al, 2006). For the current study, 54 of these individuals (64.3%) were reassessed.

Procedure

Young adults were contacted by mail or phone concerning the follow-up study. These young people were eligible for follow-up because of their parents' previous involvement; however, this was the first point in time in which they were able to choose themselves whether to continue their participation. Of the 317 who could be located, 234 (73.8 %) consented to participate in this phase of the study. Participants were significantly more likely to be female than were nonparticipants ($X^2_{(1)} = 18.3, p = .000$). From the Special Education group, 64.3% volunteered and they did not differ from non-participants on ethnicity, full scale IQ, or income; however, those returning were more likely to be female and slightly older. Of those identified at birth, 110 (25.8%) could not be located and no further information is available about them. Of those who could be located, 7 were deceased (2.2%), 23 had moved out of State (7.3%), and 19 (6%) were temporarily unavailable (i.e., in college, in the military, in prison). Only 33 who were located and available refused to participate (10.4%). Participants in the adult follow-up were compared to all nonparticipants on demographic and background variables, with no differences found in group status (alcohol-exposed or control), amount of alcohol reported by mother in pregnancy, age, ethnicity, income at birth or adolescence, gestational age, birth weight, birth head circumference, or previous IQ; however, those participating in the adult follow-up had higher dysmorphia scores at birth ($t_{(210)} = -2.5, p = .012$) than did nonparticipants.

Those who were interested in participating completed a consent procedure in which the study goals and procedures were explained; consent forms approved by the Emory University School of Medicine Institutional Review Board were signed. During this process, the confidentiality of participants' mothers was protected; that is, no information about mother's alcohol or other substance use during the pregnancy of the now-adult child was revealed. For the evaluation visit, participants were picked up by project outreach workers and transported to the laboratory for a day-long evaluation which included the neuropsychological assessment, a medical evaluation, and an interview session. In addition to transportation, participants received lunch and compensation for their time and effort.

Measures

Assessment of Learning and Memory—In the current study, different modalities were evaluated using similar recall tasks. There are 8 learning trials for both the VSRT and the NVSRT. For the verbal task, we used Form 2 of the VSRT in which the individual is required to recall a list of 12 unrelated words that are orally presented at the rate of 1 word every 2 seconds (The words are: Shine, Disagree, Fat, Wealthy, Drunk, Pin, Grass, Moon, Prepare, Prize, Duck, Leaf). After the first recall trial, the person is read, in the same order that they were initially presented, only those words that were not recalled on the last trial. The person is then instructed to recall all the words, including those they recalled on the previous trial. On subsequent trials, the same administration occurs, with reminders given

only of the words that were not recalled on the last trial, and the person is asked to say all the words on the list. In an analogous procedure, the NVSRT consists of an array of 8 different dot patterns, each arranged in a separate box (see Figure 1). The examiner points one at a time to a targeted dot in each of the boxes, and, at recall, the person is required to point to the targeted dot in each of the 8 boxes. After the first trial, the examiner points, in the same order that they were initially presented, to boxes where a dot placement was not recalled, and the person is asked again to locate the correct target dot in all of the 8 boxes, including those that did not require reminders. After a 30 minute delay, the participant is asked to recall the stimulus items. These Selective Reminding measures were embedded in a longer battery of neuropsychological measures and were not presented sequentially.

There are a number of dependent variables for both tasks as listed below and described in more detail in Table 1. These include total correct recall (TR) of words and dots, which is the same outcome used on most “list-learning” memory procedures; long term storage (LTS): number of words or dots recalled without reminding on at least two consecutive trials; consistent long-term retrieval (CLTR): number of words or target dots recalled without reminding on at least two consecutive trials and then consistently to the end of Trial 8; and delayed recall (DR): the number of words or target dots recalled, without pre-warning, after 30 minutes.

To capture any difference in the efficiency of the learning process we also calculated the “slope” of the line for recall performance.¹ To differentiate short term memory from long term storage, we examined CLTR and LTS. Finally, to evaluate forgetting, we subtracted DR from the final learning trial (Trial 8) for TR, CLTR and LTS, creating three measures of items forgotten for each test. This permitted us to differentiate the learning process (encoding) from later retrieval of the information.

Assessment of Ability—Ability (that is, IQ) was assessed using the Wechsler Abbreviated Scale of Intelligence (WASI; Wechsler, 1999). This is a short form of the most widely used measure of adult ability and demonstrates a high correlation with longer versions of this test. The WASI was administered as part of a larger neuropsychological battery by a psychologist or graduate student trained in assessment who was blind to alcohol group status.

Medical Evaluation. Dysmorphia Exam—A nursing examination was carried out for each participant. This included a screen of hearing and vision, a medical history, as well as completion of the Dysmorphia Checklist for this adult visit. Nurses carrying out the examination were trained by a geneticist familiar with the signs associated with prenatal alcohol exposure.

Alcohol Use—Abusive alcohol use is often associated with memory deficits. For this reason, adult participants were interviewed regarding their current alcohol and other drug use. They completed the Addiction Severity Index (ASI; McLellan et al, 1992) and the Drug Checklist (Coles, et al., 1992). Information about alcohol use included a quantity/frequency measure that allowed calculation of ounces of absolute alcohol used each week (oz/AA/wk) as well as information about alcohol use history. In addition, urine samples were requested to screen for drug use and blood samples were collected to evaluate the effects of alcohol on liver function. Laboratory tests were used to confirm self report.

¹Slopes were calculated using the formula $(\text{mean}\{TR\} = \text{mean}\{TR\}_1 \text{ to } \{TR\}_8; \text{covariate}\{TR\} = -7*({TR}_1 - m\{TR\}) - 5*({TR}_2 - m\{TR\}) - 3*({TR}_3 - m\{TR\}) - 1*({TR}_4 - m\{TR\}) + 1*({TR}_5 + 3*({TR}_6 - m\{TR\}) + 5*({TR}_7 - m\{TR\}) + 7*({TR}_8 - m\{TR\}))$ with $\text{Slope} = \text{covariate}\{TR\}/168$).

Archival records—Information was obtained from archival records regarding maternal drug and alcohol use and results of previous measurements of participants' ability and physical effects of alcohol (i.e., dysmorphia checklist scores). These previous measures were used to categorize the groups for recruitment.

Results

Characteristics of the Study Sample

The demographic, physical and exposure characteristics of the study participants are shown in Table 2. There were no significant group differences in gender, income or education. There were significantly fewer African-Americans in the Special Education group. On the factors that characterize the effects of alcohol (dysmorphic features, ability level, and growth), the DYSM group was significantly different from the other three groups who did not differ. Maternal prenatal alcohol and other substance use were greater in both the alcohol-exposed groups. Recruitment occurred before the cocaine "epidemic" of the late 1980's so the level and frequency of such use in this sample was low. Young adults in the Special Education and the Exposed but not Dysmorphic groups reported higher levels of current alcohol use than those in the Control group. However, these rates of reported use were low overall particularly for this age group. To rule out any effects on memory function of current alcohol use by these young adults, their reported ozAA/wk (Table 2) was correlated with all of the outcomes variables. No significant correlations were found and, therefore, current alcohol use was not included in further analyses.

Memory

Memory was evaluated using the Verbal and Nonverbal Selective Reminding Tasks. We used five dependent variables for the Verbal and Nonverbal Selective Reminding tasks: Total Recall, Consistent Long-Term Recall, Long Term Storage, Delayed Recall, and Learning over trials (see Measures Section and Table 2 for a detailed description). To differentiate short term memory from long term storage, we examined CLTR and LTS. Finally, to evaluate forgetting, we subtracted DR from the final learning trial (Trial 8) for TR, CLTR and LTS, creating measures of items forgotten. In the initial analysis of data, gender was used as a factor. While there were some significant differences in outcomes with males performing better on nonverbal tasks and females on verbal tasks, there was no interaction with alcohol exposure group on any of these measures. Therefore, subsequent analyses collapsed across this factor.

Learning and Recall

The outcome variables for the Verbal and Nonverbal tests were analyzed using multivariate analysis of variance (MANOVA) with Exposure group (4) as the independent factor. In addition to Mean Total Recall, the 8th learning trial was selected as representing the "best" immediate performance on the three recall measures. Two separate MANOVAs were done as one fewer subject completed the Nonverbal task due to a motor disability. In addition, separate analyses were carried out for Delayed recall, since there were several fewer subjects for this variable, and Learning Slope. Subject loss was due to physical disability, fatigue and experimenter error and was unrelated to exposure group. Tukey's Honestly Significant Difference (Tukey HSD) procedures (Barnett, & McLean, 1998) were performed *post hoc* to identify group differences. Results are shown in Table 3. For the Verbal measure, all comparisons (Means, Standard Deviations, and Statistics are shown in Table 3) resulted in significant differences in performance with the Control group performing significantly better than all other groups. In addition, the Alcohol Exposed/Nondysmorphic group performed significantly better than both the Dysmorphic and Special Education groups on Trial 8 TR and CLTR and than the Special Education group alone on Mean Recall

and Trial 8 for LTS. For Delayed Verbal Recall, the Control and Alcohol Exposed groups did not differ and were both significantly higher than the Dysmorphic and Special Education groups. For the Nonverbal measure, Mean Recall and Recall at trial 8 for CLTR and LTS showed significant differences in performance with the Control group having higher scores than all other groups. On trial 8 of TR, the Control group performed better than both the Dysmorphic group and the Special Education group but not the Alcohol Exposed group. For Delayed recall, the overall F statistic was not significant but using planned comparisons, the Control group was significantly higher than both the Dysmorphic ($p < .02$) and the Special Education ($p < .05$) groups.

Both multivariate procedures were repeated with WASI Full Scale IQ (FSIQ) (Wechsler, 1999) included as a covariate (MANCOVA). In both analyses, FSIQ contributed significant variance to memory performance (For Mean Verbal task measures and FSIQ: TR: $F_{(1, 225)} = 50.15$; $p < .000$; Trial 8 TR: $F_{(1, 225)} = 41.01$; $p < .000$; Trial 8 LTS: $F_{(1, 225)} = 40.44$; $p < .000$; Trial 8 CLTR: $F_{(1, 225)} = 34.48$; $p < .000$); However, the pattern of significance was unchanged except that for Mean TR, Control was no longer significantly higher than the Exposed/Nondysmorphic group with IQ controlled. (For Mean Nonverbal task measures and FSIQ: Mean TR: $F_{(1, 224)} = 45.12$; $p < .000$; Trial 8 TR: $F_{(1, 225)} = 48.71$; $p < .000$; Trial 8 LTS: $F_{(1, 225)} = 43.11$; $p < .000$; Trial 8 CLTR: $F_{(1, 225)} = 38.04$; $p < .000$), but the addition of this variable did not change the pattern of previous results with the exception of LTS Trial 8 for the Nonverbal scale, which lost significance ($F_{(1, 225)} = 2.15$; $p = .095$).

Using the analysis of variance procedure (ANOVA), Total Delay scores for verbal and nonverbal tasks were also examined controlling for IQ (Verbal DR: FSIQ: $F_{(1, 223)} = 27.67$; $p < .000$; Group: $F_{(3, 223)} = 5.14$; $p < .002$). This analysis found that the Control group remained significantly greater than the Dysmorphic and Special Education groups but not the Alcohol Exposed group. For the Nonverbal DR: FSIQ $F_{(1, 222)} = 34.73$; $p < .000$; Group: $F_{(3, 222)} = 1.77$; ns. As with the previous analyses, while it contributed significant variance, the addition of FSIQ did not change the pattern of memory effects shown in Table 2. Planned Contrasts were done for each analysis of DR which compared the Control group to each of the other groups. For Verbal Delay, the Control group's performance was significantly higher than the Dysmorphic Group ($p < .01$) and the Special Education group ($p < .000$). For the Nonverbal Delay, the Control Group's performance was significantly higher than only the Special Education group ($p < .02$).

Learning Slope—Figure 2 shows the learning slopes for TR for each group for each test. To facilitate analysis of these patterns, as noted above, the slope for the total recall variable was calculated for each participant and used as the dependent variable in a MANOVA procedure with Exposure group as the independent variable. This learning slope was significantly different by group for Verbal but not Nonverbal memory although a trend was noted (Table 3). Planned comparisons indicated that, for the Verbal task, the mean for the Control group was significantly greater than those of both the Dysmorphic and Special Education Groups; the mean for the Alcohol Exposed group was significantly greater than that for the Dysmorphic group. For the Nonverbal task, the Control group performed significantly better than the Dysmorphic Group.

Modality—To determine whether Modality of learning (Verbal versus Nonverbal task) affected the rate of learning, a repeated measures analysis of variance was done using the transformed z-scores for each of the TR trials for each test. Z-scores based on the whole group were used to control for the difference in the number of items in the memory tasks. The first within subject factor (8 levels) was Trial and the second within subject factor (2 levels) was Modality: (Verbal and Nonverbal task). This design allowed assessment of both learning over the 8 trials and the effect of modality by Group. There was a significant

difference among Groups with the Control group performing better than all other groups ($F_{(3, 223)} = 7.47, p < .000$) (see Figure 2). There were no significant within-group main effects for Trial or Modality. There was a Group by Trial interaction ($F_{(21, 1561)} = 2.04, p < .004$) reflecting a linear trend for the Control group to demonstrate accelerated memory performance over trials relative to the other groups. There was no significant Modality by Group interaction ($F_{(1, 223)} = 1.18, ns$), indicating that there is no significant difference in performance on total recall for group as a function of task type. Finally, there was no three-way interaction of these factors. To examine the effect of ability level on these factors, this analysis was recalculated with Full Scale IQ included as a covariate. Although IQ interacted significantly with Trial ($F_{(7, 1561)} = 3.07, p < .003$) indicating that those with higher IQs recalled more over time, it did not interact with Modality ($F < 1$) and the significant Group differences remained.

Similar analyses were done for LTR, CLTR and Delayed recall with similar results. There were no effects of modality and no interaction of modality with group.

Encoding and Retrieval—To discriminate learning effects from forgetting or retrieval, we created three “forgetting” indices by subtracting DR from trial 8 scores for TR, CLTR and LTS for each subject. This procedure was consistent with the recommendations of Trahan and Larrabee (1993) who suggest that TR at trial 8 represents a combination of both long and short term memory and thus it may exaggerate the amount actually “learned” while DR represents the amount that can be retrieved from long term memory. LTS is believed to represent the material that has been encoded into long term memory while CLTR reflects the material that has been encoded and can be consistently retrieved during the learning process. Therefore it might be expected that LTS and CLTR would reflect the material that could be retrieved at DR. Thus, discrepancies between these two outcomes and DR would reflect “forgetting” rather than encoding deficits. When all these “forgetting” measures were analyzed with Group as the independent variable, there were no differences on any of these measures, in either modality, with all groups showing the same patterns of forgetting. Verbal: TR-DR: $F_{(3, 225)} < 1, ns$; LTS-DR: $F_{(3, 225)} < 1, ns$; CLTR-DR: $F_{(3, 225)} = 1.85, ns$; Non Verbal: TR-DR: $F_{(3, 225)} < 1, ns$; LTS-DR: $F_{(3, 225)} < 1, ns$; CLTR-DR: $F_{(3, 225)} < 1, ns$.

Discussion

This research explored effects of prenatal alcohol exposure on memory in young adults, an age group that has received very little attention previously. While there are a number of discussions in the existing research and clinical literature about the impact of alcohol exposure on learning and memory in children, memory function is not the same during the process of development as it is in young adulthood when it can be expected to be most efficient. These results suggest that academic and adaptive demands that rely on the expectation of effective memory performance may need to be altered for alcohol-affected individuals.

In this study, it was the intention to test several hypotheses. The first general hypothesis was that there is an effect of prenatal alcohol exposure on memory functioning, specifically on encoding and recall. The corollary was that this effect on encoding and recall would be more significant in individuals who exhibit the physical effects of their exposure. The results of this assessment, as shown in Table 3, suggest that these assumptions are accurate. Compared to unexposed controls, both groups of alcohol-exposed individuals had more difficulty in performing efficiently on these tasks and this effect was more apparent in the Dysmorphic group than in the group who were exposed but not physically affected although the pattern of results suggests a continuum of effects of exposure. That is, the alcohol-exposed but not dysmorphic group was usually intermediate between the Control and Dysmorphic groups.

This result is made more interesting when it is noted that the IQ range for the Control and the Nondysmorphic groups is the same. It seems evident that there are some individuals in the Exposed/Nondysmorphic group who are affected by their exposure and some who do not demonstrate any memory problems.

To address the suggestion that cognitive deficits observed in alcohol-affected individuals are simply secondary to global delays (that is, ability differences), the results of the study were analyzed controlling for FSIQ with the finding that while FSIQ makes a significant contribution to performance, it does not account for the alcohol-related deficits in encoding and recall observed here. This finding is consistent with previous studies (Kerns, et al., 1997; Roebuck-Spencer & Mattson, 2004) and indicates that there is a specific impact of alcohol on memory over and above that caused by global deficits although the two effects are probably additive.

Another issue addressed in this research was whether memory deficits observed in alcohol-affected individuals are phenotypically different from those observed in individuals with learning deficits that can be attributed to different etiologies. If there are unique patterns of deficit in FASD, such behavioral differences could be used diagnostically to identify individuals who have been alcohol exposed even when maternal alcohol use could not be confirmed. Based on these results, in which alcohol-affected young adults with documented exposure were compared to unexposed young adults who qualified in childhood for special education services, there is limited evidence to argue that the encoding and recall deficits seen in FASD are discriminable from those found in others with identified learning disorders. The level and pattern of impairment appears to be similar for both disability groups. Thus, alcohol exposure may be one of many conditions that, by affecting neurodevelopment, lead to significant learning disabilities.

Based both on previous research with children by other investigators (Kaemingk, Mulvaney & Halverson, 2003; Mattson et al., 1996; Mattson et al., 1998) and our own observations concerning the learning problems associated with prenatal alcohol exposure (i.e., Coles, et al, 1997; Coles, et al., 2007), we anticipated that the problems with recall shown by alcohol-affected individuals would be based on encoding rather than retrieval even though it is often reported by parents and teachers that children with FASD “know something one day and forget it the next”. Using the paradigms employed in the current study, which are designed to discriminate learning and retrieval effects, it appears that “memory” problems are, in fact, *learning* problems and that alcohol-affected individuals experience difficulty in encoding efficiently. There was no support for the suggestion that deficits resulted from forgetting previously learned material. However, the paradigms used in this study require recall at a delay of only 30 minutes. Therefore, it is possible that an assessment a day later or a week later might show a different pattern of results.

A potential weakness of our study is that we did not assess cued recall or recognition memory. At the time of this investigation, we were not aware of cued recall and recognition procedures available for the NVSRT. Since one purpose of our paper was to compare performance on the verbal and nonverbal memory measures, we felt it necessary to use similar indices. The indices used in this paper were selected based on the logic presented in the paper by Trahan and Larrabee (1993) describing the Selective Reminding procedure. In addition, a study by Beatty et al. (1996) using the VSRT in patients with multiple sclerosis supported the concept that LTS is sensitive to storage deficits, whereas CLTR is sensitive to retrieval. Nevertheless, we acknowledge that information about performance on recognition could have provided additional support for an encoding versus a retrieval deficit. It is well known that retrieval is dependent on effective encoding of information and, when encoding is impaired, it can be difficult to access information even if it has been recalled previously.

There are limits to the “list learning” approach for the understanding of memory function. To understand the pattern of alcohol-related deficits in memory and their adaptive implications, a more comprehensive assessment of the various aspects of memory may be warranted.

There is a good deal of discussion in the FASD research literature regarding relative deficits in visual-spatial versus verbal processing, some of it contributed by the authors of this paper (e.g., Coles, et al., 2002). In the current study of encoding and recall, we did not find support for the suggestion that verbal memory is less affected than nonverbal. Indeed, examination of the data would suggest the opposite. The group differences in recall were more clearly observed on the Verbal task both during the process of encoding and at delayed recall although a direct analysis of modality of learning was not statistically significant and performance, overall, was better on the nonverbal task. These findings may be influenced by the characteristics of the tests used in the study. The verbal task may have proved more of an opportunity for strategy use than the current nonverbal paradigm. However, the verbal list was not constructed to facilitate verbal strategy use and this possibility could not be assessed in the current study. The extent to which strategy use can be employed intentionally to improve memory function in this clinical group as well as the extent to which there are developmental changes in strategy use over development would be appropriate next steps in understanding memory function in FASD.

Like other research results, generalization from the findings this study may be limited by the characteristics of the research design, discussed above, and of the participants. All are of low socio-economic status (SES), or at least were so at the time of initial ascertainment, and social factors (e.g., academic experience) may have influenced performance. In addition, as in all human studies of prenatal exposures, there may be other group differences besides alcohol exposure that affect learning and memory. These may include both other exposures as well as potential differences in caregiving environment in families in which substance abuse is a problem. In this cohort, unlike many of the clinic-based studies of FASD, few of the young adult participants experienced foster care or adoption. Finally, there may be a self-selection bias present as some of the individuals in this longitudinal cohort were available and willing to return for this young adult assessment while others were not for a variety of reasons (e.g., armed services, college, incarceration, death). How such a selection bias might affect memory function cannot be ascertained fully. However, the lack of significant differences on the many participant characteristics that could be measured suggests that these data may be representative of the whole cohort.

Conclusion

Memory deficits were observed in alcohol-affected young adults. The study was designed to allow the comparison of verbal to nonverbal memory function and to evaluate the contribution of encoding and retrieval to performance. Results indicated that the encoding aspect of memory, in general, is affected rather than learning in a specific modality and that deficits can be attributed to difficulties with encoding of new information rather than retrieval of what has been learned. Data also suggest that there is a spectrum of deficits in these functions with those individuals who demonstrate physical effects of exposure showing the most severe outcomes. Finally, based on the results of this study, there is little to discriminate the performance deficit associated with prenatal alcohol exposure from that shown by other young adults who qualified for special education services as children. However, only limited aspects of memory performance were evaluated in this study and a number of questions regarding memory functioning in this group remain to be explored.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Figure 2.

Table 1

Description of Dependent Variables for the Selective Reminding Tasks

Total Recall (TR)	The number of total words or dots correctly recalled. For the VSRT, the maximum is 12 words x 8 trials=96. For the NVSRT, the maximum is 8 dots x 8 trials=64.
Long Term Storage (LTS)	The number of words or dots recalled without reminders on two consecutive trials, regardless of whether they are ever again recalled. For example, a word or dot that was recalled on Trials 2 and 3 would be considered to be in LTS, even if that item was never again recalled.
Consistent Long Term Retrieval (CLTR)	The number of words or dots recalled without reminders on two consecutive trials all the way through trial 8 (the last trial). For example, a word or dot that was recalled on Trials 2 and 3 would be considered to be in CLTR only if that item was repeatedly recalled without reminders through Trial 8.
Delayed Recall (DR)	Unwarned recall of the 12 words or 8 dots that were originally presented. The delay period is 30 minutes after the final trial.

Table 2
Demographic, Prenatal Exposure, and Growth Characteristics for Participants (N=234) by Exposure Group

Variable	Group				Statistic ^b
	Control (n= 59) ^a	EtOH (n= 74) ^a	Dysm (n= 47) ^a	Special Ed (n= 54) ^a	
Gender – % male	39.0	33.8	48.9	53.7	$\chi^2_{(3)}=6.16$, NS
Ethnicity – % African-American	100	98.6	97.9	85.2	$\chi^2_{(6)}=21.74$, p=.001
Age at testing, M (SD)	22.80 (1.75)	22.45 (1.80)	22.66 (2.10)	23.31 (1.39)	$F_{(3,230)}=2.61$, p=.053, EtOH<Spec Ed
Monthly income – \$ in past 30 days, M (SD) n=228	1191 (1453)	763 (754)	1011 (967)	1293 (1596)	$F_{(3,224)}=2.30$, p=.078
Education completed – years, M (SD) n=230	12.45 (1.77)	12.05 (1.82)	11.80 (1.44)	12.20 (1.59)	$F_{(3,226)}=1.39$, NS
Full-scale IQ, M (SD) n=233	86.07 (11.56)	84.31 (13.88)	75.66 (13.02)	84.94 (13.90)	$F_{(3,229)}=6.54$, p<.000, Dysm<all
Dysmorphia rating at adult visit, M (SD) n=224	3.12 (3.30)	4.51 (3.48)	9.87 (7.66)	4.67 (4.22)	$F_{(3,220)}=19.38$, p<.000, Dysm>all
Current Alcohol Use by Participant (oz/AA/wk), n=230	0.67 (1.92)	1.86 (3.73)	0.93 (1.63)	2.65 (5.70)	$F_{(3,226)}=3.36$, p=.019, C<SpecEd
Adult weight–lbs., M (SD) n=232 ^c					$F_{(3,224)}=7.24$, p<.000, Dysm<all
Male	203.08 (48.16)	195.34 (45.72)	162.76 (49.98)	188.66 (46.56)	
Female	180.05 (44.13)	170.33 (53.31)	135.44 (31.18)	179.84 (66.78)	
Adult head circumference–cm, M (SD) n=235 ^c					$F_{(3,225)}=9.33$, p<.000, Dysm<all
Male	59.70 (1.64)	59.14 (2.32)	56.39 (2.89)	58.92 (2.07)	
Female	57.51 (2.93)	57.32 (2.41)	54.93 (3.23)	57.40 (5.98)	
Amount of alcohol exposure during pregnancy – AA per week, M (SD) n=180	0.00 (0.00)	8.03 (10.66)	13.59 (13.02)	---	$F_{(2,177)}=27.53$, p<.000, C<EtOH, Dysm; EtOH<Dysm
Cigarettes during pregnancy – % using, n=178	32.2	64.4	87.0	---	$\chi^2_{(2)}=33.37$
Marijuana during pregnancy – % using, n=180	13.6	47.3	29.8	---	$\chi^2_{(2)}=17.37$
Cocaine during pregnancy – % using, n=171 ^d	0.0	12.7	4.9	---	$\chi^2_{(2)}=8.82$

^aIf data for a variable are not available for some participants, the n used for the analysis is noted next to the variable name.

^b Posthoc comparisons completed with Tukey HSD test.

^c Two-way Group X Gender analyses of variance were completed for growth variables. In addition to group effects noted in table, gender was significant for adult weight and for adult head circumference. No interaction effects were significant.

^d Recruitment occurred before the "cocaine epidemic" on the 1980s; "crack" use did not occur and frequency and amount of cocaine use was low.

Table 3

Recall Outcomes by Exposure Group and Modality (N=234)

	Control	ETOH	DSYM	Special Ed	Statistic
Recall Outcome					
Verbal (N=231)	M (SD)	M (SD)	M (SD)	M (SD)	
	(n=59)	(n=73)	(n=45)	(n=54)	
Learning Slope	0.357(0.13)	0.321 (0.138)	0.266 (0.142)	0.285 (1.39)	$F_{(3,227)}=4.61, p<.004$ C> DYSM & SpecED EIOH>DYSM ²
Mean Recall	8.49 (1.36)	7.78 (1.68)	7.13 (2.05)	7.04 (2.06)	$F_{(3,227)}=7.92, p<.000$ C>ALL EIOH>SpecED ²
Recall 8th Trial Final Learning Trial	10.29 (1.79)	9.27 (2.14)	8.40 (2.54)	8.48 (2.41)	$F_{(3,227)}=8.67, p<.000$ C> ALL EIOH>DYSM & SpecED ²
CLTR Trial 8	8.59 (2.91)	7.07 (3.02)	5.78 (3.42)	5.78 (3.40)	$F_{(3,227)}=9.83, p<.000$ C> All EIOH>DYSM & SpecED ²
LTS Trial 8	10.47 (2.00)	9.63 (2.69)	8.27 (3.27)	8.13 (3.30)	$F_{(3,227)}=8.821, p<.000$ C> All EIOH>SpecED ²
Delayed Recall N=229	8.47 (2.6)	7.61(2.84)	6.27 (2.98)	6.52 (2.83)	$F_{(3,225)}=7.14, p<.000$ C & EIOH> DYSM & SpecED ²
Nonverbal (N=230)					
	(n=58)	(n=72)	(n=47)	(n=53)	
Learning Slope	0.220 (0.10)	0.208 (0.116)	0.170 (0.143)	0.177 (0.125)	$F_{(3,226)}=2.19, p=.09, NS$ C>DYSM ²
Mean Recall	5.97 (1.15)	5.42 (1.33)	5.18 (1.58)	5.33 (1.34)	$F_{(3,226)}=3.48, p<.02$ C>ALL ²
Recall 8th Trial Final Learning Trial	7.21 (1.06)	6.61 (1.77)	6.15 (2.13)	6.32 (1.91)	$F_{(3,226)}=3.87, p<.01$ C> DYSM & SpecED ²
CLTR Trial 8	6.41 (1.64)	5.53 (2.48)	5.11 (2.56)	5.36 (2.32)	$F_{(3,226)}=3.42, p<.02$ C>ALL ²
LTS Trial 8	7.31 (1.03)	6.75 (1.66)	6.57 (1.54)	6.72 (1.38)	$F_{(3,226)}=3.84, p<.04$ C>ALL ²
Delayed Recall N=228	6.12 (1.61)	5.69 (1.91)	5.15 (2.27)	5.37 (2.04)	$F_{(3,224)}=2.49, p=.06, ns$ C> DYSM & SpecED ²

¹N for each analysis shown in Table; Ns vary due to subject fatigue/illness; experimenter error & time constraints. Subject loss was random with no systematic group difference.

²Tukey HDL post hoc analysis