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The Effects of Prenatal Alcohol Exposure on Episodic Memory Functioning: A Systematic Review

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Abstract

Objective: This paper systematically reviews the literature on the effects of prenatal alcohol exposure (PAE) on episodic memory. Specifically, the review focuses on recurring questions of whether memory deficits are consistent across memory domains, whether the impairments are consistent across the stages of episodic memory, and whether the impairments are primary episodic memory impairments or secondary to a global performance deficit or a higher order deficit.
Method: In total, 33 relevant studies were identified through searches on electronic databases. Journal articles were limited to those that included human subjects and that were published in English-language journals.
Results: The vast majority of reviewed studies examined memory in school-aged children and adolescents. Twenty-three studies examined verbal memory and 19 studies examined visual-spatial memory. Although all of the reviewed studies examined encoding of new material, only 10 studies examined retention of the learned material over time. Ten studies controlled for IQ, either statistically or with matched controls, when analyzing memory task performance.
Conclusion: In general, studies show that PAE results in impaired verbal and visual-spatial episodic memory performance in affected individuals and these impairments are unlikely to be secondary to a global impairment. However, impairments on some memory tests are specific to the encoding stage, whereas retention is relatively spared; suggesting that the episodic memory deficit might be influenced, at least in part, by higher order cognitive processes.

Keywords: Prenatal alcohol exposure; Episodic memory; Verbal memory; Visual-spatial memory

Introduction

Prenatal alcohol exposure (PAE) is well documented to have teratogenic effects on the developing fetus with resulting physical, behavioral, and cognitive impairments (Flak et al., 2014; Mattson, Crocker, & Nguyen, 2011; Moore, Migliorini, Infante, & Riley, 2014; Riley, Infante, & Warren, 2011). These alcohol-related impairments fall under the broad diagnostic banner of fetal alcohol spectrum disorder (FASD); with fetal alcohol syndrome (FAS) situated on the severe end of the continuum. The prevalence of FASD is estimated to range from 1% to 5% in the United States and Western Europe to as high as 9% in certain communities in South Africa (May et al., 2009, 2013, 2014; Sampson et al., 1997; Viljoen et al., 2005).

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Both animal and human studies have consistently demonstrated that PAE has adverse effects on the development of certain brain regions, including the corpus callosum, cerebellum, basal ganglia, and hippocampi (Barnes & Walker, 1981; Berman & Hannigan, 2000; Donald et al., 2015; Hoff, 1988; Livy, Miller, Maier, & West, 2003; Moore et al., 2014; Sutherland, McDonald, & Savage, 1997). These adverse effects on the developing brain have been associated with neuropsychological and cognitive impairments in attention, executive functioning, processing speed, visual and spatial processing, language, academic achievement, and memory (Mattson et al., 2011). However, despite these associated impairments, a precise understanding of the core cognitive deficits attributed to PAE is needed to aid in diagnosis and intervention.

One area of cognition that has been examined in multiple studies involving PAE individuals is episodic memory. Episodic memory is the branch of declarative memory that can be defined as the explicit and voluntary storage and retrieval of specific events and comprises of several dynamic stages (LaBar & Cabeza, 2006; Tulving, 1984). First, an experienced episode or event is encoded. The encoding of the event results in a new and fragile memory trace that is subsequently stabilized during the consolidation stage. The memory can then be reactivated during the retrieval phase (Tulving, 1985). Episodic memory is a cognitive function that is crucial for developing an individual's identity, gaining independence, educational achievement, and accomplishing both routine and novel tasks of daily life (Bauer et al., 2013).

The hippocampus is a brain structure that is critically involved in episodic memory function (Frankland & Bontempi, 2005; Penfield & Milner, 1958; Scoville & Milner, 1957; Squire & Bayley, 2007; Sutherland & Lehmann, 2011; Winocur, Moscovitch, & Bontempi, 2010). That is, this structure is essential for the acquisition, retention, and retrieval of declarative memories that deal with conscious recollection of episodes. In contrast, the hippocampus seems minimally involved in non-declarative memory forms, such as motor and perceptual learning, priming, and the learning of habits, skills, and rules (Winocur et al., 2010).

Research involving primates and rodents has demonstrated that the hippocampi are particularly vulnerable to the toxic properties of alcohol. That is, both pre- and postnatal alcohol exposure is documented to lead to alterations in the hippocampal anatomy, morphology, and electrophysiology; restricting normal development and function of the structure (Berman & Hannigan, 2000; Bonthius & West, 1990, 1991; Hoff, 1988; Kelly, 1996; Klintsova et al., 2007; Livy et al., 2003; Maier & West, 2001; Miller, 1995; Puglia & Valenzuela, 2010; Sutherland et al., 1997; West & Hamre, 1985).

Behavioral evidence has demonstrated that, in comparison to nonexposed rodents, pre- and postnatal alcohol exposed rats display deficits in spatial memory. These deficits have been confirmed on hippocampal dependent tasks such as the Morris Water Maize (MWM; Morris, 1984); in which even after extensive training, exposed rats demonstrate longer path length and time to find an escape platform (Berman & Hannigan, 2000; Blanchard, Riley, & Hannigan, 1987; Gianoulakis, 1990; Goodlett & Johnson, 1997; Goodlett, Kelly, & West, 1987; Johnson & Goodlett, 2002; Kim et al., 1997; Savage, Becher, Torre, & Sutherland, 2002; Sutherland, McDonald, & Savage, 2000). Spatial memory deficits have also been reported on other spatial tasks in rodent (e.g., see La Fiette, Carlos, & Riley, 1994; Nagahara & Handa, 1997; Reyes, Wolfe, & Savage, 1989) and primate studies (e.g., see Clarren, Astley, Gunderson, & Spellman, 1992; Schneider, Moore, & Kraemer, 2001) (for an in-depth review of animal studies, please see Schneider, Moore and Adkins, 2011).

Research has, therefore, shown that PAE effects the development of, amongst other brain regions, the hippocampi (brain structures that are critically involved in episodic memory), whereas animal behavioral studies show supportive evidence that PAE results in episodic memory impairments. The aim of this paper is to review systematically the literature that reports on the effects of alcohol exposure on episodic memory in children, adolescents, and adults. Although previous reviews have focused on cognitive and neuropsychological impairments associated with PAE (e.g., see Davis, Gagnier, Moore, & Todorow, 2013; Flak et al., 2014; Kaemingk & Paquette, 1999; Kodituwakku & Kodituwakku, 2014; Kodituwakku, 2009; Mattson et al., 2011; Wacha & Obrzut, 2007), and specifically memory impairments (e.g., see Manji, Pei, Loomes, & Rasmussen, 2009), this review will focus on the current literature surrounding three recurring questions concerning episodic memory impairments in PAE exposed individuals. First, are episodic memory deficits consistent across memory domains (i.e., verbal vs. visual-spatial memory)? For instance, some studies report that verbal memory is more affected than visual-spatial memory (e.g., see Pei, Rinaldi, Rasmussen, Massey, & Massey, 2008; Willford, Richardson, Leech, & Day, 2004), whereas other studies have reported an inverse pattern (e.g., see Mattson & Roebuck, 2002; Rasmussen, Horne, & Witol, 2006). Second, are the memory impairments consistent across the components of episodic memory (i.e., an encoding vs. a retrieval impairment). Several studies that have documented memory impairments in PAE individuals have noted that once learning differences have been taken into consideration, memory retrieval remains preserved (e.g., see Coles, Lynch, Kable, Johnson, & Goldstein, 2010; Crocker, Vaurio, Riley, & Mattson, 2011; Mattson, Riley, Gramling, Delis, & Jones, 1998) although other studies have documented impaired retrieval or recognition even after taking learning differences into account (e.g., see Lewis et al., 2015; Mattson & Roebuck, 2002). Lastly, are the reported episodic memory impairments due to the teratogenic effects of PAE on specific brain structures responsible for episodic memory functioning, or due (perhaps in part) to secondary impairments such as a global performance impairment (e.g., see Kaemingk, Mulvaney, & Halverson, 2003;

Kaemingk & Tanner Halverson, 2000; Mattson et al., 1998) or impairments in higher order processes that are equally characteristic of PAE (Green et al., 2009; Khoury, Milligan, & Girard, 2015; Rasmussen, 2005).

Methods

Identification of Relevant Studies

An initial electronic search was conducted to identify studies through the following databases: PubMed, PsycINFO, and Google Scholar. Relevant studies were identified through the following combinations of search terms and keywords: "alcohol," "fetal alcohol spectrum disorders," "fetal alcohol syndrome," "FAS," "FASD," "prenatal alcohol exposure," "episodic memory," "learning," "encoding," "retrieval," "verbal memory," "visual memory," "spatial memory," "visual-spatial memory," "nonverbal memory," "children," and "adults." The search was not restricted to starting date limits and extended to July 2015. Journal articles were limited to those that included human subjects and that were published in English-language journals. Abstracts were manually examined in order to confirm relevance. Further studies were identified by searching the reference lists of studies identified in the initial database search.

A systematic narrative approach was adopted for this review instead of a quantitative comparison such as meta-analysis. This approach was implemented for the following reasons: (a) Data were not consistently available in order to compute effect sizes. (b) Methodology between studies examining episodic memory varied substantially, and thus prevented clear comparisons. (c) There were significant differences in secondary variables across different studies examining episodic memory, such as age, gender, extent, and timing of alcohol exposure and polysubstance exposure. Therefore, a systematic qualitative approach was deemed more suitable for this particular review that incorporates over two decades of studies on the effects of PAE on episodic memory functioning.

A total of 33 relevant articles were identified and included in this review. Due to the relatively small number of identified articles, we included studies that used samples from longitudinal cohorts and post hoc clinical samples, as well as studies that did not include a control group.

Results

Studies reporting on the effects of PAE on episodic memory have been widely reported and 33 relevant studies were identified for this review and are listed alphabetically in Table 1.

The majority of the 33 studies used samples of participants ranging from childhood to mid-adolescence. A few studies ($n = 5$) included young adults, whereas a small number (approximately $n = 3$) investigated preadolescent children exclusively. One study focused on adults only (Kerns, Don, Mateer, & Streissguth, 1997). The widest age range in a sample was a 25-year longitudinal study (Streissguth, 2007) and the smallest age range was 1.5 years in a sample of preschool children (Janzen, Nanson, & Block, 1995).

Although the study designs varied, most used a control group to compare with the PAE participants. The majority ($n = 18$) of the studies did not differentiate between PAE participants with different diagnoses. Of the remainder, one study compared PAE participants with FAS features with unexposed controls (Carmichael Olson, Feldman, Streissguth, Sampson, & Bookstein, 1998) and two studies compared groups of PAE participants with FAS versus those without FAS features, in addition to a control group (Coles et al., 2010, 2011). Furthermore, one of these studies (Coles et al., 2010) included an additional special education needs group for comparison. Similarly, another study (Mattson & Riley, 1999) included a non-PAE Down's Syndrome group and another (Crocker et al., 2011) compared PAE with and without Attention deficit hyperactivity disorder (ADHD) groups. Neither of these two studies differentiated between FAS and non-FAS in their PAE sample. Only five studies used no control group, although several of these compared levels of prenatal alcohol intake, either in discrete groups such as moderate to heavy versus abstaining to light, or by correlational methods. The reviewed studies that investigated FAS and non-FAS presenting samples showed that although there was some variation in performance on episodic memory tasks, the between-group differences appeared to be a matter of degree, rather than distinctly different profiles of clinical symptoms, suggesting that it was appropriate to review studies that grouped FAS and non-FAS participants together with studies that investigated separate groups.

The majority of studies ($n = 14$) examining verbal memory have utilized wordlists, whereas relatively few have examined memory for stories ($n = 4$) and names ($n = 2$). In contrast, studies examining visual and spatial memory have most commonly used dot and design location tasks ($n = 9$). Less frequently used visual-spatial tasks include mazes ($n = 2$), memory for figures or designs ($n = 2$), and facial memory ($n = 4$).

Table 1. Studies examining episodic memory in PAE samples

Author	*N* = PAE	N = Control	Site	Age (years)	Focus of the study	Test/s	Episodic memory findings
Aragón et al. (2008)	24	32	Albuquerque, NM, USA	7–17	Assessed cognitive functioning in a group of PAE children and adolescents diagnosed as FAS, Partial FAS, or FASD. The study applied a hierarchical model of simple versus complex information processing to examine cognitive function.	• LS/LTM • DRST	PAE groups displayed impairments in learning on both the LS and LT. In addition, they performed more poorly on delayed recall of the LT, which is the more difficult spatial memory task. However, the PAE group preformed similarly to control on the easier DRST spatial tasks (both immediate and delayed recall) and on the recognition task.
Carmichael Olson et al. (1998)	9	174	Seattle, WA, USA	14–16	Assessed the neuropsychological profile of a PAE group diagnosed with FAS, in comparison with a large unexposed control group.	• SSM	PAE group performed worse than the control cohort; and even when they were compared to a smaller IQ controlled group ($n = 52$).
Coles et al. (2010)	121	59	Atlanta, GA, USA	$M = 22.78$, $SD = 1.79$	Assessed verbal and visual-spatial memory functioning in a PAE group (separated into a Dysmorphic PAE group, $n = 47$, and a Non-dysmorphic PAE group, $n = 74$), a Control Group, and a Special Education Group ($n = 54$). The PAE group was recruited from a longitudinal cohort.	• VSRT • NVSRT	The Dysmorphic PAE group displayed impairments in both verbal and visual-spatial memory performance in comparison to the Controls but they did not differ from the Special Education group. The Non-dysmorphic PAE group was intermediate in their performance and performed better than the Dysmorphic and Special Education groups but worse than the controls. After controlling for differences in encoding, the PAE groups showed similar retention rates to the controls.
Coles et al. (2011)	66	26	Atlanta, GA, USA	$M = 22.97$, $SD = 1.83$	Assessed impact of PAE on memory and brain development in a Dysmorphic group ($n = 30$), a ARND group ($n = 36$), and a Control group. The study assessed verbal and visual-spatial memory, and collected structural MRI data. The PAE group was recruited from a longitudinal cohort.	• VSRT • NVSRT	In comparison to the control group, the dysmorphic group showed impaired verbal and visual-spatial memory performance in both learning rate and recall. The ARND group was intermediate in performance, indicating a spectrum of impact. Both PAE groups demonstrated significantly lower total brain volume than controls, as well as lower volume in specific regions including the hippocampi. Mediation analyses showed memory performance were associated with effects of PAE, which was in turn linked with dysmorphic severity through

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Table 1. (*continued*)

Author	*N* = PAE	N = Control	Site	Age (years)	Focus of the study	Test/s	Episodic memory findings
							hippocampal volume; in particular the right hippocampus.
Crocker et al. (2011)	22	22	San Diego, CA, USA	7–14	Assessed verbal learning and memory in a PAE group with ADHD, an unexposed group with ADHD ($n = 22$) and a control group.	• CVLT-C	PAE group recalled fewer words on learning trials than the ADHD group, whereas the Control group preformed the best. On the delayed recall trial, the PAE and ADHD groups showed similar performances, and both were worse than the Control group. However, differences on the delayed recall trial were not apparent in the PAE group after researchers controlled for initial learning. The PAE group displayed poorer recognition memory than both the ADHD and Control groups.
Fryer et al. (2012)	21	7	San Diego, CA, USA	9–21	Assessed the relationship between structural brain alterations and memory in a PAE group and a control group.	• CVLT-C	PAE groups performed significantly more poorly on the immediate recall task but performed similar to the controls on the delayed recall task. Memory performance was associated with volume of the caudate nuclei.
Hamilton et al. (2003)	8	8	Albuquerque, NM, USA	9.5–16.5	Assessed place learning and cued-navigation in a Virtual Morris water task in a PAE and Control group	• VMWT	PAE group displayed an impaired performance on the hidden-target acquisition (learning) trials and on the probe trial. The PAE group and control group showed a similar performance on the visible target cue-navigation trials.
Janzen et al. (1995)	10	10	Saskatoon, Saskatchewan, Canada	3.5–5	Assessed neuropsychological performance of preschool-aged children with FAS in comparison to a Control group.	• MSCA	There were no significant differences in memory performance between PAE and control children. Not clear if memory performance was verbal, visual-spatial, or a combination of both.
Kaemingk and Tanner Halverson (2000)	20	20	Tucson, AZ, USA	6–16	Assessed visual-spatial and verbal memory in a PAE group (FAS, $n = 11$; FAE, $n = 9$) and a control group.	• WRAML • QLT	PAE group displayed impaired memory performance on visual-spatial and verbal memory tasks. Authors concluded that PAE results in a general, rather than a material specific, memory impairment.
Kaemingk et al. (2003)	20	20	Tucson, AZ, USA	6–16	Assessed verbal and visual-spatial learning in a PAE group and a control group.	• WRAML	PAE group displayed weaker learning on both verbal and visual-spatial learning trials and on the delayed recall trials. However, retention differences on the delayed recall trial were not apparent after the authors controlled for initial learning.
	16	NA	Seattle, WA, USA	16–27		• CVLT	

Kerns et al. (1997)					Assessed general cognitive deficits in a PAE adult group that ranged from above average to low IQ.		Participants showed deficits in verbal learning and memory that were greater than what would be predicted on the basis of IQ alone.
Korkman et al. (2003)	27	39	Helsinki, Finland	12–14	Assessed neurocognitive status in PAE early adolescents and examined whether duration of exposure predicts the cognitive profile in a longitudinal cohort.	• NEPSY-names, faces and narrative	The PAE group showed impairments related to duration of exposure on verbal memory test (notably the memory for names subtest). Delayed recall was not examined. PAE group was not directly compared to the control group.
Kully-Martens, Pei, Job, and Rasmussen (2012)	19	38	Edmonton, Alberta, Canada	6–12	Assessed source monitoring ability and performance a PAE group of children and a Control group.	• Source monitoring task	PAE children showed poorer performance than controls across all conditions in both recognition memory and memory for source.
Lewis et al. (2015)	Cape Town = 91, Detroit = 40	Cape Town = 60, Detroit = 251 (classed as Abstainer/ Light)	Cape Town, South Africa & Detroit, MI, USA	Cape Town (M = 10.3 years), Detroit, MI, USA (14.4 years)	Assessed verbal memory in two independent longitudinal cohorts (Cape Town and Detroit, MI) to determine whether (a) effects on encoding are also seen at moderate exposure levels, (b) these deficits are specific or secondary to an IQ impairment, (c) effects on retrieval can be detected over and above effects on initial encoding, and (d) effects on learning are due to the use of a less efficient learning strategy.	• CVLT-C	After adjusting for IQ, the heavy PAE Cape Town cohort displayed impairments in encoding, whereas both cohorts displayed retrieval impairments. However, after controlling for initial encoding, retrieval impairments were no longer significant in the Cape Town cohort. By contrast, the Detroit cohort displayed recognition memory impairments after controlling for initial learning. PAE children with full or partial FAS were less likely to use a semantic cluster encoding strategy.
Mattson and Riley (1999)	21	21	San Diego, CA, USA	8–18	Assessed implicit and explicit verbal memory in a PAE group, a Down syndrome group (n = 11), and a Control group.	• FRRMT	The PAE and Down syndrome groups performed worse than control subjects on the free recall task. However, the PAE group performed equivalently to the control children on the recognition and priming tasks.
Mattson and Roebuck (2002)	35	35	San Diego, CA, USA	8–16	Assessed verbal and visual-spatial learning and memory in a PAE and a Control group.	• CVLT-C • WRAML • BFLT	PAE children recalled less verbal and visual-spatial information on learning trials and displayed lower rates of acquisition. PAE group also recalled less verbal and visual-spatial information on the delayed recall trials. However, this difference on the delayed recall trial was not apparent in verbal memory after researchers controlled for initial

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Table 1. (*continued*)

Author	*N* = PAE	N = Control	Site	Age (years)	Focus of the study	Test/s	Episodic memory findings
							learning. Thus, the authors reported differences in the retention of verbal versus visual-spatial memory.
Mattson et al. (1996)	20	20	San Diego, CA, USA	5–16	Assessed verbal learning and memory in a PAE and a Control group.	• CVLT-C	PAE children displayed impaired verbal memory (both immediate and delayed recall performance) in comparison to the control group. The PAE group also tended to make a greater number perseverative errors and made more false-positive errors on recognition testing.
Mattson et al. (1998)	25	25	San Diego, CA, USA	5–16	Compared three groups (a PAE group that met the FAS diagnostic criteria, $n = 15$, a PAE group that did not meet FAS diagnostic criteria, $n = 10$, and a Control group) on a battery of neuropsychological tests.	• CVLT-C	PAE groups recalled fewer words on the learning trials and delayed recall trial, and displayed poorer recognition. Differences in the retention of the wordlist were not apparent after researchers controlled for differences in initial learning.
Pei et al. (2008)	30	NA	Edmonton, Alberta, Canada	9–16	Assessed memory function in a PAE group	• CMS	The authors reported that the recall of word pairs was more impaired than the recall of stories in their PAE group. Immediate recall was significantly more impaired than delayed recall, indicating a deficit in encoding. In contrast, PAE was NOT associated with poorer memory performance on the visual memory subtests (spatial and object) on either the immediate or delayed recall.
Pei et al. (2011)	35	35	Edmonton, Alberta, Canada	6–12	Assessed visual-spatial ability, memory, and executive functioning in PAE and control groups.	• ROCF	PAE group performed worse than controls in copying the figure as well as on immediate and delayed recall memory tasks.
Rasmussen et al. (2006)	50	NA	Edmonton, Alberta, Canada	6–16	Assessed cognitive functioning in a PAE group that had been diagnosed with FASD.	• CMS	PAE was associated with poor performance on immediate and delayed recall of faces but average performance on immediate and delayed recall of the dot location task. PAE was NOT associated with poor memory performance on verbal tasks. However, the authors also noted possible ethnic differences in memory performance of PAE children.
Rasmussen et al. (2013)	32	30	Edmonton, Alberta, Canada	6–16	Compared the performance of a PAE group and a control group on subtests of NEPSY-II.	• NEPSY-II–Memory for names/ delayed	PAE groups performed more poorly on both immediate and delayed conditions, compared with controls.

Richardson et al. (2002)	593	NA	Pittsburgh, PA, USA	10–13	Longitudinal study that reported the effects of prenatal alcohol and marijuana exposure on neuropsychological development at 10 years of age.	• WRAML	PAE predicted poorer learning and memory for verbal material (words and stories) and visual-spatial material (pictures and designs).
Roebuck-Spencer and Mattson (2004)	35	34	San Diego, CA, USA	9–16	Reanalyzed data from a previous study (Mattson & Roubuck, 2002) to determine if the presence of an implicit verbal learning strategy may account for the finding of spared memory retention in that study.	• CVLT-C • WRAML-Verbal learning subtests	PAE group displayed poorer learning on both verbal memory tests. However, in comparison to the control group, the PAE group showed similar retention rates on the CVLT-C and worse retention on the WRAML. The authors attribute this retention difference to the use of an implicit learning strategy on the CVLT-C.
Sowell et al. (2007)	11	16	San Diego, CA, USA	7–15	Assessed fMRI activation patterns corresponding to verbal paired associate learning in a PAE and a Control group.	• PAL	The PAE group performed significantly worse than the Control group on the verbal learning test. However, after the PAE group was compared with a smaller IQ-matched control group, the significant difference was not apparent.
Sowell et al. (2008)	18	18	San Diego, CA, USA	8–25	Assessed the relationship between cognitive dysfunction and cortical thickness in a PAE and Control group.	• CVLT-C	The PAE group preformed significantly poorer than the Control group on the verbal memory test. Verbal recall was correlated with cortical thickness. In addition, significant group by test score interactions were found in right dorsal frontal regions for verbal memory.
Streissguth (2007)	±500	NA	Seattle, WA, USA	0–25	Reports on a 25-year longitudinal study that assessed memory in a PAE cohort when they were 7, 14, and 21 years old	• CMS • SSM	The PAE predicted consistent deficits in visual-spatial memory across all age measurement points.
Uecker and Nadel (1996)	15	15	Tucson, AZ, USA	$M = 10.03$, $SD = 2.31$	Assessed visual-spatial memory in a PAE and Control group.	• 16 Object Task	PNA group preformed similarly to controls on the immediate recall trials but worse on the delayed recall trial (24 hr delay) and displayed a general spatial memory deficit.
Uecker and Nadel (1998)	15	15	Tucson, AZ, USA	$M = 10.03$, $SD = 2.33$	Assessed object (cue) and spatial (place) memory in a PAE and a Control group.	• Ellis task	PAE group demonstrated both immediate and delayed recall spatial (place) memory impairments. There were no performance differences between the PAE and control groups on object (cue) memory.
	55	55		6–16		• CVLT-C	

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Table 1. (*continued*)

Author	*N* = PAE	N = Control	Site	Age (years)	Focus of the study	Test/s	Episodic memory findings
Vaurio et al. (2011)			San Diego, CA, USA		Assessed neuropsychological performance of a PAE group in comparison to in IQ-matched control group.		PAE group demonstrated impaired learning but intact retention of verbal material in comparison to the control group.
Wheeler et al. (2012)	19	21	Toronto, Ontario, Canada	10–15	Assessed memory for faces in a PAE and a Control group.	• CMS—Faces subtest • TOMAL—Facial memory subtest	PAE group displayed memory impairments on both facial memory tests. In addition, the PAE group performed worse on the more difficult CMS facial memory task compared to the easier TOMAL task.
Willford et al. (2004)	580	NA	Pittsburgh, PA, USA	13–16	Assessed the effects of moderate PAE on learning and memory in adolescents from a longitudinal cohort.	• CMS	PAE during the first trimester was associated with verbal deficits in learning and recall of word pairs; although the authors suggest that these deficits were mediated by impairment in learning. In contrast to the effects on verbal memory, PAE during the first trimester was NOT associated with deficits in learning and recall of visual and spatial information.
Willoughby et al. (2008)	19	18	Toronto, Ontario, Canada	9–15	Assessed whether learning and recall abilities in PAE children are associated with abnormal hippocampal volumes.	• CMS • CVLT-C • EMQ • ROCF	PAE children had smaller left hippocampi and displayed poorer verbal learning and verbal and visual-spatial recall performance in comparison to the control group. In addition, there were positive correlations between selective memory indices and hippocampal volumes only in the PAE group, whereas hippocampal volumes increased significantly with age only in the control group.

Note: BFLT = Biber Figure Learning Test (Glosser, Goodglass, & Biber, 1989); CMS = Children's Memory Scale (Cohen, 1997); CVLT = California Verbal Learning Test (Delis, Kramer, Kaplan, & Ober, 1987); CVLT-C = California Verbal Learning Test-Children's Version (Delis, Kramer, Kaplan, & Ober, 1994); DRST = Delayed Recognition Span Test of Memory (Moss, Albert, Butters, & Payne, 1986); Ellis task (Ellis, Katz, & Williams, 1987; Ellis, Woodley-Zanthos, & Dulaney, 1989; Katz & Ellis, 1991); EMQ = Everyday Memory Questionnaire (Isaacs et al., 2000; Sunderland, Harris, & Baddeley, 1984; Vargha-Khadem et al., 1997); FRRMT = Free Recall and Recognition Memory Test (Salmon, Shimamura, Butters, & Smith, 1988); LS/LTM = Lhermitte Spatial/Logical Test of Memory (Lhermitte & Signoret, 1972); MSCA = McCarthy Scales of Children's Abilities (McCarthy, 1972); NVSRT = Nonverbal Selective Reminding Memory Test (Fletcher, 1985); PAL = Paired Associate Learning Test; PAE = Prenatal alcohol exposed; QLT = Quadrant Localization Task (Uecker, 1993); ROCF = Rey-Osterrieth Complex Figure task (Osterrieth, 1944; Rey, 1941); SSM = Stepping Stone Maze (Milner, 1965); TOMAL = Test of Memory and Learning (Reynolds & Bigler, 1994); WRAML = Wide Range Assessment of Memory and Learning (Sheslow & Adams, 1990); VMWT = Virtual Morris Water Task (Hamilton, Driscoll, & Sutherland, 2002; Hamilton & Sutherland, 1999); VSRT = Verbal Selective Reminding Memory Test (Buschke & Fuld, 1974); 16 Object Task (Smith & Milner, 1981).

Although all of the reviewed examined memory, only 10 studies examined retention of the learned material over time. In addition, although most of the studies measured IQ as part of the procedures, only 10 studies controlled for IQ, either statistically or with matched controls, when analyzing memory task performance.

Seven of the reviewed studies were longitudinal or drew samples from a longitudinal cohort. Furthermore, at least 14 studies reused the same participant samples. The remainder of studies used post hoc, often clinically referred, samples.

Discussion

The present review focuses on studies that have investigated the effects of PAE on episodic memory. As presented in Table 1, episodic memory deficits have been consistently reported in PAE school-aged children, adolescents, and adults (although, see Janzen et al. (1995) for an exception in a preschool cohort). In specific, the present review aimed to address three recurring questions: Are episodic memory deficits consistent across memory domains? Are the memory impairments consistent across the components of episodic memory? And, are the memory impairments a primary deficit or secondary (at least in part) to a global performance impairment or impairments in higher order processes?

Verbal Versus Visual-Spatial Episodic Memory

Verbal memory deficits have been documented on a variety of verbal memory tests in PAE individuals. The California Verbal Learning Test-Children's Version (CVLT-C) is one such test that involves children encoding and retrieving an orally presented wordlist and has been used in at least 10 studies documented in this review. Some of these studies have reported that, in comparison to typically developing controls, PAE children display poorer learning of the wordlist (Fryer et al., 2012; Sowell et al., 2008), whereas others have also reported impairments in recalling and/or recognizing the words (Crocker et al., 2011; Lewis et al., 2015; Mattson, Riley, Delis, Stern, & Jones, 1996; Mattson et al., 1998; Mattson & Roebuck, 2002; Roebuck-Spencer & Mattson, 2004; Vaurio, Riley, & Mattson, 2011; Willoughby, Sheard, Nash, & Rovet, 2008). Impairments on other wordlist tasks have been echoed in both PAE adult and children samples (Coles et al., 2010; Kaemingk et al., 2003; Pei et al., 2008; Willford et al., 2004).

Studies that have utilized stories in addition to, or instead of, wordlists are less common. However, studies have reported that, in comparison to normally developing controls, PAE children show impairments in story recall at immediate and delayed time points (Willoughby et al., 2008). PAE has also been shown to predict story recall on the Wide Range Assessment of Memory and Learning (WRAML) in 10-year olds (Richardson, Ryan, Willford, Day, & Goldschmidt, 2002), but not in the same longitudinal cohort at 14 years of age using the story subtest of the Children's Memory Scale (CMS) (Willford et al., 2004). These inconsistent findings could possibly be attributed to either differences in memory task demands or that impairments may resolve with age through improved memory strategy. Pei et al. (2008) reported that PAE children were more impaired on the wordlist task than on the story task of the CMS. However, notable limitations of Pei et al.'s (2008) study were methodological scoring differences between the tasks (only intrusions on the wordlist task were penalized) and the authors did not compare memory performance of the PAE group against a control group, making extrapolation of their findings difficult (Mattson et al., 2011).

In addition to wordlists and stories, studies that have examined memory for names (such as the Memory for Names subtest found in the NEPSY and NEPSY-II) have reported that, in comparison to normally developing controls, PAE children and adolescents performed more poorly on immediate (Korkman, Kettunen, & Autti-Rämö, 2003) and delayed conditions (Rasmussen et al., 2013).

Studies investigating the effects of PAE on visual-spatial memory have reported somewhat less consistent findings. Two commonly used visual-spatial memory tasks are dot location (e.g., a subtask of the CMS) and design location (e.g., a subtask of the WRAML), and both entail participants learning the spatial layout of a series of dots or nonverbal designs over a series of trials. Studies utilizing these kinds of tasks have reported that PAE predicts impairments in both learning and delayed recall (Richardson et al., 2002), although this finding has not been confirmed in two other studies that used smaller clinically recruited samples (Pei et al., 2008; Rasmussen et al., 2006). However, impairments on location tasks have been demonstrated when PAE children and adults are compared to control groups (Coles et al., 2010, 2011; Kaemingk et al., 2003; Kaemingk & Tanner Halverson, 2000; Mattson & Roebuck, 2002); with greater impairments associated with more difficult tasks (Aragón et al., 2008).

In an early study examining memory for objects and their spatial locations, Uecker and Nadel (1996), using a task believed to be sensitive to hippocampal damage, reported impairments in immediate and 24 hr delayed memory for the location of objects. However, memory for the objects themselves was only impaired at the delayed memory stage. In a subsequent study,

the same FAS children displayed only a spatial (and not an object) memory impairment on a different task (Uecker & Nadel, 1998).

Studies that have utilized spatial maze tasks have reported fairly consistent impairments in spatial memory. Streissguth, Barr, Sampson and Bookstein (1994) reported that performance on the Stepping Stone Maze (SSM) was a strong predictor of PAE. In an subsequent study, Hamilton, Kodituwakku, Sutherland and Savage (2003) used a virtual water maze task that is considered a human analog of the MWM. The authors reported that, in comparison to typically developing controls, PAE children displayed slower rates of learning the location of the hidden platform on the acquisition trials. In addition, when the platform was absent from the maze, the PAE group were less efficient at searching the area where the target should have been. However, when the PAE group searched for a visible platform, they showed no difference in performance in comparison the controls; implying that difference in performance on the hidden platform trial was not attributable to visual-motor or motivational deficits.

Studies examining memory for figures (such as the Rey-Osterrieth Complex Figure task; ROCF) or designs (such as the Biber Figure Learning Test; BFLT) have also reported impairments in comparison to control participants (Mattson & Roebuck, 2002; Pei, Job, Kully-Martens, & Rasmussen, 2011; Willoughby et al., 2008).

In addition, memory impairments have also been documented in facial memory tasks (Rasmussen et al., 2006), with greater impairments detected on more difficult tasks when compared with typically developing controls (Wheeler, Stevens, Sheard, & Rovet, 2012). However, findings in this area are inconsistent as some studies have failed to detect facial recognition impairments in PAE individuals (e.g., see Kaemingk & Tanner Halverson, 2000; Uecker & Nadel, 1996).

Due to methodological differences in testing verbal versus visual-spatial memory, direct comparisons between the effects of PAE on the domains are difficult to establish. Interestingly, studies that have utilized the same scale to measure verbal and visual-spatial memory have reported somewhat inconsistent results. For instance, both Pei et al. (2008) and Willford et al. (2004), who utilized the CMS, reported that PAE was associated with a verbal but not a visual-spatial memory impairment, whereas Rasmussen et al. (2006) reported the reverse pattern using the same battery. However, none of these studies compared the performance of the PAE children to a control group (Other studies that have utilized the CMS have reported that PAE children display impaired performance on both the stories and word pairs subtests (Willoughby et al., 2008) and faces subtest (Wheeler et al., 2012) when compared to control participants.). Rather, studies that compared PAE children and adults to controls on a single scale (such as the WRAML and Verbal/Nonverbal Selective Reminding Memory Test, VSRT/NVSRT) have reported impairments in both verbal and visual-spatial memory (Coles et al., 2010, 2011; Kaemingk et al., 2003).

Thus, of the 23 studies addressing verbal memory, 18 reported deficits in memory compared to a control group. In addition, two longitudinal studies reported that PAE was a significant predictor of poorer verbal memory performance. In comparison, of the 19 reviewed studies examining visual-spatial memory, 13 reported an impaired memory performance in comparison to a control group, whereas two longitudinal studies reported that PAE predicts impaired visual-spatial memory. Together, these findings suggest that PAE affects both verbal and visual-spatial memory and that variability in findings on the memory domains might, therefore, be attributed to differing test methodologies or study characteristics.

Encoding Versus Retention Impairment

As described earlier, a large number of studies have reported that PAE is associated with memory impairments in affected school-aged children, adolescents, and adults. However, an interesting memory pattern that is emerging from the relevant literature is that once differences in initial encoding are taken into account, impairments on delayed recall are no longer apparent. This pattern has consistently been demonstrated in studies utilizing the CVLT-C, which have reported that delayed recall deficits are negated once learning differences are taken into account (Crocker et al., 2011; Mattson et al., 1998; Mattson & Roebuck, 2002; Roebuck-Spencer & Mattson, 2004; Vaurio et al., 2011; Willoughby et al., 2008). This pattern has also been reported on other wordlist tasks in both PAE children and adult cohorts (Coles et al., 2010; Kaemingk et al., 2003; Pei et al., 2008; Willford et al., 2004).

Interestingly, the memory pattern of impaired encoding with spared retention has been most frequently reported on tests involving wordlists, although two studies have reported spared retention on spatial location tasks (Coles et al., 2010; Kaemingk et al., 2003). However, Mattson and Roebuck (2002), who in the same study reported spared retention on the CVLT-C, reported impaired retention on a design learning task.

Thus, of the 10 studies examining retention of learned material, all reported impairments in the encoding of the material but retention was spared; at least in terms of learned words. However, delayed retrieval of the encoded material is usually tested relatively soon (roughly 30 min) after the learning material in the relevant literature. The potential problem with

examining retrieval so soon after encoding is that the memory trace is yet to be consolidated. The consolidation phase entails cellular and synaptic reorganization associated with the trace and occurs over a time range that last seconds to hours, depending on the information (Winocur et al., 2010). Future studies might explore this pattern of impaired encoding and spared retention on other verbal and visual and spatial tasks and to test retrieval after longer intervals of 24 hr or more; allowing for the consolidation of the learned material.

Primary Versus Secondary Deficit

The pattern of impaired learning and spared retention, raises the interesting question of whether the memory impairments displayed by PAE individuals are purely episodic memory impairments (i.e., due to the teratogenic effects of PAE on the developing hippocampi). As mentioned earlier, the developing hippocampi are documented to be affected by PAE, whereas Willoughby et al. (2008) reported positive correlations between memory and hippocampal volumes. In addition, Coles et al. (2011) noted that memory performance in PAE individuals was associated with dysmorphic severity of hippocampal volume; in particular the right hippocampus. However, Fryer et al. (2012) recently reported that impaired memory performance on the CVLT-C was associated with volume of the caudate nuclei and not the hippocampus. Therefore, the question arises of whether memory performance in PAE individual might be affected, at least in part, by impairments in other areas of cognition.

Studies that have examined both IQ and memory performance in PAE individuals have reported an association between the two (e.g., see Kaemingk et al., 2003; Mattson et al., 1998). To directly address whether the observed memory impairments are due to a global performance deficit, some studies have either statistically controlled for differences in IQ or compared PAE individuals to an IQ-matched control group. Those studies that have statistically controlled for IQ have reported that IQ differences, in isolation, cannot account for the observed episodic memory impairments (Coles et al., 2010; Kaemingk et al., 2003; Kerns et al., 1997; Lewis et al., 2015; Roebuck-Spencer & Mattson, 2004), whereas studies that have compared PAE cohorts to IQ-matched control groups have also reported memory impairments (Carmichael Olson et al., 1998; Vaurio et al., 2011) (However, Sowell et al., 2007 failed to detect significant difference when their PAE group was compared to smaller IQ-matched control group.). In conjunction, these findings suggest that the memory impairments seen PAE individuals are independent from the global performance impairment.

In an earlier review on the topic, Manji et al. (2009) suggested that the memory (or encoding) impairments displayed by PAE individuals might rather be due to an impairments in higher order processes within memory function. In their review, the authors suggested that executive impairments such as perseverative errors and poor inhibitory control could significantly contribute to the encoding impairments (e.g., see Mattson et al., 1996; Rasmussen, Pei, Manji, Loomes, & Andrew, 2009; Rasmussen et al., 2013). In addition, impairments in executive functions such as selective attention, working memory, and poor memory strategies might also disrupt memory encoding in PAE individuals.

At a foundational level of information processing, impairments in attention are well documented in PAE individuals. Studies have noted that PAE individuals show deficits on attentional tasks such as reaction time, vigilance, and information processing (Burden, Jacobson, & Jacobson, 2005; Coles et al., 1997; Green et al., 2009; Jacobson, Jacobson, & Sokol, 1994; Jacobson, Jacobson, Sokol, Martier, & Ager, 1993; Nanson & Hiscock, 1990; Streissguth, Sampson, et al., 1994). Added to impairments in attention, studies that have examined working memory in PAE individuals have noted impairments in phonological loop of verbal working memory (Becker, Warr-Leeper, & Leeper, 1990; Burden, Jacobson, Sokol, & Jacobson, 2005; Khoury et al., 2015; Rasmussen et al., 2009; Streissguth, Barr, & Sampson, 1990; Streissguth, Sampson, et al., 1994) and on visual-spatial working memory tasks (Green et al., 2009; Khoury et al., 2015; Malisza et al., 2005; Streissguth, Barr, et al., 1994). Thus, the combination of impaired attention and working memory is likely to impede PAE individuals acquiring, maintaining, and processing information during the encoding stage of memory.

In addition to impairments in attention and working memory, PAE individuals are less likely to use efficient learning strategies (Kerns et al., 1997; Mattson & Roebuck, 2002). Roebuck-Spencer and Mattson (2004) reported that when PAE children used an implicit learning strategy on a wordlist task (the CVLT-C), it resulted in spared retention. However, when the task (the wordlist within the WRAML) did not allow for the use of an implicit strategy, PAE children displayed impaired retention of the words. This finding was confirmed recently by Lewis et al. (2015), who reported that PAE children with full or partial FAS were less likely to use a semantic cluster encoding strategy. PAE children are also reported to display impaired organization during the encoding of diagrams such as the ROCF (Pei et al., 2011). Furthermore, PAE individuals also demonstrate poor strategies when retrieving memories. That is, some studies that have reported impaired encoding but spared retention, have noted impairments in recognition memory, which suggests an executive impairment in the form of an inefficient retrieval strategy (Crocker et al., 2011; Lewis et al., 2015; Mattson et al., 1998).

Identification of the nature of the memory impairment has clear implications when forming intervention strategies for PAE individuals. That is, if PAE individuals display impairments in learning (encoding) information, then interventions might place more emphasis on forming strategies to aid in the learning of new material. For instance, providing cues for attention and the repeated presentation of to-be-encoded information might provide increased opportunity for learning as exposure to the information would be longer. Teaching learning strategies such as clustering or chunking information might ease the burden on working memory and aid PAE individuals in encoding, whereas the use of external memory reminders and memory cues may help PAE individual's memory retrieval (Manji et al., 2009).

Recent studies have reported promising findings with regard to the effects of intervention programs improving executive functioning. Post-intervention improvements have been reported in executive function domains such as inhibitory control and social cognition (Nash et al., 2015; Wells, Chasnoff, Schmidt, Telford, & Schwartz, 2012). These related improvements have also been associated with increase in frontal gray matter in PAE children (Soh et al., 2015). Unfortunately, studies reporting on interventions aiming to improve memory in PAE individuals are scarce. Recently, Loomes, Rasmussen, Pei, Manji and Andrew (2008) reported that teaching FASD children verbal rehearsal strategies increased their performance on a digit span task (attention and working memory) in comparison to a control FASD group. However, it is yet to be established if such training improves episodic memory.

Limitations

This review is subject to several notable limitations. First, studies included in the review varied in amount and timing of alcohol exposure between participant samples. Both amount and timing of alcohol exposure are reported to be important determinants of physical, behavioral, and cognitive effects of PAE (e.g., see Coles et al., 2015; Flak et al., 2014; Meintjes et al., 2014; O'Leary, Zubrick, Taylor, Dixon, & Bower, 2009; O'Leary et al., 2010; Sood et al., 2001). Second, the reviewed studies also differed in recruitment methods as some utilized longitudinal samples, whereas the majority used clinically referred samples. Clinically referred samples are often recruited retrospectively and thus important information relating to the pre-and postnatal environment, such as maternal drug use during pregnancy, is not available. Third, information pertaining to alcohol consumption during pregnancy is commonly collected through self-report measures. These self-reports are potentially problematic due to the time interval between alcohol consumption and recall, and the social stigma associated with drinking during pregnancy (Flak et al., 2014). Fourth, many of the reviewed studies used participant samples with wide age ranges. As memory performance improves throughout childhood, using wide age ranges makes interpretation of the effects of PAE on memory performance difficult as it is confounded by developmental differences. Fifth, a number of the reviewed studies reused samples. Thus, samples were not always independent of each other and, therefore, possibly subject to the same confounding variables. In addition, the reuse of samples limits the generalizability of findings. Sixth, the methodology used to examine episodic memory in the reviewed studies varied considerably. This variation makes it difficult to distinguish whether memory performance is attributed to PAE or differences in the individual attributes of the tests. Lastly, this review is subject to potential bias as it was limited to articles published in English.

In conclusion, the studies reviewed earlier demonstrate that, in general, PAE results in impaired episodic memory performance in affected school-aged children, adolescents, and adults. These impairments have been demonstrated on multiple tests in both verbal and visual-spatial episodic memory and cannot be attributed to a global memory impairment. A recent pattern to emerge is that the impairments might be specific to the encoding stage of memory, whereas retention is relatively spared. A possible explanation for this pattern is that impairments in executive functioning, including impairments in attention, working memory and memory strategies, might impede learning in PAE individuals. Further research is needed to, firstly, determine if this pattern of impaired encoding and spared retrieval is consistent across memory tests other than wordlists and secondly, to determine if interventions that focus on both the encoding of memory and executive functioning improve episodic memory functioning.

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Conflict of Interest

None declared.

References

Aragón, A. S., Kalberg, W. O., Buckley, D., Barela-Scott, L. M., Tabachnick, B. G., & May, P. A. (2008). Neuropsychological study of FASD in a sample of American Indian children: Processing simple versus complex information. *Alcoholism: Clinical and Experimental Research*, *32 (12)*, 2136–2148.

Barnes, D. E., & Walker, D. W. (1981). Prenatal ethanol exposure permanently reduces the number of pyramidal neurons in rat hippocampus. *Developmental Brain Research*, *1 (3)*, 333–340.

Bauer, P. J., Dikmen, S. S., Heaton, R. K., Mungas, D., Slotkin, J., & Beaumont, J. L. (2013). Iii. Nih toolbox cognition battery (cb): Measuring episodic memory. *Monographs of the Society for Research in Child Development*, *78 (4)*, 34–48.

Becker, M., Warr-Leeper, G. A., & Leeper, H. A. (1990). Fetal alcohol syndrome: A description of oral motor, articulatory, short-term memory, grammatical, and semantic abilities. *Journal of Communication Disorders*, *23 (2)*, 97–124.

Berman, R. F., & Hannigan, J. H. (2000). Effects of prenatal alcohol exposure on the hippocampus: Spatial behavior, electrophysiology, and neuroanatomy. *Hippocampus*, *10 (1)*, 94–110.

Blanchard, B. A., Riley, E., & Hannigan, J. H. (1987). Deficits on a spatial navigation task following prenatal exposure to ethanol. *Neurotoxicology and Teratology*, *9 (3)*, 253–258.

Bonthius, D. J., & West, J. R. (1990). Alcohol-induced neuronal loss in developing rats: Increased brain damage with binge exposure. *Alcoholism: Clinical and Experimental Research*, *14 (1)*, 107–118.

Bonthius, D. J., & West, J. R. (1991). Permanent neuronal deficits in rats exposed to alcohol during the brain growth spurt. *Teratology*, *44 (2)*, 147–163.

Burden, M. J., Jacobson, S. W., & Jacobson, J. L. (2005). Relation of prenatal alcohol exposure to cognitive processing speed and efficiency in childhood. *Alcoholism: Clinical and Experimental Research*, *29 (8)*, 1473–1483.

Burden, M. J., Jacobson, S. W., Sokol, R. J., & Jacobson, J. L. (2005). Effects of prenatal alcohol exposure on attention and working memory at 7.5 years of age. *Alcoholism: Clinical and Experimental Research*, *29 (3)*, 443–452.

Buschke, H., & Fuld, P. A. (1974). Evaluating storage, retention, and retrieval in disordered memory and learning. *Neurology*, *24 (11)*, 1019–1019.

Carmichael Olson, H. C., Feldman, J. J., Streissguth, A. P., Sampson, P. D., & Bookstein, F. L. (1998). Neuropsychological deficits in adolescents with fetal alcohol syndrome: clinical findings. *Alcohol Clin Exp Res*, *22 (9)*, 1998–2012.

Clarren, S. K., Astley, S. J., Gunderson, V. M., & Spellman, D. (1992). Cognitive and behavioral deficits in nonhuman primates associated with very early embryonic binge exposures to ethanol. *The Journal of Pediatrics*, *121 (5)*, 789–796.

Cohen, M. (1997). *Children's memory scale (CMS)*. San Antonio, TX: The Psychological Corporation.

Coles, C. D., Goldstein, F. C., Lynch, M. E., Chen, X., Kable, J. A., Johnson, K. C., et al. (2011). Memory and brain volume in adults prenatally exposed to alcohol. *Brain and Cognition*, *75 (1)*, 67–77.

Coles, C. D., Kable, J. A., Keen, C. L., Jones, K. L., Wertelecki, W., Granovska, I. V., et al. (2015). Dose and timing of prenatal alcohol exposure and maternal nutritional supplements: Developmental effects on 6-month-old infants. *Maternal and Child Health Journal*, *19 (12)*, 2605–2614.

Coles, C. D., Lynch, M. E., Kable, J. A., Johnson, K. C., & Goldstein, F. C. (2010). Verbal and nonverbal memory in adults prenatally exposed to alcohol. *Alcoholism: Clinical and Experimental Research*, *34 (5)*, 897–906.

Coles, C. D., Platzman, K. A., Raskind-Hood, C. L., Brown, R. T., Falek, A., & Smith, I. E. (1997). A comparison of children affected by prenatal alcohol exposure and attention deficit, hyperactivity disorder. *Alcoholism: Clinical and Experimental Research*, *21 (1)*, 150–161.

Crocker, N., Vaurio, L., Riley, E. P., & Mattson, S. N. (2011). Comparison of verbal learning and memory in children with heavy prenatal alcohol exposure or attention-deficit/hyperactivity disorder. *Alcoholism: Clinical and Experimental Research*, *35 (6)*, 1114–1121.

Davis, K. M., Gagnier, K. R., Moore, T. E., & Todorow, M. (2013). Cognitive aspects of fetal alcohol spectrum disorder. *Wiley Interdisciplinary Reviews: Cognitive Science*, *4 (1)*, 81–92.

Delis, D., Kramer, J., Kaplan, E., & Ober, B. (1994). *Manual for the California Verbal Learning Test-Children's Version*. San Antonio, TX: The Psychological Corporation.

Delis, D., Kramer, J. H., Kaplan, E., & Ober, B. A. (1987). *CVLT, California Verbal Learning Test: Adult version: Manual*. San Antonio, TX: Psychological Corporation.

Donald, K. A., Eastman, E., Howells, F. M., Adnams, C., Riley, E. P., Woods, R. P., et al. (2015). Neuroimaging effects of prenatal alcohol exposure on the developing human brain: A magnetic resonance imaging review. *Acta Neuropsychiatrica*, *27 (5)*, 251–269.

Ellis, N. R., Katz, E., & Williams, J. E. (1987). Developmental aspects of memory for spatial location. *Journal of Experimental Child Psychology*, *44 (3)*, 401–412.

Ellis, N. R., Woodley-Zanthos, P., & Dulaney, C. L. (1989). Memory for spatial location in children, adults, and mentally retarded persons. *American Journal on Mental Retardation*, *93 (5)*, 521–526.

Flak, A. L., Su, S., Bertrand, J., Denny, C. H., Kesmodel, U. S., & Cogswell, M. E. (2014). The association of mild, moderate, and binge prenatal alcohol exposure and child neuropsychological outcomes: A meta-analysis. *Alcoholism: Clinical and Experimental Research*, *38 (1)*, 214–226.

Fletcher, J. M. (1985). Memory for verbal and nonverbal stimuli in learning disability subgroups: Analysis by selective reminding. *Journal of Experimental Child Psychology*, *40 (2)*, 244–259.

Frankland, P. W., & Bontempi, B. (2005). The organization of recent and remote memories. *Nature Reviews Neuroscience*, *6 (2)*, 119–130.

Fryer, S. L., Mattson, S. N., Jernigan, T. L., Archibald, S. L., Jones, K. L., & Riley, E. P. (2012). Caudate volume predicts neurocognitive performance in youth with heavy prenatal alcohol exposure. *Alcoholism: Clinical and Experimental Research*, *36 (11)*, 1932–1941.

Gianoulakis, C. (1990). Rats exposed prenatally to alcohol exhibit impairment in spatial navigation test. *Behavioural Brain Research*, *36 (3)*, 217–228.

Glosser, G., Goodglass, H., & Biber, C. (1989). Assessing visual memory disorders. *Psychological Assessment: A Journal of Consulting and Clinical Psychology*, *1 (2)*, 82.

Goodlett, C. R., & Johnson, T. B. (1997). Neonatal binge ethanol exposure using intubation: Timing and dose effects on place learning. *Neurotoxicology and Teratology*, *19 (6)*, 435–446.

Goodlett, C. R., Kelly, S. J., & West, J. R. (1987). Early postnatal alcohol exposure that produces high blood alcohol levels impairs development of spatial navigation learning. *Psychobiology*, *15 (1)*, 64–74.

Green, C., Mihic, A., Nikkel, S., Stade, B., Rasmussen, C., Munoz, D., et al. (2009). Executive function deficits in children with fetal alcohol spectrum disorders (FASD) measured using the Cambridge Neuropsychological Tests Automated Battery (CANTAB). *Journal of Child Psychology and Psychiatry*, *50 (6)*, 688–697.

Hamilton, D. A., Driscoll, I., & Sutherland, R. J. (2002). Human place learning in a virtual Morris water task: Some important constraints on the flexibility of place navigation. *Behavioural Brain Research*, *129 (1)*, 159–170.

Hamilton, D. A., Kodituwakku, P., Sutherland, R. J., & Savage, D. D. (2003). Children with Fetal Alcohol Syndrome are impaired at place learning but not cued-navigation in a virtual Morris water task. *Behavioural Brain Research*, *143 (1)*, 85–94.

Hamilton, D. A., & Sutherland, R. J. (1999). Blocking in human place learning: Evidence from virtual navigation. *Psychobiology*, *27 (4)*, 453–461.

Hoff, S. F. (1988). Synaptogenesis in the hippocampal dentate gyrus: Effects of in utero ethanol exposure. *Brain Research Bulletin*, *21 (1)*, 47–54.

Isaacs, E. B., Lucas, A., Chong, W. K., Wood, S. J., Johnson, C. L., Marshall, C., et al. (2000). Hippocampal volume and everyday memory in children of very low birth weight. *Pediatric Research*, *47 (6)*, 713–720.

Jacobson, S. W., Jacobson, J. L., & Sokol, R. J. (1994). Effects of fetal alcohol exposure on infant reaction time. *Alcoholism: Clinical and Experimental Research*, *18 (5)*, 1125–1132.

Jacobson, S. W., Jacobson, J. L., Sokol, R. J., Martier, S. S., & Ager, J. W. (1993). Prenatal alcohol exposure and infant information processing ability. *Child Development*, *64 (6)*, 1706–1721.

Janzen, L. A., Nanson, J. L., & Block, G. W. (1995). Neuropsychological evaluation of preschoolers with fetal alcohol syndrome. *Neurotoxicology and Teratology*, *17 (3)*, 273–279.

Johnson, T. B., & Goodlett, C. R. (2002). Selective and enduring deficits in spatial learning after limited neonatal binge alcohol exposure in male rats. *Alcoholism: Clinical and Experimental Research*, *26 (1)*, 83–93.

Kaeming6k, K., Mulvaney, S., & Halverson, P. T. (2003). Learning following prenatal alcohol exposure: Performance on verbal and visual multitrial tasks. *Archives of Clinical Neuropsychology*, *18 (1)*, 33–47.

Kaemingk, K., & Paquette, A. (1999). Effects of prenatal alcohol exposure on neuropsychological functioning. *Developmental Neuropsychology*, *15 (1)*, 111–140.

Kaemingk, K., & Tanner Halverson, P. (2000). Spatial memory following prenatal alcohol exposure: More than a material specific memory deficit. *Child Neuropsychology*, *6 (2)*, 115–128.

Katz, E., & Ellis, N. (1991). Memory for spatial location in retarded and nonretarded persons. *Journal of Intellectual Disability Research*, *35 (3)*, 209–220.

Kelly, S. J. (1996). Effects of alcohol exposure and artificial rearing during development on septal and hippocampal neurotransmitters in adult rats. *Alcoholism: Clinical and Experimental Research*, *20 (4)*, 670–676.

Kerns, K. A., Don, A., Mateer, C. A., & Streissguth, A. P. (1997). Cognitive deficits in nonretarded adults with fetal alcohol syndrome. *Journal of Learning Disabilities*, *30 (6)*, 685–693.

Khoury, J. E., Milligan, K., & Girard, T. A. (2015). Executive functioning in children and adolescents prenatally exposed to alcohol: A meta-analytic review. *Neuropsychology Review*, *25 (2)*, 149–170.

Kim, C. K., Kalynchuk, L. E., Kornecook, T. J., Mumby, D. G., Dadgar, N. A., Pinel, J. P., et al. (1997). Object-recognition and spatial learning and memory in rats prenatally exposed to ethanol. *Behavioral Neuroscience*, *111 (5)*, 985.

Klintsova, A. Y., Helfer, J. L., Calizo, L. H., Dong, W. K., Goodlett, C. R., & Greenough, W. T. (2007). Persistent impairment of hippocampal neurogenesis in young adult rats following early postnatal alcohol exposure. *Alcoholism: Clinical and Experimental Research*, *31 (12)*, 2073–2082.

Kodituwakku, P., & Kodituwakku, E. (2014). Cognitive and behavioral profiles of children with fetal alcohol spectrum disorders. *Current Developmental Disorders Reports*, *1 (3)*, 149–160.

Kodituwakku, P. W. (2009). Neurocognitive profile in children with fetal alcohol spectrum disorders. *Developmental Disabilities Research Reviews*, *15 (3)*, 218–224.

Korkman, M., Kettunen, S., & Autti-Rämö, I. (2003). Neurocognitive impairment in early adolescence following prenatal alcohol exposure of varying duration. *Child Neuropsychology*, *9 (2)*, 117–128.

Kully-Martens, K., Pei, J., Job, J., & Rasmussen, C. (2012). Source monitoring in children with and without fetal alcohol spectrum disorders. *Journal of Pediatric Psychology*, *37 (7)*, 725–735.

La Fiette, M. H., Carlos, R., & Riley, E. P. (1994). Effects of prenatal alcohol exposure on serial pattern performance in the rat. *Neurotoxicology and Teratology*, *16 (1)*, 41–46.

LaBar, K. S., & Cabeza, R. (2006). Cognitive neuroscience of emotional memory. *Nature Reviews Neuroscience*, *7 (1)*, 54–64.

Lewis, C. E., Thomas, K. G., Dodge, N. C., Molteno, C. D., Meintjes, E. M., Jacobson, J. L., et al. (2015). Verbal learning and memory impairment in children with fetal alcohol spectrum disorders. *Alcoholism: Clinical and Experimental Research*, *39 (4)*, 724–732.

Lhermitte, F., & Signoret, J.-L. (1972). Analyse neuropsychologique et differenciation des syndromes amnesiques. *Revue Neurologique*, *126 (3)*, 162–178.

Livy, D., Miller, E. K., Maier, S. E., & West, J. R. (2003). Fetal alcohol exposure and temporal vulnerability: Effects of binge-like alcohol exposure on the developing rat hippocampus. *Neurotoxicology and Teratology*, *25 (4)*, 447–458.

Loomes, C., Rasmussen, C., Pei, J., Manji, S., & Andrew, G. (2008). The effect of rehearsal training on working memory span of children with fetal alcohol spectrum disorder. *Research in Developmental Disabilities*, *29 (2)*, 113–124.

Maier, S. E., & West, J. R. (2001). Regional differences in cell loss associated with binge-like alcohol exposure during the first two trimesters equivalent in the rat. *Alcohol*, *23 (1)*, 49–57.

Malisza, K. L., Allman, A.-A., Shiloff, D., Jakobson, L., Longstaffe, S., & Chudley, A. E. (2005). Evaluation of spatial working memory function in children and adults with fetal alcohol spectrum disorders: A functional magnetic resonance imaging study. *Pediatric Research*, *58 (6)*, 1150–1157.

Manji, S., Pei, J., Loomes, C., & Rasmussen, C. (2009). A review of the verbal and visual memory impairments in children with foetal alcohol spectrum disorders. *Developmental Neurorehabilitation*, *12 (4)*, 239–247.

Mattson, S. N., Crocker, N., & Nguyen, T. T. (2011). Fetal alcohol spectrum disorders: Neuropsychological and behavioral features. *Neuropsychology Review*, *21 (2)*, 81–101.

Mattson, S. N., & Riley, E. P. (1999). Implicit and explicit memory functioning in children with heavy prenatal alcohol exposure. *Journal of the International Neuropsychological Society*, *5 (5)*, 462–471.

Mattson, S. N., Riley, E. P., Delis, D. C., Stern, C., & Jones, K. L. (1996). Verbal learning and memory in children with fetal alcohol syndrome. *Alcoholism: Clinical and Experimental Research*, *20 (5)*, 810–816.

Mattson, S. N., Riley, E. P., Gramling, L., Delis, D. C., & Jones, K. L. (1998). Neuropsychological comparison of alcohol-exposed children with or without physical features of fetal alcohol syndrome. *Neuropsychology*, *12 (1)*, 146.

Mattson, S. N., & Roebuck, T. M. (2002). Acquisition and retention of verbal and nonverbal information in children with heavy prenatal alcohol exposure. *Alcoholism: Clinical and Experimental Research*, *26 (6)*, 875–882.

May, P. A., Baete, A., Russo, J., Elliott, A. J., Blankenship, J., Kalberg, W. O., et al. (2014). Prevalence and characteristics of fetal alcohol spectrum disorders. *Pediatrics*, *134 (5)*, 855–866.

May, P. A., Blankenship, J., Marais, A. S., Gossage, J. P., Kalberg, W. O., Barnard, R., et al. (2013). Approaching the prevalence of the full spectrum of fetal alcohol spectrum disorders in a South African population-based study. *Alcoholism: Clinical and Experimental Research*, *37 (5)*, 818–830.

May, P. A., Gossage, J. P., Kalberg, W. O., Robinson, L. K., Buckley, D., Manning, M., et al. (2009). Prevalence and epidemiologic characteristics of FASD from various research methods with an emphasis on recent in-school studies. *Developmental Disabilities Research Reviews*, *15 (3)*, 176–192.

McCarthy, D. (1972). *McCarthy scales of children's mental abilities.* New York: The Psychological Corporation..

Meintjes, E., Narr, K., van der Kouwe, A., Molteno, C., Pirnia, T., Gutman, B., et al. (2014). A tensor-based morphometry analysis of regional differences in brain volume in relation to prenatal alcohol exposure. *NeuroImage: Clinical*, *5*, 152–160.

Miller, M. W. (1995). Generation of neurons in the rat dentate gyrus and hippocampus: Effects of prenatal and postnatal treatment with ethanol. *Alcoholism: Clinical and Experimental Research*, *19 (6)*, 1500–1509.

Milner, B. (1965). Visually-guided maze learning in man: Effects of bilateral hippocampal, bilateral frontal, and unilateral cerebral lesions. *Neuropsychologia*, *3 (4)*, 317–338.

Moore, E. M., Migliorini, R., Infante, M. A., & Riley, E. P. (2014). Fetal alcohol spectrum disorders: Recent neuroimaging findings. *Current Developmental Disorders Reports*, *1 (3)*, 161–172.

Morris, R. (1984). Developments of a water-maze procedure for studying spatial learning in the rat. *Journal of Neuroscience Methods*, *11 (1)*, 47–60.

Moss, M. B., Albert, M. S., Butters, N., & Payne, M. (1986). Differential patterns of memory loss among patients with Alzheimer's disease, Huntington's disease, and alcoholic Korsakoff's syndrome. *Archives of Neurology*, *43 (3)*, 239–246.

Nagahara, A. H., & Handa, R. J. (1997). Fetal alcohol exposure produces delay-dependent memory deficits in juvenile and adult rats. *Alcoholism: Clinical and Experimental Research*, *21 (4)*, 710–715.

Nanson, J., & Hiscock, M. (1990). Attention deficits in children exposed to alcohol prenatally. *Alcoholism: Clinical and Experimental Research*, *14 (5)*, 656–661.

Nash, K., Stevens, S., Greenbaum, R., Weiner, J., Koren, G., & Rovet, J. (2015). Improving executive functioning in children with fetal alcohol spectrum disorders. *Child Neuropsychology*, *21 (2)*, 191–209.

O'Leary, C., Zubrick, S. R., Taylor, C. L., Dixon, G., & Bower, C. (2009). Prenatal alcohol exposure and language delay in 2-year-old children: The importance of dose and timing on risk. *Pediatrics*, *123 (2)*, 547–554.

O'Leary, C. M., Nassar, N., Zubrick, S. R., Kurinczuk, J. J., Stanley, F., & Bower, C. (2010). Evidence of a complex association between dose, pattern and timing of prenatal alcohol exposure and child behaviour problems. *Addiction*, *105 (1)*, 74–86.

Osterrieth, P. A. (1944). Le test de copie d'une figure complexe. *Archives of Psychology*, *30*, 206–356.

Pei, J., Job, J., Kully-Martens, K., & Rasmussen, C. (2011). Executive function and memory in children with fetal alcohol spectrum disorder. *Child Neuropsychology*, *17 (3)*, 290–309.

Pei, J., Rinaldi, C. M., Rasmussen, C., Massey, V., & Massey, D. (2008). Memory patterns of acquisition and retention of verbal and nonverbal information in children with fetal alcohol spectrum disorders. *The Canadian Journal of Clinical Pharmacology*, *15 (1)*, e44–e56.

Penfield, W., & Milner, B. (1958). Memory deficit produced by bilateral lesions in the hippocampal zone. *AMA Archives of Neurology & Psychiatry*, *79 (5)*, 475–497.

Puglia, M. P., & Valenzuela, C. F. (2010). Repeated third trimester-equivalent ethanol exposure inhibits long-term potentiation in the hippocampal CA1 region of neonatal rats. *Alcohol*, *44 (3)*, 283–290.

Rasmussen, C. (2005). Executive functioning and working memory in fetal alcohol spectrum disorder. *Alcoholism-Clinical and Experimental Research*, *29 (8)*, 1359–1367.

Rasmussen, C., Horne, K., & Witol, A. (2006). Neurobehavioral functioning in children with fetal alcohol spectrum disorder. *Child Neuropsychology*, *12 (6)*, 453–468.

Rasmussen, C., Pei, J., Manji, S., Loomes, C., & Andrew, G. (2009). Memory strategy development in children with foetal alcohol spectrum disorders. *Developmental Neurorehabilitation*, *12 (4)*, 207–214.

Rasmussen, C., Tamana, S., Baugh, L., Andrew, G., Tough, S., & Zwaigenbaum, L. (2013). Neuropsychological impairments on the NEPSY-II among children with FASD. *Child Neuropsychology*, *19 (4)*, 337–349.

Rey, A. (1941). L'examen psychologique dans les cas d'encéphalopathie traumatique. (Les problems.). [The psychological examination in cases of traumatic encepholopathy. Problems.]. *Archives de psychologie*, *28*, 215–285.

Reyes, E., Wolfe, J., & Savage, D. D. (1989). The effects of prenatal alcohol exposure on radial arm maze performance in adult rats. *Physiology & Behavior*, *46 (1)*, 45–48.

Reynolds, C. R., & Bigler, E. D. (1994) *Test of memory and learning: Examiner's manual.* Austin, TX: Pro-ed.

Richardson, G. A., Ryan, C., Willford, J., Day, N. L., & Goldschmidt, L. (2002). Prenatal alcohol and marijuana exposure: Effects on neuropsychological outcomes at 10 years. *Neurotoxicology and Teratology*, *24 (3)*, 309–320.

Riley, E. P., Infante, M. A., & Warren, K. R. (2011). Fetal alcohol spectrum disorders: An overview. *Neuropsychology Review*, *21 (2)*, 73–80.

Roebuck-Spencer, T. M., & Mattson, S. N. (2004). Implicit strategy affects learning in children with heavy prenatal alcohol exposure. *Alcoholism: Clinical and Experimental Research*, *28 (9)*, 1424–1431.

Salmon, D. P., Shimamura, A. P., Butters, N., & Smith, S. (1988). Lexical and semantic priming deficits in patients with Alzheimer's disease. *Journal of Clinical and Experimental Neuropsychology*, *10 (4)*, 477–494.

Sampson, P. D., Streissguth, A. P., Bookstein, F. L., Little, R. E., Clarren, S. K., Dehaene, P., et al. (1997). Incidence of fetal alcohol syndrome and prevalence of alcohol-related neurodevelopmental disorder. *Teratology*, *56 (5)*, 317–326.

Savage, D. D., Becher, M., Torre, A. J., & Sutherland, R. J. (2002). Dose-dependent effects of prenatal ethanol exposure on synaptic plasticity and learning in mature offspring. *Alcoholism: Clinical and Experimental Research*, *26 (11)*, 1752–1758.

Schneider, M. L., Moore, C. F., & Adkins, M. M. (2011). The effects of prenatal alcohol exposure on behavior: Rodent and primate studies. *Neuropsychology Review*, *21 (2)*, 186–203.

Schneider, M. L., Moore, C. F., & Kraemer, G. W. (2001). Moderate alcohol during pregnancy: Learning and behavior in adolescent rhesus monkeys. *Alcoholism: Clinical and Experimental Research*, *25 (9)*, 1383–1392.

Scoville, W. B., & Milner, B. (1957). Loss of recent memory after bilateral hippocampal lesions. *Journal of Neurology, Neurosurgery, and Psychiatry*, *20 (1)*, 11.

Sheslow, D., & Adams, W. (1990). *Wide range assessment of memory and learning (WRAML)*. Wide Range Incorporated.

Smith, M. L., & Milner, B. (1981). The role of the right hippocampus in the recall of spatial location. *Neuropsychologia*, *19 (6)*, 781–793.

Soh, D. W., Skocic, J., Nash, K., Stevens, S., Turner, G. R., & Rovet, J. (2015). Self-regulation therapy increases frontal gray matter in children with fetal alcohol spectrum disorder: Evaluation by voxel-based morphometry. *Frontiers in Human Neuroscience*, *9 (108)*. doi:10.3389/fnhum.2015.00108.

Sood, B., Delaney-Black, V., Covington, C., Nordstrom-Klee, B., Ager, J., Templin, T., et al. (2001). Prenatal alcohol exposure and childhood behavior at age 6 to 7 years: I. Dose-response effect. *Pediatrics*, *108 (2)*, e34–e34.

Sowell, E. R., Lu, L. H., O'Hare, E. D., McCourt, S. T., Mattson, S. N., O'Connor, M. J., et al. (2007). Functional magnetic resonance imaging of verbal learning in children with heavy prenatal alcohol exposure. *Neuroreport*, *18 (7)*, 635–639.

Sowell, E. R., Mattson, S. N., Kan, E., Thompson, P. M., Riley, E. P., & Toga, A. W. (2008). Abnormal cortical thickness and brain–behavior correlation patterns in individuals with heavy prenatal alcohol exposure. *Cerebral Cortex*, *18 (1)*, 136–144.

Squire, L. R., & Bayley, P. J. (2007). The neuroscience of remote memory. *Current Opinion in Neurobiology*, *17 (2)*, 185–196.

Streissguth, A. P. (2007). Offspring effects of prenatal alcohol exposure from birth to 25 years: The Seattle Prospective Longitudinal Study. *Journal of Clinical Psychology in Medical Settings*, *14 (2)*, 81–101.

Streissguth, A. P., Barr, H. M., & Sampson, P. D. (1990). Moderate prenatal alcohol exposure: Effects on child IQ and learning problems at age 7 1/2 years. *Alcoholism: Clinical and Experimental Research*, *14 (5)*, 662–669.

Streissguth, A. P., Barr, H. M., Sampson, P. D., & Bookstein, F. L. (1994). Prenatal alcohol and offspring development: The first fourteen years. *Drug and Alcohol Dependence*, *36 (2)*, 89–99.

Streissguth, A. P., Sampson, P. D., Olson, H. C., Bookstein, F. L., Barr, H. M., Scott, M., et al. (1994). Maternal drinking during pregnancy: Attention and short-term memory in 14-year-old offspring—a longitudinal prospective study. *Alcoholism: Clinical and Experimental Research*, *18 (1)*, 202–218.

Sunderland, A., Harris, J., & Baddeley, A. (1984). Assessing everyday memory after severe head injury. *Everyday Memory, Actions and Absentmindedness*, 191–206.

Sutherland, R. J., & Lehmann, H. (2011). Alternative conceptions of memory consolidation and the role of the hippocampus at the systems level in rodents. *Current Opinion in Neurobiology*, *21 (3)*, 446–451.

Sutherland, R. J., McDonald, R. J., & Savage, D. D. (1997). Prenatal exposure to moderate levels of ethanol can have long-lasting effects on hippocampal synaptic plasticity in adult offspring. *Hippocampus*, *7 (2)*, 232–238.

Sutherland, R. J., McDonald, R. J., & Savage, D. D. (2000). Prenatal exposure to moderate levels of ethanol can have long-lasting effects on learning and memory in adult offspring. *Psychobiology*, *28 (4)*, 532–539.

Tulving, E. (1984). Precis of elements of episodic memory. *Behavioral and Brain Sciences*, *7 (2)*, 223–238.

Tulving, E. (1985). *Elements of episodic memory*. New York: Oxford University Press.

Uecker, A. (1993). Object and spatial memory in fetal alcohol syndrome: An assessment of hippocampal dysfunction. (Unpublished doctoral dissertation), University of Arizona, Tucson.

Uecker, A., & Nadel, L. (1996). Spatial locations gone awry: Object and spatial memory deficits in children with fetal alcohol syndrome. *Neuropsychologia*, *34 (3)*, 209–223.

Uecker, A., & Nadel, L. (1998). Spatial but not object memory impairments in children with fetal alcohol syndrome. *American Journal on Mental Retardation*, *103 (1)*, 12–18.

Vargha-Khadem, F., Gadian, D. G., Watkins, K. E., Connelly, A., Van Paesschen, W., & Mishkin, M. (1997). Differential effects of early hippocampal pathology on episodic and semantic memory. *Science*, *277 (5324)*, 376–380.

Vaurio, L., Riley, E. P., & Mattson, S. N. (2011). Neuropsychological comparison of children with heavy prenatal alcohol exposure and an IQ-matched comparison group. *Journal of the International Neuropsychological Society*, *17 (3)*, 463–473.

Viljoen, D. L., Gossage, J. P., Brooke, L., Adnams, C. M., Jones, K. L., Robinson, L. K., et al. (2005). Fetal alcohol syndrome epidemiology in a South African community: A second study of a very high prevalence area. *Journal of Studies on Alcohol*, *66 (5)*, 593.

Wacha, V. H., & Obrzut, J. E. (2007). Effects of fetal alcohol syndrome on neuropsychological function. *Journal of Developmental and Physical Disabilities*, *19 (3)*, 217–226.

Wells, A., Chasnoff, I., Schmidt, C., Telford, E., & Schwartz, L. (2012). Neurocognitive habilitation therapy for children with fetal alcohol spectrum disorders: An adaptation of the Alert Program Ò. *American Journal of Occupational Therapy*, *66*, 24–34.

West, J. R., & Hamre, K. M. (1985). Effects of alcohol exposure during different periods of development: Changes in hippocampal mossy fibers. *Developmental Brain Research*, *17 (1)*, 280–284.

Wheeler, S. M., Stevens, S. A., Sheard, E. D., & Rovet, J. F. (2012). Facial memory deficits in children with fetal alcohol spectrum disorders. *Child Neuropsychology*, *18 (4)*, 339–346.

Willford, J. A., Richardson, G. A., Leech, S. L., & Day, N. L. (2004). Verbal and visuospatial learning and memory function in children with moderate prenatal alcohol exposure. *Alcoholism: Clinical and Experimental Research*, *28 (3)*, 497–507.

Willoughby, K. A., Sheard, E. D., Nash, K., & Rovet, J. (2008). Effects of prenatal alcohol exposure on hippocampal volume, verbal learning, and verbal and spatial recall in late childhood. *Journal of the International Neuropsychological Society*, *14 (6)*, 1022–1033.

Winocur, G., Moscovitch, M., & Bontempi, B. (2010). Memory formation and long-term retention in humans and animals: Convergence towards a transformation account of hippocampal–neocortical interactions. *Neuropsychologia*, *48 (8)*, 2339–2356.