

5-2018

Adverse Childhood Experiences: Impact and Outcomes on Early Childhood Development / Fetal Alcohol Spectrum Disorder: Biological and Psychological Impacts on Children and Evidence Based Interventions

Nevena Vasovic

St. Cloud State University, nvasovic@stcloudstate.edu

Follow this and additional works at: http://repository.stcloudstate.edu/cfs_etds

Recommended Citation

Vasovic, Nevena, "Adverse Childhood Experiences: Impact and Outcomes on Early Childhood Development / Fetal Alcohol Spectrum Disorder: Biological and Psychological Impacts on Children and Evidence Based Interventions" (2018). *Culminating Projects in Child and Family Studies*. 23.

http://repository.stcloudstate.edu/cfs_etds/23

This Starred Paper is brought to you for free and open access by the Department of Child and Family Studies at theRepository at St. Cloud State. It has been accepted for inclusion in Culminating Projects in Child and Family Studies by an authorized administrator of theRepository at St. Cloud State. For more information, please contact modea@stcloudstate.edu, rswexelbaum@stcloudstate.edu.

Adverse Childhood Experiences: Impact and Outcomes on Early Childhood Development

**Fetal Alcohol Spectrum Disorder: Biological and Psychological
Impacts on Children and Evidence Based Interventions**

by Nevena Vasovic

Submitted to the Graduate Faculty of
St. Cloud State University
in Partial Fulfillment of the
Requirements for the Degree of
Master of Science in
Child and Family Studies

May 2018

Committee Members:
JoAnn Johnson, Chairperson
Jane Minnema
Dennis Mergen

**Adverse Childhood Experiences:
Impact and Outcomes on Early Childhood Development**

by Nevena Vasovic

Submitted to the Graduate Faculty of
St. Cloud State University
in Partial Fulfillment of the
Requirements for the Degree of
Master of Science in
Child and Family Studies

May 2018

Table of Contents

| | Page |
|---|------|
| List of Figures | 4 |
| Chapter | |
| 1. Introduction..... | 5 |
| ACE Overview | 5 |
| Importance of the Problem..... | 9 |
| Research Project Purpose and Research Questions | 10 |
| Electronic Search Description..... | 10 |
| Definition of Terms..... | 11 |
| 2. Literature Review..... | 14 |
| Global Volumetric Changes..... | 14 |
| Limbic Circuitry..... | 15 |
| Frontal Regions | 16 |
| Basal Ganglia and Cerebellum..... | 19 |
| Structural Connectivity | 19 |
| Manifestation of Psychological Effects | 20 |
| Cognitive Development | 20 |
| Physical Health and Development | 22 |
| Emotional and Behavioral Development | 23 |
| ACEs and Caregiver Attachment Styles | 24 |
| 3. Conclusions and Summary | 28 |
| 4. Position | 34 |

Page

References.....38

Appendixes

A. Know Your ACE Scores46

B. Brain Regions Chart.....47

List of Figures

| Figure | Page |
|--|------|
| 1. Prevalence and Distribution of ACEs in MN Summary | 7 |
| 2. Prevalence and Distribution of ACEs in MN Summary | 7 |
| 3. ACE Study Model, Division of Adult and Community Health | 8 |

Chapter 1: Introduction

ACE Overview

The United States is facing a great number of social, and economic challenges that must be met head on in hopes of creating a more prosperous future. As a country, we need to foster skilled, educated, healthy adults who can participate effectively in a global economy, and can create a sense of shared responsibility toward collectivism. Currently as a society, people are debating the importance, and quality of public education; and its capacity to prepare youth for challenges in a rapidly changing workforce.

The United States has, in the last few decades, rapidly reduced funding for many public educational institutions, including early intervention and prevention (Leachman, Masterson, & Figueroa, 2017) Fortunately, increased investments are being made in some states to promote the foundations of preschool intervention, and early childhood special education (2017 Federal Budget). The benefits of investing into early childhood education show that early intervention has positive impact on adult health, and overall future opportunities (Aos, Lieb, Mayfield, Miller, & Pennucci, 2004). As such, early experiences are an important public health issue, especially considering how early prevention and intervention can mitigate later health outcomes (Goode, 2011). Recent studies indicate that positive and negative experiences in childhood have long-term impacts on future education, mental and physical health, and overall life satisfaction. Much of the scientific research in this area has been referred to as Adverse Childhood Experiences (ACEs). ACEs have been linked to risky health behaviors, chronic health conditions, low life potential, and early death. Researchers postulate that toxic stress and/or other biological/neurological changes in the brain are responsible for these health conditions, as a

direct result of ACEs. As the number of ACEs increases, so does the risk for negative outcomes (“About Adverse Childhood Experiences”, 2016).

The origin of the Adverse Childhood Experience studies was first conducted by the Center for Disease and Control Prevention and Kaiser Permanente between 1995 and 1997, with two waves of data collection that included over 17,000 Health Maintenance Organization members from Southern California physical exam data. Subjects completed confidential surveys (*Appendix A*) regarding their childhood experiences, current health conditions (obesity, heart conditions, chronic lung diseases, cancer and so on) and behaviors such as depression, suicidal ideation, and addiction (Felitti et al, 1998). In 2008, the CDC developed a set of ACE questions for states to use in the Behavioral Risk Factor Surveillance System (BRFSS); a survey used by individual states to determine status of their resident’s health based on behavioral risk factors (“Adverse Childhood Experiences in MN”, 2013). According to the CDC, ACE risk factors include physical, sexual, and emotional abuse, physical and emotional neglect, intimate partner violence, mothers treated violently, substance misuse within household, household mental illness, parental separation or divorce, incarcerated household member, and low socio-economic status. Minnesota’s 2011 BRFSS results are similar to the findings from the first ACE study by Felitti et al, (1998), and consequent national ACE studies. Research findings show more than half of Minnesotans have experienced at least one ACE. In fact, ACEs are more prevalent among Minnesotans who did not graduate from high school, who were unmarried, and who had financial insecurity defined as unemployment, worries about mortgages, rent, or food insecurity. Minnesota ACE research indicates that ACEs frequently occur together. In Minnesota, over half of Minnesotans experiencing ACEs had more than two ACE scores on average. Additionally, Minnesotans with multiple ACEs were more likely to rate their health as fair or poor, to have

been diagnosed with depression or anxiety, report smoking and chronic drinking, have been diagnosed with asthma, and to be obese.

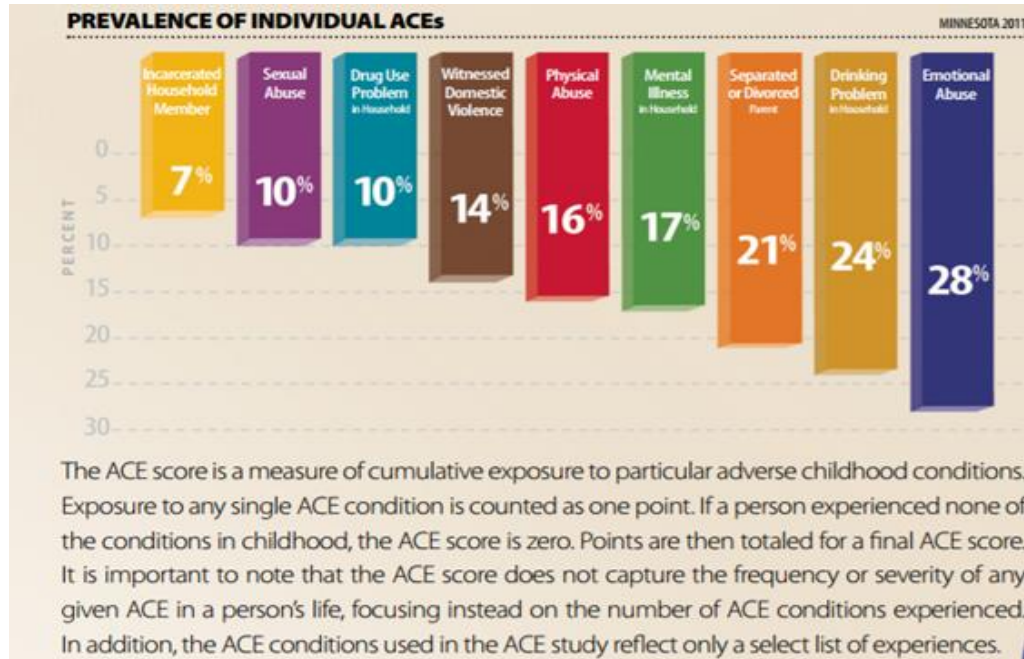


Figure 1: Prevalence and Distribution of ACEs in MN Summary (MN Report on ACES, 2011)

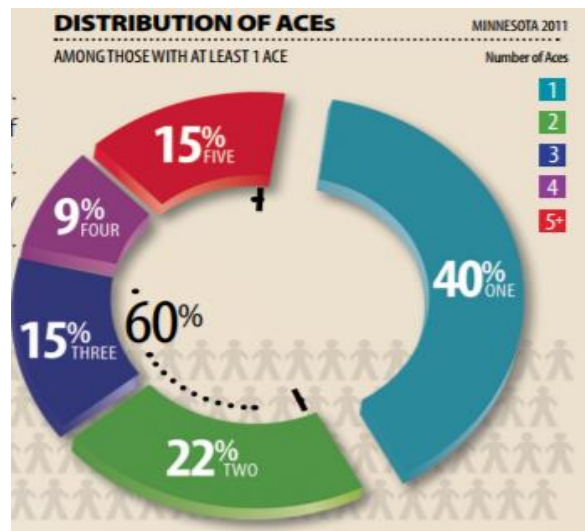


Figure 2: Prevalence and Distribution of ACEs in MN Summary (MN Report on ACES, 2011)

In the last decade and a half, many researchers have focused on impacts of the Adverse Childhood experiences on adult health and wellbeing; less research and emphasis have been placed on immediate effects of ACEs in children ages birth to third grade.

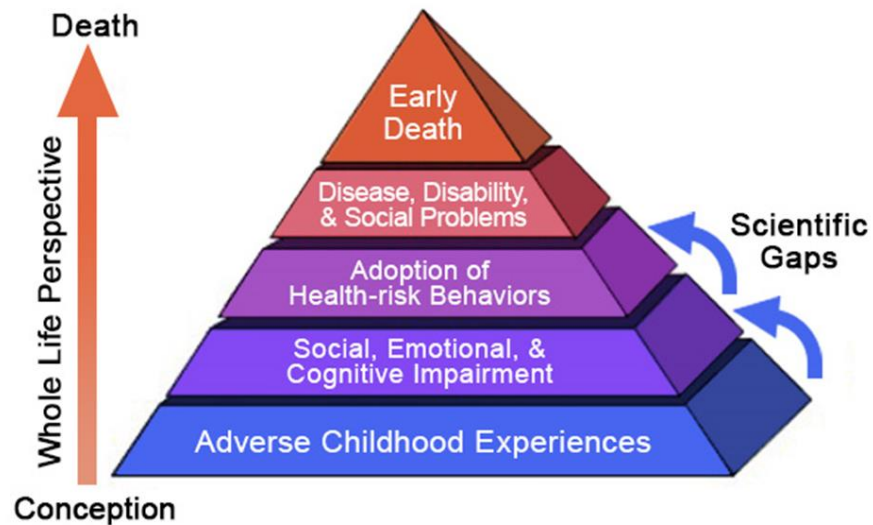


Figure 3: Ace Study Model, Division of Adult and Community Health (CDC, 2010)

Recent findings demonstrated that ACEs have neurological, biological, and psychological impacts in young children. These impacts are both measurable and observable in children's cognitive, emotional, biological and social development. Issues due to ACEs set the stage for later educational and social experiences and outcomes. Scientists have been examining how complex relationships among adverse childhood circumstances, toxic stress, and brain architecture manifest into poor physical and mental health in young children (Shonkoff et al., 2012). Another study concluded that experiencing ACEs in early childhood was associated with below-average, teacher-reported academic skills, and behavior problems by kindergarten (Jimenez, Wade, Lin, Morrow, & Reichman, 2016). Kerker et al., (2015) concluded that ACEs were associated with poor early childhood mental health, and chronic medical conditions. Especially troubling were data showing poor social development among children aged 3 to 5.

Hanson et al., (2015) determined that overall, children who experienced early life stressors (i.e., physical abuse, early neglect, or low socioeconomic status) had volumetric alterations in the amygdala and hippocampus. Individual differences in medial temporal lobe structures, particularly for the hippocampus, were associated with behavioral problems, while changes in the amygdala development lead to disruptions in fear processing portion of the brain. As a result, children would greater levels of fear and anxiety, and toxic stress syndrome (Herringa et al., 2016).

Importance of the Problem

This expanding scientific understanding on the shared roots of mental and physical health, learning, and behavior in the formative years of child's life offer a potentially transformational prospect for future of ECSE. Understanding and investigating the origins of adult diseases and addressing early symptoms during childhood are crucial first steps. Current scientific research indicates that investments in interventions to reduce ACEs early on are more likely to strengthen the foundations of physical and mental health as children grow and develop. Healthier adults can create more positive, and longer lasting benefits for all of society. Changing our current health care system from treatment of symptoms to early prevention and intervention is more cost-effective, and morally responsible. To successfully implement early intervention and prevention, and minimize the effects of ACEs, educators must consider how ACEs affect children overall. This multidimensional model of social, economic, cultural, environmental, biological, and developmental influences can help identify early onsets of delays and disabilities in children. Research of early childhood special education can lead in the realization of this idea. Early Childhood Special Educators (ECSE) work, and interact with children and parents in a variety of settings - in child care centers, school-based programs, home settings or other

educational settings. They also observe families in varied situations such as home visits, parent-child classes, or preschool classes. As such, they are the key to identifying children, birth to third grade, who are affected by ACEs, and account for them in their interventions accordingly.

Unfortunately, it is my opinion that many Early Childhood Special Educators are not sufficiently knowledgeable on the topic of ACEs. That is not to say that Early Childhood Special educators aren't familiar with individual aspects of ACEs, such as abuse, neglect, violence, and substance misuse. But the significance of these experiences, the magnitude, the overall effects, and practical interventions that can help a child may not be available to many early childhood special educators.

Research Project Purpose and Research Questions

It is my hope that in answering the research questions posed for this Starred Paper, I will further the field of ECSE and offer my fellow educators more information they can use in their educational practices, and future research. To that end, I am investigating the following research questions:

- What are immediate neurological and biological effects of ACEs on young children?
- What are the cognitive, emotional and behavioral manifestations of ACEs in young children?

Electronic Search Description

To address the previously posed questions in this research study, my focus, so far, has been on peer-reviewed articles published mostly from 2010 onwards, in journals such as Neuroscience and Biobehavioral Reviews, Biological Psychiatry, American Academy of Pediatrics, Aggression and Violent Behavior etc. Most of the articles have been acquired for free from electronic databases such as ResearchGate, JStor, AAP News & Journals Gateway, and SpringerLink. Throughout this paper I will try to review current information on the topic of

Adverse Childhood Experiences to see if what is known so far is enough to help answer research questions. Lastly, based on the data examined in the paper, I will try to summarize the information and conclude in which direction future research will need to move towards to better serve Early Childhood Special Education.

Definition of Terms

ACE – Adverse childhood experiences (ACEs) are impactful events, such as abuse and neglect, household dysfunction such as witnessing domestic violence or growing up with family members who have substance use disorders. ACEs are strongly related to the development and prevalence of a wide range of health problems throughout a person’s lifespan (“Adverse Childhood Experiences”, 2017).

ADHD - Attention-Deficit/Hyperactivity Disorder is a brain disorder marked by continuous symptoms of inattention and/or hyperactivity-impulsivity that interferes with functioning or development (“Attention-Deficit/Hyperactivity Disorder”, 2016).

BRFSS - Behavioral Risk Factor Surveillance System is a large-scale survey administered monthly throughout the year. Topics covered by the BRFSS include smoking, overweight, physical activity, fruit/vegetable consumption, chronic disease prevalence, and access to healthcare (“Behavioral Risk Factor Surveillance System”, 2017).

CBCL - The Child Behavior Checklist, also called the Achenbach System of Empirically Based Assessment, is a report form to screen for emotional, behavioral, and social problems. Questions are associated with eight different categories: anxious/depressed, withdrawn/depressed, somatic complaints, social problems, thought problems, attention problems, rule-breaking behavior, and aggressive behavior (Mazefsky, Anderson, Conner, & Minshew, 2011).

CNS - Central Nervous System is the complex of nerve tissues that controls the activities of the body. In vertebrates it comprises the brain and spinal cord (Merriam-Webster, n.d).

ELS - Early Life Stress is associated with different forms of child abuse and neglect. It has been shown to have short- and long-term effects on physiology both in the central nervous system and peripherally. ELS has been associated with risk for psychiatric disorders such as major depression, posttraumatic stress disorder and bipolar disorder (Syed & Nemeroff, 2017).

ERP – Event Related Potential are a non-invasive method of measuring brain activity during cognitive processing (“Event-related Brain Potentials”).

NSCAW II - Child and Adolescent Well-Being II is a longitudinal study intended to answer a range of fundamental questions about the functioning, service needs, and service use of children who come in contact with the child welfare system (“NSCAW II Wave 2 Report: Child Well-Being”, 2012).

ODD - Oppositional-defiant Disorder is a disruptive behavioral disorder in children and teenagers characterized by symptoms of unruly and argumentative behavior and hostile attitudes toward authority figures (“ODD - A Guide for Families by the American Academy of Child and Adolescent Psychiatry, 2009).

PFC – Prefrontal Cortex is the gray matter of the anterior part of the frontal lobe that plays a role in the regulation of complex cognitive, emotional, and behavioral functioning (Merriam-Webster, n.d).

PTSD – Post-traumatic Stress Disorder is a psychological reaction that can occur after experiencing a stressful event. It can be characterized by depression, anxiety, flashbacks, recurrent nightmares, and avoidance of reminders of the event (Merriam-Webster, n.d).

SES – Socio-economic Status is the social standing or class of an individual or group of people. It can be measured as a combination of education, income and occupation.

Chapter 2: Literature Review

Overall research on Adverse Childhood Experiences suggested that all the aspects which comprised ACEs impacted children in immediate and permanent ways (Kerker, et al., 2015). The rapid pace of child development and brain growth from birth through age 5 made a critical period of opportunity and vulnerability for children and ECSE interventions. Brain development (*Appendix B*) began with the most basic systems (brainstem, sensory motor regions) and ended with the most complex such as prefrontal cortex (Bick & Nelson, 2015). For (experienced-based) development of more complex brain areas was necessary that multitude of signals from the environment be available at certain points in development. For example, when children experienced different ranges of sounds, it furthered the growth of neural circuitry underlying auditory perception (including speech perception). This in turn was imperative for further development of higher order processes including speech and language comprehension (Cicchetti, 2015).

To better understand how ACE impacted brain development this paper focused on physiological changes in the brain as a direct result of ACEs. The research examined the impacts of ACEs on different brain structures and functions such as global volume changes, limbic structures and so on, and how these differences could have impact overall development. Furthermore, the paper also sought to understand how the effects of ACEs could impact cognitive, and emotional growth in children. Specifically, what specific portions of emotional and cognitive development showed signs of being affected by ACEs.

Global Volumetric Changes

The effects of adverse experiences on the structural properties of brain development had been examined in several studies (Bick & Nelson, 2016). In comparison to a control group,

maltreated children with and without trauma-related psychiatric disorders had exhibited reduced brain volumes, with greatest reductions observed in temporal, frontal, parietal, and occipital regions, and generally in cortical gray and white matter volume (De Brito et al, 2013; Hanson et al, 2010). Some evidence suggested that timing and chronicity of the ACEs was related to the scale of brain volume reductions; early age of onset and longer duration of ACEs had shown greater reductions in brain volumes (Morey, Haswell, Hooper, & De Bellis, 2016).

Limbic Circuitry

Understanding the relationship between limbic circuitry, emotion processing, stress regulation, learning and memory could help researchers understand effects of ACEs on this portion of the brain (Bick & Nelson, 2016). Based on previous animal studies (Lupien, McEwen, Gunnar, & Heim, 2009), research had focused on development of two main limbic structures, the amygdala and the hippocampus. Hanson et al., (2015) focused on studying behavioral problems in children who were experiencing early life stress (ELS), by measuring the effects on two brain regions involved with socioemotional functioning — amygdala and hippocampus. Studying development of these brain structures helped shed light on understanding behavioral problems following ELS. The hippocampus is involved in learning, memory, and the neuroendocrine response to stress. The amygdala then is fundamental to emotional and social information processing, with damage to this area leading to issues in understanding and responding to social stimuli (Morey et al, 2016). The results obtained from this study (Hanson et al, 2015) showed that each form of ELS assessed was related with differences in amygdala and, to a certain extent, hippocampal volumes. Smaller amygdalae were seen in children affected by physical abuse, early neglect, and low socio-economic households compared to children who were not affected by such early adversities (Hanson et al, 2015). Children exposed to threat

(angry faces in event-related potential - ERP studies) showed enlarged neural response and heightened amygdala activation (McCrory et al., 2011) even when faces were shown pre-attentively (McCrory et al., 2013). Children with heightened amygdala activation were likely to be more emotionally reactive to stress, less capable of emotional control, and more susceptible to as anxiety, hyperarousal, and dysphoria. (Dvir, Ford, Hill, & Frazier, 2014). Regarding the hippocampus, smaller volumes were seen in children affected by physical abuse relative to children in the control groups. These children exhibited less hippocampal activation during retrieval in verbal declarative memory tasks than non-maltreated children (Suárez-Pereira, Canals, & Carrión., 2015). One particularly important finding was that greater levels of stress lead to smaller volumes in both structures. Individual differences in hippocampal volumes partially mediated the contribution of ELS to increased levels of behavioral problems. Preliminary research also proposed the idea that ELS is associated to increased excitation and cell death. With greater stress or if inspected later in development, reductions in volume were expected (Fitzgerald et al, 2014).

Frontal Regions

The prefrontal cortex develops somewhat slower in comparison to other neural structures in the brain (Gogtay et al, 2006). As a result, stress due to ACEs can have a stronger impact on PFC than other areas of the brain (Arnsten, 2009). The development of this region lined up with development of complex emotional and cognitive functions, such as attention, executive function, and self-regulatory abilities (Bick & Nelson, 2015). McLaughlin, Sheridan and Lambert (2014) examined differences between deprivation (absence of predictable but complex environmental inputs) and threat (presence of experiences that signify a threat to one's physical safety) in children raised in institutional settings. These factors of deprivation were dominant for

children exposed to institutionalization, neglect, maltreatment and poverty. In opposition, experiences of threat involve events that encompass actual or threatening experiences, injuries, violations and abuse. The researchers analyzed animal biological responses on fear learning and sensory deprivation, and human research on childhood adversity and neural development. McLaughlin et al. (2014) research showed evidence that decreases in environmental input within a specific modality (e.g., hearing, vision) during development could interrupt cortical organization and decrease dendritic arborization and number of synapses within matching sensory cortex regions. In animals affected by multi-faceted deprivation, overall lack of stimulation, general decreases in cortical thickness were shown due to decreases in dendritic arborization, neuronal depth, and glia cells.

McLaughlin et al. (2014) further examined children affected by environments which share the characteristic of lacking complexity in social and cognitive inputs. Their research showed that significant social and cognitive deprivation relates to lower cognitive functions, language production and comprehension because of general decreases in grey matter volume and thickness in the brain. The greatest marked reductions were in areas of the brain which supported complex cognitive and social processing including the prefrontal cortex, superior and inferior parietal cortex, and superior temporal cortex. This lead to greater range of developmental problems, especially elevated rates of attention-deficit/hyperactivity disorder (ADHD). In terms of low parental socio-economic status (SES) research indicated decreased complexity in the amount of linguistic inputs, and lower exposure to enriching cognitive experiences in home and school environments. As such, low SES was associated with declined performance on complex cognitive tasks, including those tapping executive function and long-term memory, language skills, and cognitive and academic achievements. Also, low SES was associated with increased

levels of activation in association cortex to support performance on language and executive functioning tasks in children. Neglect, as a form of deprivation, had shown poor performance on a cognitive control tasks, and a more general form of dorsolateral the prefrontal cortex activation during studies requiring inhibitory regulation (McLaughlin et al 2014).

Research in animal studies showed general changes in the development of the prefrontal cortex following early threat, while human studies indicated that threat exposure in childhood was related with reduced volume and/or thickness of the prefrontal cortex (De Brito et al., 2013; Kelly et al., 2013). These findings which also coincided with McLaughlin et al. (2014). Researchers speculated that low development of prefrontal cortex could lead to faster pruning in this region thus causing children to perceive threat more often, have more difficult time successfully performing and completing tasks based on verbal memories, and had heightened fear reactions based on every day stimuli. In addition, a recent study documented reduced resting-state amygdala-PFC connectivity in adolescent females exposed to child abuse. Reduced amygdala lead to changed connectivity of the brain's fear circuitry and increased internalizing symptoms of anxiety and depression (Herrington et al., 2016). Changes in prefrontal cortex regions which regulate cognitive and emotional control had been noted in recent studies. For example, relative to controls, decreases in the orbitofrontal cortex, a region recognized for its role in reinforcement-based decision making and emotion regulation had been observed in children who experienced physical maltreatment (Hanson et al, 2010) or maltreatment in general (De Brito et al, 2013; Kelly et al, 2013); additional reductions had been observed in the superior frontal gyrus, a region known to support working memory (Kelly et al, 2013).

Basal Ganglia and Cerebellum

The basal ganglia are composed of multiple nuclei in the forebrain that serve a range of functions, such as regulation of motivation and reward sensitivity, while cerebellum supports higher-level learning and cognition (Bick & Nelson, 2015). Both structures have widespread impact on the frontal lobe and are part of a critical frontocerebellar network that controls behavior. Some research suggested ACEs could cause changes in the putamen (Liao et al, 2013) and in striatal circuitry of the brain (Edmiston et al, 2011). Other research had found that maltreated children with post-traumatic stress disorder (PTSD) had decreased cerebellar volumes; moreover, volume of cerebellum was positively correlated with the age of onset of the ACEs, but negatively correlated with the duration of the ACEs. Further research had found decreased size of the vermis, a sub region protracted in its development and sensitive to stress (Suárez-Pereira et al, 2015; Hanson et al, 2010).

Structural Connectivity

Studies had focused on neural pathways that supported communication between different brain regions and changes in white matter content (myelinated bundles of neurons that connect different areas of the brain) (Huang, Gundapuneedi, & Rao, 2012). The main area of research was corpus callosum, a white matter area that controls neural communication across the hemispheres. Children who have been exposed to ACEs were shown to have reduced fractional anisotropy, an indicator of white matter organization and myelination. These changes had been shown in anterior and posterior mid-body sub regions of the corpus callosum and in the splenium of the corpus callosum (Huang et al, 2012).

Manifestation of Psychological Effects

Much of the ACE research focused on the immediately observable biological changes in the brain (Bick & Nelson, 2016). Yet, it was equally important to understand how these biological changes can manifest cognitively, emotionally, behaviorally and psychologically in children. Early experiences occurred in the context of a developing brain, which lead to strong connection between neural developments, cognitive, emotional and social interactions (Van der Kolk, 2017).

Cognitive Development

In the cognitive realm, children were at increased risk for memory problems, learning difficulties, and cognitive delays, which were likely contributors to disproportionately higher rates of academic difficulties and school adjustment issues (Bick & Nelson, 2016). Additionally, affect dysregulation, defined as the impaired ability to regulate and/or tolerate negative emotional states had been associated with adverse experiences. General reduction of executive functioning (involving inhibitory control, working memory, planning, cognitive flexibility, and sustained attention) had been noted in children who suffer from ACEs (Pechtel & Pizzagalli, 2011).

In a study by Jimenez et al., (2016) Fragile Families and Child Wellbeing Study (FFCWS) was used to examine what impact ACEs had on children's developmental and behavioral outcomes. This study examined data on ACEs reported in the caregiver's 5-year follow-up combined with data on teacher-reported school performance in the last month of the child's kindergarten year. Teachers were asked to rate children's academic skills in the categories of literacy, science/social studies, and math. Additionally, teachers were also asked to describe children's classroom behavior. Researchers focused on 8 out of 10 Adverse Childhood

Experiences which fell under Centers for Disease Control and the Prevention Kaiser ACE study: 4 categories of child maltreatment (physical, sexual, psychological abuse, and neglect) and 4 categories of household dysfunction (substance use, mental illness, caregiver treated violently, and incarceration). The results analyzed from 1007 children (who had data from their children's kindergarten teachers and caregiver-reported information on ACEs) showed that 45% of the children had no ACEs, 27% had 1 ACE, 16% had 2, and 12% had ≥ 3 ACEs. The most prevalent ACEs in this research study were exposure to incarceration, physical and psychological abuse. African American children, and children with low SES were more likely than their non-African American or higher income children to experience at least one ACE. More detailed examination showed that experiencing one ACE was associated with poor teacher-reported language/literacy skills, science/social studies skills, and math skills. This study also found that one ACE was associated with teacher-reported attention problems and aggressive behavior. In a majority of cases, higher number of ACEs was associated with increased odds of below-average academic skills and behavior problems. Exposure to ≥ 3 ACEs was related to almost no emergent competencies in academic and literacy skills, and more drastic behavior problems for all outcomes examined.

Romano, Babchishi, Marqui and Frechette (2014) conducted a literature review of the childhood maltreatment characterized as ACEs and educational outcomes. Their research review indicates that children with histories of ACEs often display impairments in their academic performance, as demonstrated through indicators such as higher number of participations in special education interventions, poor performance across school subjects, lower standardized achievement measures, lower grade point averages, lower grade retention, and frequent school absences and/or changes. In terms of child characteristics, risk of academic achievement deficits

appeared to be greater for boys. Romano et al, (2014) data showed children who have experienced neglect as main form of ACEs seemed to have greater impairments in academic achievements. Furthermore, combination of two or more adverse experiences, and earlier age of onset seemed to be linked with greater academic difficulties. Researchers also found an association between childhood maltreatment, and specific academic-related outcomes such as higher-grade repetition, achievement below expectations, greater cognitive and language delays.

Physical Health and Development

Kerker et al. (2015) examined the relationship between Adverse Childhood Experiences and mental health, chronic medical conditions and development in young children. Their literature reviews showed that toxic stress (chronic, severe and prolonged levels of stress) as a direct result of ACEs was connected to neurobiological changes, as well as both structural and functional changes in the brain. In the research they questioned whether early exposure to multiple stressors had relatively immediate, and measurable consequences among young children, especially children who were at risk for removal from home. Additionally, the study wanted to explore the impact of ACEs on children who have been referred to child welfare services but have not been removed from home. The researchers studied the adverse experiences of children investigated by US child welfare agencies using data from the National Survey of Child and Adolescent Well-Being (NSCAW) II. They focused on the prevalence of ACEs among children who stayed in their families, as well as the associations between ACEs and mental health, chronic medical conditions, and social development among those children. The results come from 912 children and their caregivers. A total 13.6% scored over the problem score cutoff on the Child Behavior Checklist (CBCL) provides reports from caregivers regarding a child's competencies and behavioral/emotional problems), 35.5% were reported to have a chronic

medical condition, and 8% had a low score (<70) on the Vineland Socialization Scale. Almost all children (98.1%) were stated to have been affected by at least one ACE; 50.5% of children had 3 or more ACEs. Research also suggested that children with 3 or more ACEs, were shown to be more susceptible to physiologic disruptions, such as alterations in immune function, and increases in inflammatory markers, which are associated with many significant poor health outcomes. Researchers found that a higher number of ACEs before age 5 was related to a greater probability of mental health and chronic medical problems. However, the association between ACEs and social development was only significant among 3 to 5-year-olds. This study speculates that signs of poor health and social development are apparent in very young children and that toxic stress due to ACEs could have an immediate impact on children's welfare (Kerker et al. 2015).

Emotional and Behavioral Development

Affect regulation problems could play a role in many other psychiatric conditions, including anxiety disorders and mood disorders, specifically major depression in youth and bipolar disorder throughout the life span (Dvir et. al., 2014). In the study of 364 children who experienced ACEs, the most common diagnoses in order of frequency were separation anxiety disorder, oppositional defiant disorder, phobic disorders, PTSD, and ADHD. Other studies found problems with unmodulated aggression and impulse control, attentional and dissociative problems, and difficulty negotiating relationships with caregivers, and peers (Van der Kolk, 2017). Literature review by Ford (2017) found that children experiencing one or more ACEs were likely to show signs of anxiety disorders, sexualized behavior, oppositional or conduct problems, school problems, attachment problems, eating disorders, dissociative disorders, and personality disorders. Research indicated that early adverse experiences lead to increased risks

for maladaptive outcomes, spanning across various developmental domains. Prolonged exposure to threatening situations such as abuse was associated with long-term changes in neural circuits that underlie emotional learning. Atypical processing of emotional information – particularly facial emotion – had also been observed. Shackman and Pollak (2014) studied the emotional development in children who've experienced neglect or maltreatment. During the experiment children struggled to discriminate emotional expressions and recognize discrete emotions. Furthermore, physically abused children exhibited a response bias for angry facial expressions and displayed the most variance across emotions. These responses were believed to reflect hyper-responsiveness or vigilance to hostile emotional cues which can lead to diagnosis of attention-deficit/hyperactivity disorder and in some cases oppositional-defiant disorder (ODD). Some evidence suggested that heightened brain activity increased risk for anxiety and tendency for negative affect during adverse peer interactions (Shackman & Pollak, 2014). Recent studies had explored whether childhood maltreatment contributed toward altered neural responses to emotional stimuli (Bick & Nelson, 2016). On a behavioral level, children exposed to maltreatment showed difficulties in correctly processing social and emotional stimuli. This suggested attentional biases toward threatening emotional cues. Such biases reflected maladaptive patterns of social information processing, and/or signal difficulties with emotional or stress regulation (da Silva Ferreira, Crippa, & de Lima Osorio, 2014).

ACEs and Caregiver Attachment Styles

Due to the nature of Adverse Childhood Experiences (e.g. abuse, neglect, caregiver mental illness, and alcohol/drug abuse in family and so on), researchers looked at behavioral and psychological development occurring within a social context consisting of caregivers or group members. This context ideally should provide children with most of their developmental needs,

such as feeding, sensory stimulation, and emotional warmth. Yet, caregiver behavior and environment can equally serve as an important indication of short-term and long-term development (Kundakovic & Champagne, 2015). When it comes to adverse experiences, children often do not receive appropriate emotional, cognitive and behavioral stimuli. Thus, children must learn how to control their actions and emotions by predicting their caregivers' responses to them. They internalized the affective and cognitive characteristics of their primary relationships (Mersky, Topitzes, & Reynolds, 2013). These early patterns of attachment predicted the quality of information processing throughout life (Crittenden, 1992). Differences in the attachment styles were related with either resilience to psychological distress (e.g., secure attachment) or increased occurrence of psychopathology as observed in disorganized attachment (Kundakovic & Champagne, 2015). Secure infants learned to depend on what they feel and how they understand the world. This helped them rely on their emotions and thoughts to respond to different types of situations and experiences (Van der Kolk, 2017). Secure children learned a complex vocabulary to define different types of emotional experiences (e.g., love, hate, pleasure, disgust and anger). They were able to devote more time to describing physiological needs such as hunger and thirst, as well as emotional states than maltreated children (Cicchetti, 2015). In a majority of cases parents could assist their distressed children to establish a sense of safety and control: the security of the attachment alleviates trauma induced terror. When children were affected by adverse experiences (neglect or caregiver mental illness), their reaction was likely to imitate that of the parent – the more disorganized the parent, the more disorganized the child. When distress was not addressed properly, or when the caregivers are the cause of the distress, children couldn't control their arousal. As a result, children experienced problems processing, integrating and categorizing their emotions. (Murphy et. al., 2016). Neurobiological impact of

low parental care, abuse, and neglect were sometimes theorized as separate forms of adverse early-life adversities, yet there was significant interrelation between these forms of experiences. Disorganized attachment was observed in 48–80% of maltreated/abused infants (Kundakovic & Champagne, 2015) and was predictive of increased rates of personality disorder, dissociation, and self-harm (Bernard, Butzin-Dozier, Rittenhouse, & Dozier, 2010).

At the center of adverse experiences and its' biological effects was a breakdown in the ability to regulate internal states. If such distress was not addressed effectively and timely, children dissociate: the necessary sensations, affects and cognitions cannot be connected (they are broken into sensory fragments). These children had difficulties in comprehending what was happening or devising and executing suitable plans of action (Van der Kolk, 2017). Children experiencing disorganized or insecure attachment had difficulties bonding and relying on people around them. They had issues controlling their emotional states by themselves and experience high levels of anxiety, irritation and longing. Such feelings could precipitate dissociative states or self-defeating aggression. Because children spent prolonged periods of times under stress, they alternated between spacing out and feeling hyperarousal and trying to adjust to these adverse experiences by ignoring what they feel or what they perceive (Van der Kolk, 2017). Shipman, Zeman, Fitzgerald, & Swisher (2011) noted that girls who experienced assault believed they would receive less emotional support and were fearful of expressing negative emotional states such as sadness to parents or anger to peers. They showed more interpersonal conflict and attempted to hold back displays of such emotions. In preschool-aged children, such behaviors may appear as a variety of emotional reactions. Reduced threshold for internalized experiences combined with lack of language abilities can translate into diminished interest which is manifested as restricted play, and detachment as social withdrawal (Dvir et al., 2015). When

reminded of a trauma (sensations, physiological states, images, sounds, situations) children acted as if they were experiencing traumatic events all over again (Ford, 2017). Depending on what aspect of ACEs children were experiencing, it could be characterized as efforts to diminish perceived threat and to control emotional distress. Unless caregivers recognized the behavior of such re-enactments for what it was, they were likely to label the child as “oppositional”, ‘rebellious”, “unmotivated”, and “antisocial” (Murphy et. al., 2016). Recent studies (Boxer & Sloan-Power, 2013; Cole, Eisner, Gregory, & Ristuccia, 2013) identified that a caring relationship with an adult can be a protective factor that nurtures resiliency in young children experiencing ACEs. There was promising indication that early intervention could provide normalization in activation of key circuitry related to cognitive control; the extent to which it reduces the risk for attentional or cognitive-related difficulties should be examined in future work (Cole et al., 2013).

Chapter 3: Conclusions and Summary

Adverse Childhood Experiences, a classification that encompasses different types of trauma, is a relatively new concept in early childhood special education. Majority of studies on ACEs have focused on adults or adolescents. The results of research clearly show that adults affected by adverse experiences early in life are more likely to experience low self-esteem, chronic pain and disease, harmful behaviors (such as eating disorders, substance abuse and suicide attempts), relationship challenges and emotional difficulties (“Adverse Childhood Experiences”, 2017). In the workplace, ACEs are responsible for increased absenteeism, higher costs in health care, crisis response, mental health and behaviors resulting in criminal justice. The estimated medical costs of ACEs in adults is \$124 billion per year (Campbell, Walker, & Egede, 2016). With such high medical costs for adults, researchers and practitioners are starting to look at the effects of ACEs on younger children. Strides have been made within mental health, primary health care and education to change focus from a treatment of symptom (what’s wrong with you) to prevention and early intervention (what happened to you) approaches (Employee & Family Resources).

The field of early childhood education recognizes that neurological and biological brain development can be affected by early adverse experiences; trauma and the stress it produces can cause brain to develop atypically. Typical brain development starts with the most basic systems such as brainstem, sensory motor regions, and ends with the most complex systems such as prefrontal cortex (Bick & Nelson, 2015). Because of atypical development, child’s cognitive, behavioral and emotional responses can range from developmental delays, school problems, oppositional or conduct problems to anxiety disorders, attachment problems, eating disorders, dissociative disorders, personality disorders etc. (Dvir et al., 2014). When the effects of adverse

experiences such as toxic stress occur in young children, results show that global brain volumes are reduced compared to control groups; especially in temporal, frontal, parietal, and occipital regions, and generally in cortical gray and white matter volume (De Brito et al, 2013; Hanson et al, 2010). Further reductions were found in areas of the brain which control socioemotional functioning — amygdala and hippocampus. The hippocampus is central in learning, memory, and the neuroendocrine response to stress (Hanson et al, 2015). ACE affected children show less hippocampal activation during retrieval in verbal declarative memory tasks (Suárez-Pereira et al., 2015). Amygdala controls emotional and social information processing, with damage to this area leading to issues in understanding and responding to social stimuli (Edmiston et al, 2011). Researchers have found that many children who experience toxic stress often have overtaxed amygdala, heightened emotional reactions, and a predisposition toward negative emotions (McCrary et al., 2013).

The prefrontal cortex, while slower to develop, is similarly affected by adverse experiences. The development of PFC lines up with regulation of more complex emotional and cognitive functions, such as attention, executive function, and self-regulatory abilities (Bick & Nelson, 2015). Researchers have looked at two types of ACEs (neglect, and abuse) and its' resulting byproducts - deprivation and threat in children. McLaughlin et al. (2014) found that both animals and children who lack auditory, visual or multi-faceted deprivation shows general decreases in cortical thickness of the brain. The MRI scans show arborization of dendritic cells, neuronal depth, and glia cells corresponding to each sensory region. Researchers believe such reductions can lead to deficiency in social and cognitive abilities, due to lower cognitive functions, language production and comprehension (Ford 2017). Chronic stress, caused by children experiencing or observing abuse, seems to be positively correlated with reduced volume

and/or thickness of the PFC (De Brito et al., 2013). This reduction can further increase perception of threat causing children to struggle in performing and completing tasks based on verbal memories and have heightened fear reactions to every day stimuli (Van der Kolk, 2017). The basal ganglia and cerebellum, areas of the brain which control responses to motivation, reward sensitivity, higher-level learning and cognition, can be negatively impacted by ACEs as well (Suárez-Pereira et al, 2015; Hanson et al, 2010; Liao et al., 2013). Structural connectivity, composed of connections which support communication between different brain regions have shown disorganization between neural pathways, and reduction in myelination. The result is that children struggle in performing complex tasks and lack language abilities to express abstract ideas due to compromised connection between brain regions (Huang et al, 2012).

Being able to measure and observe changes in the brain as a direct result in ACEs has allowed current science to make a strong connection between these neurological changes and manifestations on children's cognitive, emotional and behavioral development. Due to atypically developing brain structures, researchers are finding that children have reduced capacity for executive functioning involving inhibitory control, working memory, planning, cognitive flexibility, and sustained attention (Pechtel & Pizzagalli, 2011). Jimenez et al., (2016) calculated that 55% of children had 1 or more ACE scores. Children with at least one ACE had poor skills in language/literacy, science/social studies, and math. These children also exhibited attention problems and aggressive behaviors. Exposure to ≥ 3 ACEs was related to almost no emergent competencies in academic and literacy skills, and more drastic behavior problems for all outcomes examined. Romano et al. (2014) found that children who were affected by adverse experiences were more likely to participate in special education programs. Children also had

poorer performance across school subjects, lower standardized achievement measures, lower grade point averages, lower grade retention, and frequent school absences and/or changes.

Another study found that 50.5% children at risk of being removed from home had 3 or more ACEs. Same data also suggest these children to be more susceptible to alterations in immune function, and increases in inflammatory markers, which leads to significantly poorer health outcomes. The conclusion was that higher number of ACEs before age 5 is related to a greater probability of mental health and chronic medical problems (Kerker et al. 2015).

Adverse experiences also have an impact on emotional regulation (Murphy et. al., 2016). Children who experience abuse, or neglect are at risk for developing any of a number of psychiatric disorders (Dvir et al., 2014). These can include posttraumatic stress disorder (PTSD), depression and self-harming behavior, anxiety disorders, sexualized behavior, oppositional or conduct problems, attachment problems, eating disorders, dissociative disorders, and personality disorders (Ford, 2017). Main finding suggest that enlarged neural response and heightened amygdala activation can lead to difficulties in distinguishing between emotional expressions and recognizing discrete emotions (McCrory et al., 2011; McCrory et al., 2013). Some children were more likely to label regular facial expressions as angry, had stronger reactions to stress, were less capable of emotional control, but also express anxiety, hyperarousal, and dysphoria. (Dvir et al., 2014). These responses are believed to reflect hyper-responsiveness or vigilance to hostile emotional cues which can lead to diagnosis of attention-deficit/hyperactivity disorder (ADHD), and in some cases oppositional-defiant disorder (McLaughlin et al., 2014).

Due to the nature of adverse experiences, researchers have looked at the relationship between caregivers and children, and the differences in attachment styles. Area of interest are differences between secure attachments (characterized by children without ACEs) and

disorganized attachments (characterized by children who experience at least one ACE). Secure infants can learn complex vocabulary to define different types of emotional experiences and spend more time describing physiological needs (Cicchetti, 2015). Disorganized children spend much of their time in heightened states of arousal, thus experiencing higher levels of anxiety, irritation and longing (Kundakovic, & Champagne, 2015). This can prevent them from developing the same level of language abilities and create difficulties bonding and relying on people around them. Lack of complex vocabulary skills, combined with lack of secure attachments may create dissociative states or self-defeating aggression (Van der Kolk, 2017). Children might alternate between spacing out and feeling hyperarousal. These feelings may manifest as restricted play, while detachment may manifest as social withdrawal (Dvir et al., 2015).

If proper behavior isn't attributed to the correct causes, these children may be considered oppositional, disobedient, unmotivated, and antisocial. Such labels can be used to seek diagnosis for disorders which may only address part of the problem, rather than consider the more complex relationship between biological and psychological interplay. Maslow (1943, 1954) theorized that children are motivated to realize their needs but that certain needs take priority over others. Their most basic need is for physical survival, and this will always be the first thing that motivates their actions. Once that level is fulfilled the next level up is what motivates them to develop further. Yet, if children are experiencing treat, stress, abuse, feelings of insecurity, lack of stimuli, and lack proper attachments, they are also struggling to properly advance in more complex ways. Overall, the impact of adverse childhood experiences which begins on a neurological level with reduction in volumetric brain matter, and overstimulation in others, can impact children's ability to develop cognitive abilities, successfully regulate emotions, and help

them form secure attachments. If children are lacking in deficiency needs, they will be unable to participate in the society. In conjunction with the research on the impact of adverse experiences, scientists are also focusing on resilience as a factor of overcoming them. Promising new research indicates that early education can promote brain development and normalize it enough so that children can continue to develop typically (Cole et al., 2013).

Chapter 4: Position

Being a part of early childhood special education settings has given me an opportunity to see the effects of adverse conditions on children, and to experience the challenges of working with them in an educational environment. Personally, I was cognizant that trauma and stress would leave some marks on children but for the most part I associated such trauma with adult psychopathology. At the time, I was familiar with various aspects of ACEs, yet didn't realize this could be considered one classification, and certainly didn't know the effects ACEs might have on children's neurological development. I was more experienced with cognitive, emotional and behavioral displays due to traumatic and stressful adversities, but I lacked the knowledge on interconnectedness between biology and psychology. When presented with children who were affected by some type of trauma, I put more focus on their emotional or behavioral expressions, rather than trying to understand how the whole cycle of development might be affected by such experiences. In my professional work, I lacked the proper training to address more severe cognitive delays, or emotional dysregulation. I first heard about ACEs during Minnesota's 43rd annual prevention program and sharing conference on October 18th, 2017. At the time, the practitioners were mostly focused on addressing ACEs in adults. As an ECSE student, I couldn't help but feel that better results could be accomplished if we spent more time studying how ACEs affect children, while they are still in their formative years of development; primarily birth to 5.

As I investigated this issue, it became obvious that there were two very important questions that needed to be answered. First question was whether ACEs had any measurable and observable effect on children's neurological development, and if so, how did adverse experiences manifest. Continuation of the first question had to address the implications that if there were measurable and observable impacts, did they translate into delayed or atypical cognitive,

emotional and behavioral development. Throughout the process of answering these questions, I have come to believe that ACEs indeed have clearly detectable impact on children's brain and that such changes translate into children's atypical or delayed cognitive and emotional development.

The basis of this opinion rests on studies which have tried to answer the very same questions through new and innovative methods. Literature review in general focuses on adolescence and adult development, mostly for the ease of data collection. Yet, with the improvements in neuroimaging techniques, detection of neurological changes has never been easier, even if the subjects are very young children. Research indicates that ACEs do in fact have a great effect on development in children, and that ACEs can cause various structures of the brain to develop atypically. Overall reduction in volumetric brain sizes was detected, particularly in, amygdala and the hippocampus. Much of the research has focused on these two structures because they are involved in learning, memory, emotional and social information processing, with damage to this area leading to issues in understanding and responding to social stimuli. Researchers have noted reduced sizes in amygdala and hippocampus in children with ACEs. Moreover, amygdala produces enlarged neural responses and heightened activation in children who experienced threat, abuse or who usually live with toxic stress. Children who have reduced amygdala have more trouble regulating their emotional responses and have biases for negative emotions. Prefrontal cortex, which controls attention, executive function, and self-regulatory abilities, is often affected by deprivation or threat. PFC seems to develop slower, which leads to lower cognitive functions, language production and general comprehension. Other areas such as basal ganglia, cerebellum and neural synapsis all show reduced sizes and lesser production of neural pathways, which is necessary for a typically developing brain communicate with the rest

of the brain structures. Such neurological changes, especially in young children, can lead to problems in successfully completing more complex cognitive tasks, language acquisition, behavioral and emotional responses. Considering that children typically live in environments that yield ACEs, they can have very difficult time connecting to people around them and forming secure attachments.

Despite all the research that has been published in the last decade or so, it is obvious that by and large a majority of the research is based on adolescent and adult mental and physical health. Furthermore, the term ‘adverse childhood experiences’ is not an established psychological definition, nor are ACEs recognized as diagnostic criteria; this makes it difficult to classify research under one umbrella. Consolidating research becomes a more complicated process than it would otherwise be, especially due to researchers focusing on more easily detectable ACEs. In trying to better understand neurological implications of ACEs, scientists have tried to examine the effects of stress on the brain and the subsequent reactions. This is not unusual as the effects of stress can clearly be measured and quantified, but adverse experiences consist of more than just stress (e.g. deprivation in cases on neglect or maltreatment) for which there is less research. While the science of neuroimaging allows for insight between ACEs and brain development of children, there needs to be more research into the effects of cognitive and emotional manifestations. It would also be helpful to examine longitudinal studies so that practitioners and researchers can better understand how early intervention affects children’s outcomes later in life, and what types of interventions would work better for various types of ACEs. More of this type of information would have been helpful in trying to answer the research questions posed in this paper.

The importance of further research cannot be underestimated. The more familiar people become with the idea that adverse experiences in childhood can have impacts throughout the lifetime, the more emphasis can be placed on early intervention. Understanding how children develop when exposed to adverse conditions allows early childhood educators to develop suitable interventions with the intention of helping children overcome them or counteract the effects. Currently, it's vital that many early childhood practitioners become as familiar with what Adverse Childhood Experiences are and how they can affect children. By doing so, many educators can create appropriate interventions and suitably implement appropriate interventions that target most affected developmental domains based on the type of adverse experience a child is experiencing. Until and if ACEs become a diagnostic criterion, as educators we can only focus on certain aspects of ACEs rather as a developmental disorder, but even so, mitigating early delays and promoting early brain development can help children in tangible ways in all developmental domains. Hopefully, the more early childhood practitioners are prepared to suitably address the effects of ACEs during interventions, the more likely it is to prevent children from being diagnosed later with mental health disorders which could have been mitigated if the issue was addressed suitably early on.

References

- 2017 Federal Budget *Increases to child care and early education programs*. (2017, May 16). Retrieved February 17, 2018, from <https://brightbeginningsmc.org/2017-federal-budget-increases-child-care-early-education-programs/>
- About Adverse Childhood Experiences. (2016, April 1). Retrieved October 10, 2017, from https://www.cdc.gov/violenceprevention/acestudy/about_ace.html
- About the CDC-Kaiser ACE Study, (2016, April 1). Retrieved October 10, 2017, from https://www.cdc.gov/violenceprevention/acestudy/about_ace.html
- Adverse Childhood Experiences Questioner. (2014). Retrieved October 1, 2017, from <https://www.ncjfcj.org/sites/default/files/Finding%20Your%20ACE%20Score.pdf>
- Adverse Childhood Experiences Executive Summary, (2013, February) Retrieved October 10, 2017 www.health.state.mn.us/divs/cfh/program/ace/content/document/pdf/acesum.pdf
- American Psychological Association (n.d.). Socioeconomic status. Retrieved February 01, 2018 from <http://www.apa.org/topics/socioeconomic-status/index.aspx>
- Aos, S., Lieb, R., Mayfield, J., Miller, M., & Pennucci, A. (2004). Benefits and costs of prevention and early intervention programs for youth.
- Arnsten, A. F. (2009). Stress signalling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience*, *10*(6), 410.
- Attention Deficit Hyperactivity Disorder (n.d.). Retrieved February 01, 2018, from <https://www.nimh.nih.gov/health/topics/attention-deficit-hyperactivity-disorder-adhd/index.shtml>
- Bernard, K., Butzin-Dozier, Z., Rittenhouse, J., & Dozier, M. (2010). Cortisol production patterns in young children living with birth parents vs children placed in foster care

- following involvement of Child Protective Services. *Archives of pediatrics & adolescent medicine*, 164(5), 438-443.
- Boxer, P., & Sloan-Power, E. (2013). Coping with violence: A comprehensive framework and implications for understanding resilience. *Trauma, Violence, & Abuse*, 14(3), 209-221.
- Bick, J., & Nelson, C. A. (2016). Early adverse experiences and the developing brain. *Neuropsychopharmacology*, 41(1), 177.
- Behavioral Risk Factor Surveillance System (n.d.). Retrieved February 01, 2018, from <http://www.health.state.mn.us/divs/chs/brfss/>
- Campbell, J. A., Walker, R. J., & Egede, L. E. (2016). Associations Between Adverse Childhood Experiences, High-Risk Behaviors, and Morbidity in Adulthood. *American Journal of Preventive Medicine*, 50(3), 344–352. <http://doi.org/10.1016/j.amepre.2015.07.022>
- Carrión, V. G., Haas, B. W., Garrett, A., Song, S., & Reiss, A. L. (2009). Reduced hippocampal activity in youth with posttraumatic stress symptoms: an FMRI study. *Journal of Pediatric Psychology*, 35(5), 559-569.
- Cicchetti, D. (2015). Neural plasticity, sensitive periods, and psychopathology. *Development and Psychopathology*, 27(2), 319.
- Cole, S.F., Eisner, A., Gregory, M. & Ristuccia, J. (2013). *Helping traumatized children learn: Creating and advocating for trauma sensitive schools*. Boston, MA: Massachusetts Advocates for Children.
- Crittenden, P. M. (1992). Quality of attachment in the preschool years. *Development and psychopathology*, 4(2), 209-241.

- De Brito, S. A., Viding, E., Sebastian, C. L., Kelly, P. A., Mechelli, A., Maris, H., & McCrory, E. J. (2013). Reduced orbitofrontal and temporal grey matter in a community sample of maltreated children. *Journal of child psychology and psychiatry*, *54*(1), 105-112.
- da Silva Ferreira GC, Crippa JA, de Lima Osorio F (2014). Facial emotion processing and recognition among maltreated children: a systematic literature review. *Front Psychol* *5*: 1460.
- Dvir, Y., Ford, J. D., Hill, M., & Frazier, J. A. (2014). Childhood maltreatment, emotional dysregulation, and psychiatric comorbidities. *Harvard review of psychiatry*, *22*(3), 149.
- Edmiston EE, Wang F, Mazure CM, Guiney J, Sinha R, Mayes LC et al (2011). Corticostriatal- limbic gray matter morphology in adolescents with self-reported exposure to childhood maltreatment. *Arch Pediatr Adolesc Med* *165*: 1069–1077.
- Event-related Brain Potentials (n.d.). Retrieved February 01, 2018, from <https://brainlang.georgetown.edu/research/erplab>
- Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., ... & Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American journal of preventive medicine*, *14*(4), 245-258.
- Ford, J. D. (2017). Treatment implications of altered affect regulation and information processing following child maltreatment. *Psychiatric Annals*, *35*(5), 410-419.
- Fitzgerald, P. J., Whittle, N., Flynn, S. M., Graybeal, C., Pinard, C. R., Gunduz-Cinar, O., ... & Holmes, A. (2014). Prefrontal single-unit firing associated with deficient extinction in mice. *Neurobiology of learning and memory*, *113*, 69-81.

- Sue Goode. (2011). *The Importance of Early Intervention for Infants and Toddlers with Disabilities and their Families* [Brochure]. Author. Retrieved March 01, 2018, from <http://www.nectac.org/~pdfs/pubs/importanceofearlyintervention.pdf>
- Hanson, J. L., Nacewicz, B. M., Sutterer, M. J., Cayo, A. A., Schaefer, S. M., Rudolph, K. D., ... & Davidson, R. J. (2015). Behavioral problems after early life stress: contributions of the hippocampus and amygdala. *Biological psychiatry*, 77(4), 314-323.
- Hanson, J. L., Chung, M. K., Avants, B. B., Shirliff, E. A., Gee, J. C., Davidson, R. J., & Pollak, S. D. (2010). Early stress is associated with alterations in the orbitofrontal cortex: a tensor-based morphometry investigation of brain structure and behavioral risk. *Journal of Neuroscience*, 30(22), 7466-7472.
- Herrington RJ, Burghy C.A., Stodola D.E., Fox M.E., Davidson R.J., & Essex M.J. (2016). Enhanced prefrontal-amygdala connectivity following childhood adversity as a protective mechanism against internalizing in adolescence. *Biological psychiatry: cognitive neuroscience and neuroimaging*, 1(4):326-334. doi:10.1016/j.bpsc.2016.03.003.
- Huang H., Gundapuneedi T., & Rao U. (2012). White matter disruptions in adolescents exposed to childhood maltreatment and vulnerability to psychopathology. *Neuropsychopharmacology* 37: 2693–2701.
- Gogtay, N., Nugent, T. F., Herman, D. H., Ordonez, A., Greenstein, D., Hayashi, K. M., ... & Thompson, P. M. (2006). Dynamic mapping of normal human hippocampal development. *Hippocampus*, 16(8), 664-672.
- Jimenez, M. E., Wade, R., Lin, Y., Morrow, L. M., & Reichman, N. E. (2016). Adverse experiences in early childhood and kindergarten outcomes. *Pediatrics*, peds-2015.

- Kelly, P. A., Viding, E., Wallace, G. L., Schaer, M., De Brito, S. A., Robustelli, B., & McCrory, E. J. (2013). Cortical thickness, surface area, and gyrification abnormalities in children exposed to maltreatment: neural markers of vulnerability?. *Biological psychiatry*, *74*(11), 845-852.
- Kerker, B. D., Zhang, J., Nadeem, E., Stein, R. E., Hurlburt, M. S., Heneghan, A., ... & Horwitz, S. M. (2015). Adverse childhood experiences and mental health, chronic medical conditions, and development in young children. *Academic pediatrics*, *15*(5), 510-517.
- Kundakovic, M., & Champagne, F. A. (2015). Early-life experience, epigenetics, and the developing brain. *Neuropsychopharmacology*, *40*(1), 141.
- Leachman M., Masterson, K., Figueroa E. (2017) "A Punishing Decade for School Funding." Center on Budget and Policy Priorities, www.cbpp.org/sites/default/files/atoms/files/11-29-17sfp.pdf
- Liao M, Yang F, Zhang Y, He Z, Song M, Jiang T et al (2013). Childhood maltreatment is associated with larger left thalamic gray matter volume in adolescents with generalized anxiety disorder. *PLoS One* 8: e71898.
- Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews in the Neurosciences*, *10*, 434–445.
- Maslow, A. H. (1943). A Theory of Human Motivation. *Psychological Review*, *50*(4), 370-96.
- Mazefsky, C. A., Anderson, R., Conner, C. M., & Minshew, N. (2011). Child behavior checklist scores for school-aged children with autism: preliminary evidence of patterns suggesting the need for referral. *Journal of psychopathology and behavioral assessment*, *33*(1), 31-37.

- Maslow, A. H. (1954). *Motivation and personality*. New York: Harper and Row
- McCrory, E. J., De Brito, S. A., Kelly, P. A., Bird, G., Sebastian, C. L., Mechelli, A., ... & Viding, E. (2013). Amygdala activation in maltreated children during pre-attentive emotional processing. *The British Journal of Psychiatry*, *202*(4), 269-276.
- McCrory, E. J., De Brito, S. A., Sebastian, C. L., Mechelli, A., Bird, G., Kelly, P. A., & Viding, E. (2011). Heightened neural reactivity to threat in child victims of family violence. *Current Biology*, *21*(23), R947-R948.
- McLaughlin, K. A., Sheridan, M. A., & Lambert, H. K. (2014). Childhood adversity & neural development: deprivation & threat as distinct dimensions of early experience. *Neuroscience & Biobehavioral Reviews*, *47*, 578-591.
- Merriam-Webster. (n.d.). Central Nervous System. Retrieved February 01, 2018 from <http://www.merriam-webster.com/dictionary/central%20nervous%20system>
- Merriam-Webster. (n.d.). Prefrontal Cortex. Retrieved February 01, 2018 from <https://www.merriam-webster.com/dictionary/prefrontal%20cortex>
- Merriam-Webster. (n.d.). Post-traumatic Stress Disorder. Retrieved February 01, 2018 from <https://www.merriam-webster.com/dictionary/post-traumatic%20stress%20disorder>
- Mersky, J. P., Topitzes, J., & Reynolds, A. J. (2013). Impacts of adverse childhood experiences on health, mental health, and substance use in early adulthood: a cohort study of an urban, minority sample in the US. *Child abuse & neglect*, *37*(11), 917-925.
- ODD. A Guide for Families by the American Academy of Child and Adolescent Psychiatry. 2009 (n.d.). Retrieved February 01, 2018, from https://www.aacap.org/App_Themes/AACAP/docs/resource_centers/odd/odd_resource_center_odd_guide.pdf

- Morey, R. A., Haswell, C. C., Hooper, S. R., & De Bellis, M. D. (2016). Amygdala, hippocampus, & ventral medial prefrontal cortex volumes differ in maltreated youth with and without chronic posttraumatic stress disorder. *Neuropsychopharmacology*, *41*(3), 791.
- Murphy, A., Steele, H., Steele, M., Allman, B., Kastner, T., & Dube, S. R. (2016). The clinical Adverse Childhood Experiences (ACEs) questionnaire: Implications for trauma-informed behavioral healthcare. In *Integrated early childhood behavioral health in primary care* (pp. 7-16). Springer, Cham.
- NSCAW II Wave 2 Report: Child Well-Being, (n.d, 2012). Retrieved February 01, 2018, from <https://www.acf.hhs.gov/opre/resource/nscaw-ii-wave-2-report-child-well-being>
- Pechtel, P., & Pizzagalli, D. A. (2011). Effects of early life stress on cognitive and affective function: an integrated review of human literature. *Psychopharmacology*, *214*(1), 55-70.
- Romano, E., Babchishin, L., Marquis, R., & Fréchette, S. (2015). Childhood maltreatment and educational outcomes. *Trauma, Violence, & Abuse*, *16*(4), 418-437.
- Shackman, J. E., & Pollak, S. D. (2014). Impact of physical maltreatment on the regulation of negative affect and aggression. *Development and psychopathology*, *26*(4pt1), 1021-1033.
- Shonkoff, J. P., Garner, A. S., Siegel, B. S., Dobbins, M. I., Earls, M. F., McGuinn, L., ... & Committee on Early Childhood, Adoption, and Dependent Care. (2012). The lifelong effects of early childhood adversity and toxic stress. *Pediatrics*, *129*(1), e232-e246.
- Shipman, K., Zeman, J., Fitzgerald, M., & Swisher, L. M. (2003). Regulating emotion in parent-child and peer relationships: A comparison of sexually maltreated and nonmaltreated girls. *Child Maltreatment*, *8*(3), 163-172.

- Suárez-Pereira, I., Canals, S., & Carrión, A. M. (2015). Adult newborn neurons are involved in learning acquisition and long-term memory formation: The distinct demands on temporal neurogenesis of different cognitive tasks. *Hippocampus*, 25(1), 51-61.
- Syed, S. A., & Nemeroff, C. B. (2017). Early Life Stress, Mood, and Anxiety Disorders. *Chronic Stress*, 1, 2470547017694461.
- Van der Kolk, B. A. (2017). Developmental Trauma Disorder: Toward a rational diagnosis for children with complex trauma histories. *Psychiatric annals*, 35(5), 401-408.

Appendix A. Know Your ACE Scores

Finding your ACE Score ra hbr 10 24 06

While you were growing up, during your first 18 years of life:

1. Did a parent or other adult in the household **often** ...
Swear at you, insult you, put you down, or humiliate you?
or
Act in a way that made you afraid that you might be physically hurt?
Yes No If yes enter 1 _____
2. Did a parent or other adult in the household **often** ...
Push, grab, slap, or throw something at you?
or
Ever hit you so hard that you had marks or were injured?
Yes No If yes enter 1 _____
3. Did an adult or person at least 5 years older than you ever...
Touch or fondle you or have you touch their body in a sexual way?
or
Try to or actually have oral, anal, or vaginal sex with you?
Yes No If yes enter 1 _____
4. Did you **often** feel that ...
No one in your family loved you or thought you were important or special?
or
Your family didn't look out for each other, feel close to each other, or support each other?
Yes No If yes enter 1 _____
5. Did you **often** feel that ...
You didn't have enough to eat, had to wear dirty clothes, and had no one to protect you?
or
Your parents were too drunk or high to take care of you or take you to the doctor if you needed it?
Yes No If yes enter 1 _____
6. Were your parents **ever** separated or divorced?
Yes No If yes enter 1 _____
7. Was your mother or stepmother:
Often pushed, grabbed, slapped, or had something thrown at her?
or
Sometimes or often kicked, bitten, hit with a fist, or hit with something hard?
or
Ever repeatedly hit over at least a few minutes or threatened with a gun or knife?
Yes No If yes enter 1 _____
8. Did you live with anyone who was a problem drinker or alcoholic or who used street drugs?
Yes No If yes enter 1 _____
9. Was a household member depressed or mentally ill or did a household member attempt suicide?
Yes No If yes enter 1 _____
10. Did a household member go to prison?
Yes No If yes enter 1 _____

Appendix B. Brain Regions Chart

| Brain Region | Location | Function |
|---------------------|--|--|
| Dura Mater | Part of the meninges forming the outer connective layer surrounding the brain. | Encapsulates the brain and contains the CSF (Cerebrospinal fluid) |
| Cerebrum | Forebrain. Forms the bulk of the brain's mass. | Sensory, motor and higher mental functions. |
| Cerebral Cortex | Thin layer of gray matter on the exterior of the cerebrum. | Contains 75% of the neural cell bodies. Cortex includes the convolutions of the gyri. |
| Frontal Lobe | Anterior brain. Includes the 2 hemispheres of the cerebrum. | Concentration, planning, problem-solving, cognition, frontal eye fields. |
| Parietal Lobe | Posterior to frontal lobe | Understanding speech, using grammar and word organization. |
| Occipital Lobe | Posterior of cerebral hemispheres. | Combines visual images and visual recognition of objects and shapes. |
| Temporal Lobe | Inferior to frontal and parietal lobes. | Sensory interpretation. Memory of visual and auditory patterns. |
| Broca's area | Deep, frontal lobe | Motor speech area. |
| Wernicke's area | posterior section of the <u>superior temporal gyrus</u> in the dominant cerebral hemisphere (which is the left hemisphere in about 90% of people). | perception and language processing |
| Cerebellum | Posterior / inferior | Mostly white matter. Reflex for sensory information. Coordinates complex skeletal movements. |
| Spinal Cord | Inferior brain into spinal column. CNS superhighway. | Connect the body to the brain. |
| Medulla Oblongata | Extends from pons (brain stem) into the foramen magnum of the cranium. | Vital visceral functions like cardiac, vasomotor (change in vessel diameter), and respiratory functions. |
| Pons | Anterior bulge on underside of brain stem. | Neural connection between the cerebrum and cerebellum. |
| Pituitary Gland | Attached to the base of the brain. | Regulates hormonal control |
| Infundibulum | Located behind the optic chiasma. | Attachment point for the pituitary gland. |
| Hypothalamus | Located in Diencephalon | Maintains the body's homeostasis. Regulates visceral activity and is the primary link to the endocrine /hormonal system. |
| Thalamus | Located in the midbrain within the diencephalon region. | Relay station for ascending sensory impulses incoming from the CNS. |
| Corpus Callosum | Inferior to cerebral hemispheres. | Forms the neural connection between the right and left cerebral hemispheres. |
| Limbic System | Located within the Diencephalon. Includes the thalamus, hypothalamus and basal nuclei. | Regulates emotional responses including sex drive, elation, happiness, anger, frustration. |

**Fetal Alcohol Spectrum Disorder: Biological and Psychological
Impacts on Children and Evidence Based Interventions**

by Nevena Vasovic

Submitted to the Graduate Faculty of
St. Cloud State University
in Partial Fulfillment of the
Requirements for the Degree of
Master of Science in Child and Family Studies

May 2018

Table of Contents

| | Page |
|---|------|
| List of Tables | 4 |
| Chapter | |
| 1. Introduction..... | 5 |
| Overview..... | 5 |
| Historical Perspectives..... | 5 |
| FASD Classification | 8 |
| FASD Prevalence in the US and MN | 8 |
| Statement of Intent..... | 9 |
| Literature Search Description | 10 |
| Definition of Terms..... | 11 |
| 2. Literature Review..... | 14 |
| FASD Types and Symptoms..... | 14 |
| Impact of FASD on Human Development..... | 17 |
| Impact of FASD on Cognitive Development..... | 19 |
| Impact of FASD on Adaptive Functioning..... | 22 |
| Impact of FASD on Physical Health..... | 22 |
| FASD and Intervention Strategies | 23 |
| Parent education interventions..... | 24 |
| Attention and self-regulation interventions | 26 |
| Adaptive functioning interventions..... | 27 |
| Supplements and medications..... | 29 |

| Chapter | Page |
|--------------------------------|------|
| 3. Summary and Conclusion..... | 35 |
| 4. Position | 40 |
| References..... | 42 |

List of Tables

| Tables | Page |
|---|------|
| 1. Brain Regions..... | 18 |
| 2. Summary of Parent Education and Training Intervention Strategies | 31 |
| 3. Summary of Attention and Self-Regulation Intervention Strategies | 32 |
| 4. Summary of Adaptive Functioning Intervention Strategies | 33 |
| 5. Summary of Supplements and Medication Intervention Strategies..... | 34 |

Chapter 1: Introduction

Overview

The use of alcohol during pregnancy can be a controversial topic among people even today. Generally, in the US, doctors discourage drinking during pregnancy, yet every so often we see popular news articles promoting mixed messages regarding the topic (Ruiz, 2014; Howard, 2017; Montgomery, 2018; Cook, 2017; LeWine, 2018). With so many conflicting information, women may not have a clear idea of how harmful alcohol can be to a developing fetus.

Physicians are often asked what is the safe limit for alcohol use while pregnant. Currently, the use of alcohol that is guaranteed not to cause damage to the developing fetus is unknown. What is known is that the risk of Fetal Alcohol Spectrum Disorder (FASD) raises significantly once the level of frequent drinking (4 to 8 drinks per occasion), is reached. Research also shows that at even lower levels, 1 to 3 drinks per occasion, can have some long-term impacts on attention, cognition, and learning (May et al., 2013; Olson, Feldman, Streissguth, Sampson, & Bookstein, 1998). Binge drinking while pregnant can have harmful effects due to high peak alcohol concentration and is often associated with an increased risk of FASD. The teratogenic effects of alcohol are greatest during embryogenesis; but, development can be disrupted beyond the first trimester (Young, Giesbrecht, Eskin, Aliani, & Suh, 2014).

Historical Perspectives

Perhaps this problem stems from historical roots of alcohol use. Even a few decades ago, from 1960 to 1980 doctors often used alcohol as an anti-contraction medication or an anti-suppressant in mothers who were at risk for premature labor. This practice began with Dr. Fritz Fuchs, the chairman of the department of obstetrics and gynecology at Cornell University Medical College. In the 1970s and 1980s doctors also recommended alcohol as a method to calm

the uterus during contractions in early pregnancy or during Braxton Hicks contractions. In later stages of pregnancy, alcohol was used intravenously, and caused women to experience “intoxication, nausea and vomiting, and potential alcohol poisoning, followed by hangovers when the alcohol was discontinued” (Armstrong, 2003). These practices often put mothers at risk for aspiration due to vomiting and have most certainly resulted in children born with Fetal Alcohol Spectrum Disorder (FASD) (Armstrong, 2003).

Despite such controversial practices, alcohol has been associated with birth defects even during Roman and Ancient Greek times. The earliest scientific evidence of likely associations between maternal alcohol use, and fetal damage was first recorded in 1899 by Dr. William Sullivan, a Liverpool prison physician. He observed higher rates of stillbirth for 120 alcoholic female prisoners compared to their sober female relatives; he suggested that the use of alcohol in mothers was the cause of stillbirths (Sullivan, 1899). It wasn't until late 1960s and early 1970s that researchers started to seriously examine what effects alcohol had on developing fetus. In 1968 Dr. Paul Lemoine published a study in a French medical journal on children with characteristic features whose mothers were known to be alcohol dependent (Williams & Smith, 2015). Following Lemoine's study, in 1968–1969 Christy Ulleland and colleagues from the University of Washington Medical School conducted an 18-month study examining the risk of maternal alcohol use among the offspring of 11 women who were alcohol dependent (Jones, Smith, Ulleland & Streissguth, 1973). Following these seminal studies, two researchers, Drs. Kenneth Lyons Jones and David Weyhe Smith of the University of Washington Medical School in Seattle recognized "craniofacial, limb, and cardiovascular defects associated with prenatal onset growth deficiency and developmental delay" (Jones, et al., 1973). The significance of this study relied on the fact that all eight non-related children whose mothers were classified as

addicted to alcohol came from three different ethnic groups. The main findings from all these research studies identified the patterns of malformations signifying that the damage done to children was prenatal. At the time these findings were controversial, and surprising to many experts and medical professionals. Smith and Jones first devised the term Fetal Alcohol Syndrome (FAS) in 1973 due to connecting agent of the symptoms (Williams & Smith, 2015). They rationalized the name would promote prevention, and that people would understand better the connection between maternal alcohol consumption and the symptoms; abstinence during pregnancy would follow from patient education to public awareness. Few years later, the Washington and Nantes research were validated by a study conducted in Gothenburg, Sweden in 1979. The experts in France, Sweden, and the United States were confounded by how identical all the children looked, even though none of them were related, and how they all behaved in the similar unfocused, and hyperactive way (Williams & Smith, 2015). The importance of these studies could not be underestimated; in the following nine years, many researchers focused on animal studies, especially monkey studies, to examine the link between alcohol and pregnancy. One of the most notable studies was carried out at the University of Washington Primate Center by Dr. Sterling Clarren who established that alcohol was an agent or factor that causes malformation of an embryo. By 1978, 245 cases of FAS had been noted by medical experts, and the syndrome started to be labeled as the most commonly understood cause of intellectual disability. During that time, most experts were unaware of the number of possible birth defects associated with FASD or how common it was compared to other intellectual disabilities. As further research and clinical studies were conducted, the more researchers started to understand the range of symptoms (physical, behavioral, and cognitive) that stemmed from prenatal alcohol exposure that can affect people throughout their lifetime. Since then, Fetal Alcohol Spectrum

Disorder became a broad term composed of FAS as well as other conditions ensuing from prenatal alcohol exposure (Williams & Smith, 2015).

FASD Classification

It's important to point out that FASD is not a diagnostic classification nor a medical condition. If alcohol exposure is determined, child's symptoms will fall under following diagnosis: Fetal Alcohol Syndrome, Partial Fetal Alcohol Syndrome (pFAS), Static encephalopathy/Alcohol Exposed (SE/AE), Neurobehavioral Disorder/Alcohol Exposed (ND/AE) and Neurobehavioral Disorder-Prenatal Alcohol Exposed (ND-PAE). Depending on the severity of the exposure and subsequent symptoms, the professionals will determine which diagnostic outcome would apply to a patient. The severity of brain dysfunction increases as one advances from ND/AE to SE/AE to FAS/PFAS (Astley, 2015).

FASD Prevalence in the US and MN

Due to such varying ranges of FASD, and different ways of measuring, it can be difficult to estimate with absolute certainty how prevalent FASD is in the US. Research suggests that FAS affects 6 to 9 children per 1,000 live births. But even more worrisome is that some studies also suggest the prevalence of SE/AE and ND/AE are 5 to 10-fold higher than that of FAS/PFAS. In total, current data show estimates of FASD to be 2-4% or 24-48 per 1,000 children (Williams & Smith, 2015, "Fetal Alcohol Spectrum Disorders in Minnesota", 2017). Each year, approximately 7,061 babies are born in Minnesota with prenatal alcohol exposure. If the data are accurate, the current prevalence rate to the total K-12 enrollment in public schools in Minnesota would equal 32,900+ students impacted by prenatal alcohol exposure. Such high numbers may seem questionable until we examine national data from 2011-2013 which show that about 50% of nonpregnant women report alcohol use, and about 1 in 5 admit binge drinking

in the past 30 days (binge drinking is classified as 4 or more drinks in one sitting). Among pregnant women, 1 in 10 report alcohol use and 1 in 33 reported binge drinking in the past 30 days (“Fetal Alcohol Spectrum Disorders in Minnesota”, 2017). Among pregnant women, the highest occurrence of any alcohol use was among 35-44 years old, college educated, unmarried women. Among all women who reported binge drinking in the past 30 days, pregnant women reported an average of 4.6 binge drinking episodes. This was higher than the average of 3.1 such episodes reported by nonpregnant women. Comparatively, Minnesota was one of the states with the highest percentages of women (ages 18 – 44) who reported drinking at least once a month, and the number of binge drinking episodes (“State-level estimates of alcohol use among women”, 2015). Another study conducted by Center for Disease and Control Prevention (CDC) in 2015 reported that 58.4 % of women in Minnesota drank in the past 30 days. The estimated lifetime cost of care, including social and health care services, for each child born with FASD is up to \$2.44 million, and costs Minnesota \$131 million each year. The calculated expense of raising a child with FASD is 30 times the cost of preventing the FASD (“Fetal Alcohol Spectrum Disorders in Minnesota”, 2017).

Statement of Intent

Based on this information, it’s imperative that practitioners of Early Childhood Special Education understand as much as possible about impacts FASD can have on children, and how to best help them. Thankfully, FASD research has expanded rapidly. Since the 1970s there have been more than 4000 research papers published on the topic of FASD, and at least 36% of that research has been published in the last 7 years. As it stands, educators may be familiar with aspects of Fetal Alcohol Spectrum Disorder, but a bigger push is needed to help teachers stay up to date with new theories, and intervention strategies. Considering that FASD is the most

common preventable intellectual disorder, special educators must take an active role in helping parents understand the harmful effects of alcohol during pregnancy. As providers, we have an opportunity to work with caregivers and children in variety of settings, and we can create a trusting relationship with different types of families. Through our work with children, we impact family as a whole, and our knowledge and experience can help them make safer choices. When it comes to FASD, that knowledge and experience can save children's lives.

To accomplish this endeavor, I plan to focus on the following research questions:

- What are medical, cognitive, psychological and behavioral problems caused by FASD?
- What types of interventions have proven to be effective for children with FASD?

I will try to understand the relationship between biological damages caused by FASD and psychological, behavioral, and cognitive manifestations in children; my hope is that understanding these relationships will help others create intervention strategies that address children's immediate needs. By focusing on immediate effects on children, rather than the effects FASD will have later in life, we can reduce negative impacts experienced by adolescents and adults by using appropriate empirically proven interventions. Throughout the paper I will try to review current information on the topic of FASD to see if what is known so far is enough to help answer these research questions. Lastly, based on the data examined in the paper, I will try to summarize the information and conclude in which direction future research will need to move towards to better serve Early Childhood Special Education.

Literature Search Description

To address the previously posed questions in this research study, my main focus, so far, has been on peer-reviewed articles published mostly from 2009 onwards, in scientific journals such as

Neuroscience and Biobehavioral Reviews, Biological Psychiatry, American Academy of Pediatrics, Aggression and Violent Behavior etc. Most of the articles have been acquired for free from electronic databases such as ResearchGate, JStor, AAP News & Journals Gateway, and SpringerLink.

Definition of Terms

Alcohol Related Neurodevelopmental Disorders (ARND): term coined by the Institute of Medicine in 1996 for patients who had central nervous system damage and prenatal alcohol exposure (Astley, 2015).

California Verbal Learning Test—C (CVLT-C): measure of episodic verbal learning and memory, which demonstrates sensitivity to a range of clinical conditions (Elwood, 1995).

Center for Disease and Control Prevention (CDC): is a United States federal agency under the Department of Health and Human Services (“CDC”, 2017).

Central nervous system (CNS): the complex of nerve tissues that controls the activities of the body. In vertebrates it comprises the brain and spinal cord (Merriam-Webster, n.d).

Collaborative Initiative on Fetal Alcohol Spectrum Disorders (CIFASD): Organization that works to educate and develop effective interventions and treatment methods for FASD (Gautam, Nuñez, Narr, Kan, & Sowell, 2014).

Diffusion tensor imaging (dtMRI): is an MRI-based neuroimaging technique which makes it possible to guess the location, direction, and anisotropy of the brain's white matter tracts (Warren, Hewitt, & Thomas 2011).

Digit-backward (DB) and digit-forward span (DF): A digit-span task measures working memory's number storage capacity (Gautam et al., 2014).

Fetal Alcohol Spectrum Disorder (FASD): is a term describing effects in an individual whose mother drank alcohol during pregnancy (Astley, 2015).

Fetal Alcohol Syndrome (FAS): is a birth defect syndrome caused by maternal alcohol consumption during pregnancy (Astley, 2015).

Fetal Alcohol Effects (FAE): introduced in 1978, this term was used to describe irregularities seen in people that were correlated with those caused by prenatal alcohol exposure, but the pattern was not sufficiently complete to render a diagnosis of FAS (Astley, 2015).

Functional MRI (fMRI): measures brain activity by detecting changes related with blood flow. This method depends on the fact that cerebral blood flow and neuronal activation are joined (Warren, et al., 2011).

Magnetic resonance imaging (MRI): a form of medical imaging that measures the response of the atomic nuclei of body tissues to high-frequency radio waves when placed in a strong magnetic field, and that produces images of the internal organs (Merriam-Webster, n.d).

Neurobehavioral Disorder/Alcohol Exposed (ND/AE): is an outcome classification for patients who have moderate CNS dysfunction and prenatal alcohol exposure (Astley, 2015).

Neurobehavioral Disorder-Prenatal Alcohol Exposed (ND-PAE): is a classification recently introduced by the DSM-5. It replaces the term ARND and adopts the "outcome-exposure" approach to terminology (Astley, 2015).

Partial Fetal Alcohol Syndrome (pFAS): is a classification for patients who some form of growth deficiency and/or facial features of FAS, severe CNS abnormalities and prenatal alcohol exposure (Astley, 2015).

Static encephalopathy/Alcohol Exposed (SE/AE): This classification is for patients who present with severe CNS abnormalities and prenatal alcohol exposure (Astley, 2015).

Socioeconomic status (SES): s an economic and sociological combined total measure of a person's work experience and of an individual's or family's economic and social position in relation to others, based on income, education, and occupation (Merriam-Webster, n.d).

Trail Making Test (TMT-A) and (TMT-B): The Trail Making Test is a neuropsychological test of visual attention and task switching. It consists of two parts in which the subject is instructed to connect a set of 25 dots as quickly as possible while still maintaining accuracy (Arnett & Labovitz, 1995).

Wechsler Intelligence Scale for Children, Fourth Edition (WISC-IV): a test of intellectual ability for children (Gautam et al., 2014).

Chapter 2: Literature Review

FASD Types and Symptoms

It's important to point out that FASD is not a term used for clinical diagnosis, and a child will never be diagnosed as having FASD. The last few decades have seen great changes in how medical professionals and providers classify exposure of alcohol, and its effects. In 1978 Fetal Alcohol Effects (FAE) was used to label abnormalities observed in children similar with those caused by prenatal alcohol exposure, but the pattern was not strong enough to give a diagnosis of fetal alcohol syndrome. Almost immediately the term FAE became contentious. Many medical experts expressed their concern about the clinical validity of the term FAE. Fetal Alcohol Effect implied a causal association between prenatal alcohol exposure and abnormalities observed in an individual patient that could not be scientifically confirmed. In 1996, Alcohol Related Neurodevelopmental Disorders (ARND) term was created instead. Using a term such as 'related' still implies alcohol exposure as a main cause for neurodevelopmental disorder or effect. These two terms have been slowly phased out of use (Astley, 2015). Each component of the FASD diagnosis can vary in severity depending on age, environmental variables, and the quantity and quality of prenatal alcohol exposure. This creates a problem because many patients with FASD will present different types of characteristics and disabilities. In response to these varying symptoms four diagnoses fall under the term FASD: Fetal Alcohol Syndrome (FAS), Partial FAS (pFAS), Static Encephalopathy/Alcohol Exposed (SE/AE), and Neurobehavioral Disorder/Alcohol Exposed (ND/AE).

Fetal Alcohol Syndrome is diagnosed based on the following symptoms: growth deficiency (height or weight less than 10th percentile), severe central nervous system abnormalities (structural, neurological, and/or functional abnormalities), minor facial anomalies

(small eyes, thin upper lip, smooth philtrum) and prenatal alcohol exposure (confirmed or unknown). The pervasiveness of FAS is projected to be 1 to 9 per 1,000 live births. This is roughly equal to the frequency of Down Syndrome. FAS is the principal known cause of intellectual disabilities and, unlike Down Syndrome, FAS is completely avoidable (Williams & Smith, 2015).

A second term to fall under the umbrella of FASD is Partial Fetal Alcohol Syndrome. Partial FAS is diagnosed if children potentially have some form of growth deficiency and/or facial features of FAS, have severe central nervous system (CNS) abnormalities (structural, neurological, and/or functional abnormalities), and prenatal alcohol exposure in mothers is confirmed (Astley, 2015).

Third classification of FASD is Static Encephalopathy/Alcohol Exposed (SE/AE). In this category, children who are exposed to prenatal alcohol exhibit significant abnormal conditions of the structure or function of brain tissues, which are 'static' and unchanging (neither improving nor regressing). Diagnostic criteria for SE/AE are severe Central Nervous System (CNS) abnormalities (structural, neurological, and/or severe functional abnormalities), and confirmed prenatal alcohol exposure but no FAS facial phenotype.

Last classification is Neurobehavioral Disorder/(Prenatal)Alcohol Exposed (ND/(P)AE). This is a diagnostic classification for patients who exhibit moderate CNS dysfunction and confirmed prenatal alcohol exposure but no FAS facial phenotype. To qualify for a diagnosis with ND-PAE, the mother must have consumed more than minimal levels of alcohol before the child's birth, which the American Psychiatric Association (APA) defines as more than 13 alcoholic drinks per month of pregnancy (that is, any 30-day period of pregnancy) or more than 2 alcoholic drinks in one sitting (Astley, 2015).

Presently, FAS is the only medical diagnosis within the range of FASD and has the ICD-10 code Q86.0 (“ICD-10 code” n.d.). There are several measures and approaches to diagnosing a FAS. The University of Washington 4-Digit Code is one of the most commonly used diagnostic tool. It relies on quantitative, objective measurable scales and defined case definitions. The 4 digits in the code reflect the degree of expression of the 4 key diagnostic features of FAS in the subsequent order: growth deficiency, the FAS facial features, central nervous system damage/dysfunction, and prenatal alcohol exposure. The extent of expression of each feature is ranked independently on a 4-point Likert scale with 1 reflecting complete absence of the FAS feature and 4 reflecting a strong "classic" presence of the FAS feature (Millar, 2017).

A Practical Clinical Approach to Diagnosis of Fetal Alcohol Spectrum Disorders: Clarification of the 1996 Institute of Medicine Criteria (IMO), states that for children to qualify as having FAS (with or without confirmed maternal alcohol exposure), they have to exhibit abnormalities in all domains (facial dysmorphic features, growth and brain growth or structure). In the partial FAS group (with or without confirmed maternal alcohol exposure), children must show typical facial dysmorphic features and abnormalities in one of the other areas (growth or CNS structure or function).

Fetal Alcohol Spectrum Disorders: Canadian Guidelines for Diagnosis is a combination of the University of Washington and revised IOM guidelines. It uses screening and referral, the physical examination and differential diagnosis, the neurobehavioral assessment, treatment and follow-up to diagnose children. The National Task force on FAS and FAE “Guidelines for Referral and Diagnosis” are measured and consensus-derived. They have high clinical specificity, but address only FAS; they do not support diagnostic criteria for other FASDs (Millar, 2017).

Impact of FASD on Human Development

Overall symptoms of FASD can be combined into two categories: primary features disabilities, and secondary/co-existing conditions. Primary features are mostly related to children's growth, face and brain development. The most obvious features of the FASD include reflecting small eyes (small palpebral fissures), smooth philtrum, thin upper lip, small body size for gestational age and central nervous system dysfunction (Riley, Infante, & Warren, 2011). Impact of brain dysfunction can be a continuum ranging from small neurobehavioral deficits to larger structural irregularities (Mattson et al., 2013). Furthermore, understanding the relative strengths and weaknesses in children's cognitive/ behavioral profiles can help researchers and treatment providers create interventions that target weaknesses while using strategies that engage existing strengths.

Imaging technologies, such as magnetic resonance imaging (MRI), are highly useful in clarifying alcohol's neuro-pathological effects. Due of the lifetime of learning and neurobehavioral deficits that are associated with FAS and other types of FASD, the central nervous system indisputably is the most important system negatively impacted by prenatal alcohol exposure. Imaging and neurobehavioral studies in individuals with FASD have shown that certain brain regions are particularly sensitive to prenatal alcohol exposure, while other areas seemingly less impacted. Predominantly vulnerable regions include the frontal cortex, caudate, hippocampal formation, corpus callosum, and components of the cerebellum, including the anterior cerebellar vermis. Exposure to alcohol during fetal development can lead to decrease of total brain weight (approximately 12%) due to reduced protein synthesis, which leads to decreased DNA translation. Both pre- and postnatal exposure of alcohol have shown damage to

the developing neurons in the hippocampus in rat, mouse, and human brains, leading to impaired learning and behavioral and memory function (Young, 2014).

Table 1. Brain Regions

| Brain Region | Location | Function |
|---------------------|---|--|
| Dura Mater | Part of the meninges forming the outer connective layer surrounding the brain. | Encapsulates the brain and contains the CSF (Cerebrospinal fluid) |
| Cerebrum | Forebrain. Forms the bulk of the brain's mass. | Sensory, motor and higher mental functions. |
| Cerebral Cortex | Thin layer of gray matter on the exterior of the cerebrum. | Contains 75% of the neural cell bodies. Cortex includes the convolutions of the gyri. |
| Frontal Lobe | Anterior brain. Includes the 2 hemispheres of the cerebrum. | Concentration, planning, problem-solving, cognition, frontal eye fields. |
| Parietal Lobe | Posterior to frontal lobe | Understanding speech, using grammar and word organization. |
| Occipital Lobe | Posterior of cerebral hemispheres. | Combines visual images and visual recognition of objects and shapes. |
| Temporal Lobe | Inferior to frontal and parietal lobes. | Sensory interpretation, Memory of visual and auditory patterns. |
| Broca's area | Deep, frontal lobe | Motor speech area. |
| Wernicke's area | posterior section of the superior temporal gyrus in the dominant cerebral hemisphere (which is the left hemisphere in about 90% of people). | perception and language processing |
| Cerebellum | Posterior / inferior | Mostly white matter. Reflex for sensory information. Coordinates complex skeletal movements. |
| Spinal Cord | Inferior brain into spinal column. CNS superhighway. | Connect the body to the brain. |
| Medulla Oblongata | Extends from pons (brain stem) into the foramen magnum of the cranium. | Vital visceral functions like cardiac, vasomotor (change in vessel diameter), and respiratory functions. |
| Pons | Anterior bulge on underside of brain stem. | Neural connection between the cerebrum and cerebellum. |
| Pituitary Gland | Attached to the base of the brain.. | Regulates hormonal control |
| Infundibulum | Located behind the optic chiasma. | Attachment point for the pituitary gland. |
| Hypothalamus | Located in Diencephalon | Maintains the body's homeostasis. Regulates visceral activity and is the primary link to the endocrine /hormonal system. |
| Thalamus | Located in the midbrain within the diencephalon region. | Relay station for ascending sensory impulses incoming from the CNS. |
| Corpus Callosum | Inferior to cerebral hemispheres. | Forms the neural connection between the right and left cerebral hemispheres. |
| Limbic System | Located within the Diencephalon. Includes the thalamus, hypothalamus and basal nuclei. | Regulates emotional responses including sex drive, elation, happiness, anger, frustration. |

One technology that is fairly new for examining the impacts of prenatal alcohol exposure is a specific form of MRI called diffusion tensor imaging (dtMRI). This technique allows researchers to visually track changes in bundles of nerve fibers (i.e., white matter tracts) in the brains. These tracts now are understood to be negatively affected by prenatal alcohol exposure

and can relate to changes in cognition processing in the brain. In addition, another MRI technology called functional MRI (fMRI) is being used to link behavioral and cognitive deficits with alterations in the workings of certain CNS regions (Warren, Hewitt, & Thomas 2011).

Impact of FASD on Cognitive Development

To be able to potentially answer how FASD impacts children's cognition, it's important to examine the biological implications FASD has on the children's brain. Gautam, Nuñez, Narr, Kan, & Sowell, (2014) examined the connection between brain structure and cognition in children affected by FASD. The researchers wanted to show that specific changes in the brain impacted children's cognitive abilities, so they targeted white matter development in the brain for that purpose. Gautam et al., hypothesized that rate of white matter volumetric growth would be atypical in children with FASD when compared to typically developing children. They also supposed that the rate of change in cognitive function would relate to differential white matter development between groups. In the study frontal and parietal regions were chosen to examine brain-behavior connection with an executive function battery. Children experienced attention and working memory deficits due to FASD, and these structures were hypothesized to be the ones likely related to the deficits in function. For the study, researchers chose participants who were part of the longitudinal Collaborative Initiative on Fetal Alcohol Spectrum Disorders (CIFASD), an organization that worked to educate and develop effective interventions and treatment methods for FASD. A total of 103 participants were chosen for the study, out of which 49 were classified as having FASD and 54 as controls. Mean age for controls was 11.83 (± 2.93) years and for the FASD group was 11.15 (± 2.81) years. Verbal working memory was measured through digit-backward span (DB), while attention was measured through digit-forward span (DF), both subtests of the Wechsler Intelligence Scale for Children, Fourth Edition (WISC-IV). Attention

and processing speed were measured through the Trail Making Test A (TMT-A) and mental flexibility was measured with the Trail Making Test B (TMT-B). Lastly, free recall was measured through California Verbal Learning Test - C (CVLT-C) for delayed total free-recall.

The results show participants with FASD had significantly smaller regional volumes in frontal and parietal regions. Both groups improved with age significantly on all cognitive tests, but controls performed consistently better than those with FASD on all cognitive tests except TMT-A. This study showed that while the rate of development of white matter volume is comparable with age in both groups, there are differential relationships between cognitive function and white matter change over time. For those with FASD, age-related increases in volumes were related to better cognitive function, while for controls, this positive relationship was not detected. Overall, findings propose that in typically developing children, cognitive function may rely more heavily on other neurodevelopmental changes in brain macro- or microstructure. Gautam et al., (2014) suggested that increasing white matter volume with age allows those with FASD to improve in their cognitive abilities. Specifically, increasing of white matter might lead to better communication among brain regions and consequently improve cognitive abilities. These primary brain abnormalities caused by FASD impact children's adaptive functioning, language/learning, attention, reasoning, and memory; they are called secondary disabilities/co-existing conditions and they usually appear later in life due to neurological deficits.

Language functioning in children with FASD is impaired, and children will often have difficult time understanding complex language and figures of speech; use of language for social or behavioral purposes can be difficult. Proven, Ens, and Beaudin (2016) studied school age children diagnosed with FASD to see what level of language abilities these children possessed and described their language strengths and weaknesses. A reflective chart review methodology

was applied to examine language abilities of children diagnosed with FASD. Additional data from 124 children aged 5 to 18 years, who were diagnosed with FASD between January 2005 and October 2010, were included in the study. Results from the CELF-4 language assessment tool were administered to compare the language abilities of these children. The researchers found three key findings. First, around 85% of children had some level of language impairment with almost 70% qualifying with severe language deficits. What's most troubling is that only 33% of all children in the sample had received some form of speech and language intervention. Proven et al., (2016) hypothesized that caregivers may not understand the levels of their communication impairment as most of the focus is placed on the child's other areas of difficulties (i.e., poor attention span, sleep difficulties, and behavioral issues). The findings from this study showed that language services have to be considered as a necessity given the needs of the population. Second finding indicated that a factor connected to lower scores on the CELF-4 was age. The sample showed that younger children scored better than older children. The current research, however, is not clear on whether language performance changes over time, the direction of the change, or if language remains stable. Davis, Desrocher, and Moore, (2011) in their research also showed a consistent performance decrease in language testing over time. Wyper and Rasmussen (2011) pointed to current research distinguishing language deficits in older and younger children; older children with FAS showed deficits most specifically in syntax, whereas younger children have more global language deficits. Lastly, researchers found almost no differences between the scores of participants with either Alcohol-Related Neurodevelopmental Disorder or Partial Fetal Alcohol Syndrome; language and communication scores were similar regardless of diagnosis. This is an important finding because physical traits of FAS and pFAS can provide evidence of the brain disorder and are more recognizable. But, children with ARND appear "typical" and may not be

considered by parents and teachers as having language difficulties; as a result, they would not receive any language therapy, which can be detrimental in their overall development.

Impact of FASD on Adaptive Functioning

Adaptive functioning and ability to live independently are often on a level of half the patient's chronological age; even though most have low or average intellectual abilities. Because of that adults usually don't have necessary life and social skills to function independently. Language functioning in children with FASD is impaired, and children will often have difficulty understanding complex language and figures of speech; use of language for social or behavioral purposes can be difficult. Many children with FASD have attention problems from a very young age. These problems differ from typical ADHD symptoms due to more complex comorbidities, and a different response to medications. Reasoning and memory difficulties are more obvious when a child reaches school age. Children affected by FASD are usually slow to acquire new skills and have difficulties learning from previous experiences.

Impact of FASD on Physical Health

Additionally, Moore and Riley (2015) proposed that children affected by FASD may have poorer general physical health. This conclusion stemmed from experimental animal studies showing that prenatal alcohol exposure can cause long-term immunity disorders such as autoimmune diseases (Zhang et al., 2012), and an increased vulnerability to the influenza virus (McGill et al., 2009). Another recently published systematic review (Popova et al., 2016) examined the frequency of comorbid conditions in children with FAS. The researchers found that hearing loss, visual impairment and blindness were remarkably more common in children impacted by FAS than among the general population in the US. A study by Doney et al., (2016) examined whether visual perception or fine motor coordination and visual motor impairments

were present in children affected by FASD, and whether that could account for visual-motor difficulties these children experience. Their results showed that children with FASD and control had 'average' scores in visual perception. Children with FASD had particularly lower fine motor coordination scores and higher rates of moderate impairment in fine motor coordination than children without prenatal alcohol exposure. In fact, more than half of the children with FASD had moderate impairment of fine motor coordination (52.4%), which was significantly higher than children without prenatal alcohol exposure (23.3%) and children with confirmed prenatal alcohol exposure but no diagnoses of FASD (15.4%). This study postulated that children diagnosed with FASD had difficulties coordinating or integrating these two skill sets.

FASD and Intervention Strategies

These results may be of importance in providing opportunities that enhance social, emotional, psychological and behavioral development of those with FASD. Support was particularly important, as previous research has shown that the biological parents of those with FASD had more health problems, lower education and socioeconomic status (SES) than those of typically developing children (Streissguth et al., 2004; May et al., 2013). Streissguth et al., 2004 has suggested that at least six protective factors are related with lower rates of secondary disabilities in the FASD population: living in a stable nurturing home of good quality, not having frequent changes of household, not being a victim of violence, having received disability services, and having been diagnosed before the age of 6 years. For Early Childhood Special Educators those factors can guide practice. To help children and families with FASD, practitioners must work to disseminate and implement evidence-based interventions.

Studies on interventions for individuals with FASD has grown in recent years. Petrenko and Alto (2017) suggested that while most current interventions still focus on preschool and

school-aged children, new programs continued to be developed. Programs that focused different areas, such as parent education and training, attention and self-regulation, adaptive functioning, nutrition, and medication. Practitioners, and policy makers must also consider issues such as intervention content, age of child, setting of delivery, type of provider, format of sessions, frequency and duration of sessions, and scientifically supported outcomes when choosing interventions for implementation, adaptation, or further evaluation.

As practitioners, one must consider the consequences of less-than-optimal intervention practices, how they impact children in ECSE settings and then later in life. The time for a more thorough examination of interventions and their practical use in variety of settings has come. It is important to consider interventions which address a variety of domains, such as parent education and training, attention and self-regulation, adaptive functioning, nutrition, and medication.

Parent education interventions. Programs that focus on caregiver education and training are an important first step in helping caregivers recognize and respond to the neurodevelopmental disabilities of their children. Interventions such as these work on improving caregiver-child relationship, increasing psychoeducation, building positive behavior support, finding, and accessing community resources. Interventions focused on building positive and supportive relationships if applied in infancy and early childhood have shown to an effective tool at improving family functioning and reducing adverse outcomes in children in high-risk populations (Bernard et al., 2012). This is important because children with FASD were more likely to have insecure attachments with their caregivers, and research showed that more responsive caregiving reduced the risk for depression in children, and improved neurobehavioral functioning (O'Connor, Quattlebaum, Castañeda, & Dipple, 2016).

Breaking the Cycle (BTC) focused on the relationship in substance-abusing mothers and their young children. This model arranged comprehensive services, such as home visits and community outreach. Studies of the program showed it is effective in reducing substance use, improving maternal mental health, mother-child relationships, and child outcomes (Koren, 2013). The Coaching Families (CF) program educated caregivers, helped with accessing resources, and provided support in advocacy. Additionally, CF incorporated mentoring program. Mentors were qualified individuals with two years of post-secondary education, and two years of experience in the field. This program was tailored for families with children as young as a few months to children as old as 23 years. Retrospective, pre-post evaluation of the program in Alberta, Canada found declines in reported family needs, caregiver stress and increased goal attainment (Leenaars, Denys, Henneveld, & Rasmussen, 2012).

Parent-Child Interaction Therapy (PCIT) was an evidence-based intervention which entails in vivo caregiver training. The training promoted healthy parent-child relationships, improves child's social skills, decreased problem behaviors, and helped caregivers develop manageable discipline programs. (Petrenko & Alto, 2017). Families Moving Forward (FMF) program worked to lessen children's problematic behaviors and improve family relationships by altering parenting attitudes and responses toward challenging behaviors. Mental health professionals provided in-home visits and trained caregivers in 90- min sessions every other week for 9-11 months. In a randomized controlled trial in Seattle, Washington with families raising children (5 to 11 years old) with FASD, FMF increased caregiver self-efficacy and selfcare, and reduced challenging behaviors relative to treatment as usual (Tibbett & Jeffery, 2015).

Attention and self-regulation interventions. One of the biggest challenges for children with FASD are the deficits in attention and self-regulation (Kable et al., 2015b). To help children improve in this domain, a number of programs have been developed. The Pay Attention intervention focused on visual and auditory sustained attention. The intervention was composed of 12, 30-min sessions administered daily over 3 weeks. Relative to a contact control condition, children in the Pay Attention group, showed greater improvements in sustained attention. Generalized improvements were also recorded on selective and alternating attention, and on measures of nonverbal reasoning (Vernescu, 2009).

Computerized Progressive Attention Training (CPAT) was a computer program used to help children improve skills such as sustained and selective attention by automatically adjusting the difficulty based on child's performance. Children would also receive individualized coaching to help them apply the knowledge. This intervention would take place 4 times a week in 30-min sessions, totaling 16 h of training. Intervention could be conducted in a school, and training was done by a teacher or a qualified professional with secondary education. Findings from a small study suggested CPAT can improve sustained and selective attention, spatial working memory, and reading and math fluency (Kerns, MacSween, Vander-Wekken, & Gruppuso, 2010).

Another intervention, GoFAR, incorporated a computer game, parent training, and behavioral analogue therapy sessions. GoFAR focused on helping children develop metacognitive and affective control strategies to improve adaptive functioning. The program was composed of 10, one-hour long sessions, where children went through computerized training sessions, therapist worked with parents on skill building, and behavior-analogue training was focused on parent and child interactions. Research indicated that GoFAR is effective in decreasing child disruptive behavior (Coles, Kable, Taddeo, & Strickland, 2018).

Initially created for children with self-regulation problems, the Alert program employed sensory integration and cognitive strategies to aid children in monitoring and adjusting their behavior (Williams and Shelleberger, 1996; Petrenko and Alto, 2017). This program was composed of 12, one-on-one sessions delivered by a trained clinician. The effectiveness of the intervention has been evaluated in a study with children with FASD. Improvements were recorded in inhibition naming, affect recognition, and behavioral regulation in a delayed-waitlist controlled trial (Nash et al., 2015). Another research study has shown that the Alert intervention promotes grey matter growth in the brain in critical regions for self-regulation (Soh et al., 2015). Similar intervention (based on the Alert program), called The Parents and Children Together (PACT) was founded on strategies helpful for children with traumatic brain injuries. PACT was based on parent-child sessions where participants met with a licensed mental health specialist. Research indicated that PACT is useful for improving executive functioning and child emotional problem-solving (Wells, Chasnoff, Schmidt, Telford, & Schwartz, 2012).

Adaptive functioning interventions. Studies suggested that children with FASD have impairments in adaptive functioning (Åse et al., 2012). Interventions in areas of adaptive functioning focused on safety awareness, social functioning, academic achievement, and substance use prevention. With the improvements in technology, virtual programs have been used to help children with FASD learn basic fire and street safety skills. A computer game used an animated dog to help children master basic safety skills by restricting incorrect or dangerous movements through small incremental steps. The game could be played with children as young as 3 years old, until children understand the basic principles and rules, which usually occurs in less than 30 minutes. This game was especially useful because it could be played anywhere, and generally required an adult to help a child grasp the basic ideas in the game. Research has

shown that the program improves general safety knowledge relatively quickly and that children have applied that knowledge to real world situations (Petrenko & Alton, 2017).

An intervention called Good Buddies was adapted based on a children's Friendship Training program. First used for children with FASD in Los Angeles, California, the program focused on children's neurodevelopmental and behavioral. The intervention focused on building social and friendship skills in children in a group setting. The program consisted of over 12 weeks in a clinic environment. In addition, caregivers also learned how to help develop children's friendships and social skills (Petrenko & Alto, 2017).

Good Buddies program has shown several positive outcomes, such as improved use and knowledge of social skills, decreased hostile attributions in peer entry situations, fewer behavior problems, and improved self-concepts (O'Connor, Quattlebaum, Castañeda, & Dipple, 2012). Children with FASD often experienced difficulties with academic achievement. Among the programs which focus on strengthening academic achievements, the Math and Interactive Learning Experience (MILE), incorporated individualized tutoring, at home activities, and parent instruction to help develop behavior and math skills. Parent workshops, case management, and consultation were provided to better assist each child's willingness to learn. The benefit of this program was that it could take place in a variety of settings and was composed of 6 weeks of sessions with trained instructors. In children 3-10 years old, MILE has shown evidence of improvements in math functioning post-intervention and at follow-up, with no differences in outcome among settings (Kable, Taddeo, Strickland, & Coles, 2015).

The Literacy and Language Training (LLT) intervention concentrated on language skills, phonological awareness, and literacy training. This group intervention was developed for the school setting led by a speech and language therapist. Intervention consisted of 2 sessions per

week for 30 min during the school year. Compared to children with FASD who did not receive LLT, research suggested that LLT lead to greater gains in specific categories of language and literacy (Petrenko & Alto, 2017).

Supplements and medications. Currently, there are no medical treatments which can cure the FASD, but recent studies have shown indication that certain nutritional supplements and medications may alleviate symptoms of FASD. A few kinds of biologic treatments that have been tested in this population are choline, stimulants, and neuroleptics (Petrenko & Alto, 2017). Based on animal studies researchers began studying the effect of choline supplements in humans. Researchers first focused on the effects of prenatal micronutrient supplementation (with or without supplemental choline) on cognitive and motor development in 6-month old infants. The initial results showed that micronutrient supplementation had a protective effect on cognitive, but not motor, development. Additionally, no substantial effects were recorded with the addition of choline supplementation (Kable et al., 2015).

Researchers then focused on two post-natal trials in supplementation of choline, vitamin-like essential nutrient. During a 9-month period, children ages 2-5 years old with FASD, were given daily dose of 500mg of choline in a powdered, fruit flavored drink mix. Initial results showed choline supplements were associated with improved explicit memory in younger children, suggesting a specific developmental period for this intervention (Wozniak et al., 2015). A second, 6-week trial, focused on older children (ages 5-10 years old with FASD) but did not find any substantial cognitive improvements over placebo (Nguyen, 2015). Additional studies indicate that weight adjusted rather than fixed dosages may have better memory results (Wozniak et al., 2015).

Neuroleptic (major tranquilizers) research focused on regulating effects of the medication on developmental outcomes of children participating in the Good Buddies program. The initial results were associated with overall improvements. Children taking neuroleptics demonstrated more self-control and assertion, and less problematic behaviors in comparison to children who were taking different types of medications or no medications. Children who were taking stimulants either had no change or their performance declined over time relative to other children (Frankel, Paley, Marquardt, & O'Connor, 2006).

Many children with FASD were also diagnosed with ADHD (Burd, Klug, Martsolf, & Kerbeshian, 2003) and were often given medications for ADHD symptoms. A few of the studies have looked at the efficacy of ADHD medications for children with FASD (Peadon & Elliot, 2010). A small research study of methylphenidate suggested that medication lessened hyperactivity and impulsivity but not inattention. A retroactive research also proposed that children with FASD and ADHD specifically responded to dexamphetamine over methylphenidate. Another small study suggested that taking regular stimulants can improve hyperactivity symptoms but not attention (Petrenko & Alto, 2017). This conclusion was reexamined in a study and found that ADHD medication helped with improved hyperactivity/impulsivity and opposition/defiance symptoms, but had no impact on attention (Doig, McLennan, & Gibbard, 2008). Currently, research on the role of nutrients and intervention is relatively new in FASD field. Potential nutrients have been tested in animal studies, such as vitamin A, DHA, folic acid, zinc, vitamin E, and selenium. Some research suggested that high concentrations of vitamin A can cause teratogenic effects, and there was evidence of increased cancer risks with folic acid supplementation. Because children with FASD can experience multitude of impairments, supplementation with one nutrient may not be enough

to fully alleviate the damage brought by alcohol consumption. Still, this area of study indicates potential avenues of exploration for the future research (Young, 2014).

Tables 2 - 5

Table 2. Summary of Parent Education and Training Intervention Strategies

| Parent Education and Training | | | |
|---|---|---|---|
| Program name | Program Information | Program Format | Outcomes |
| Break the Cycle - offers a range of services to mothers who have substance abuse problems. Works to help mothers establish relationships with children. | Services designed for Infants and toddlers, in the community based on parents and children's needs. | Single access mode of services, including home visits and outreach. | Reduced maternal substance use, improved maternal health, increased relationship capacity, improved child outcomes. |
| Coaching Families Program - Provides education, access to resources, and engages parents in advocacy. | Services designed for children 1-23 years of age. Offered at home with a mentor. Services based on needs. | Family goal-based Mentoring. | Decreased needs, increased goal attainment, decreased caregiver stress. |
| Parent-Child Interaction Therapy – services to improve relationships and increase social skills. Works to reduce problematic behaviors and create positive discipline skills. | Designed for children 3-7 years. Offered in clinical or home setting with a counselor. Group-based adaptation; Parent-child dyad with in vivo coaching. | 14 weeks, once a week, 90 min. | Improved child behavior and decreased parent stress. |
| Families Moving Forward Helps parents react appropriately to children's problematic behaviors to reduce the frequency and duration of said behaviors. | Services designed for children 5-11 years of age, at home with trained mental health professional, based on individual caregiver training needs. | 9-11 months, every two weeks, 90 min. | Improved parenting self efficacy and self-care, family needs met, reduced child problem behavior. |

Table 3. Summary of Attention and Self-Regulation Intervention Strategies

| Attention and Self-Regulation | | | |
|---|---|-------------------------------|--|
| Program name | Program Information | Program Format | Outcomes |
| Computerized Progressive Attention Training – Computer training on specific tasks with coaching in metacognitive strategies and support. | 1:1 coaching with an assistant, for children ages 8 – 11 years of age in school setting. | 16 h, 4 times a week, 30 min. | Improved sustained and selective attention, spatial working memory, and reading and math fluency. |
| Pay Attention Program – Using graded activities to improve visual and auditory attention. | For children ages 6-11, in school settings with individual instructions. | 12 daily 30-min sessions. | Increased visual, auditory sustained, selective, and alternating attention; nonverbal reasoning; Increased memory for numbers. |
| GoFAR – using computer games and behavioral strategies to teach children affective and metacognitive control strategies. | Created for children ages 5-10, in a clinical setting with a counselor, using parent training, child computer training and parent-child behavior analogue sessions. | 10 sessions, 1 hour. | Decreased disruptive behavior. |
| Alert Program for Self-Regulation – using sensory integration and cognitive processing activities organized in 3 successive stages to teach self-regulation skills. | Created for children ages 8-12, in a clinical setting with a counselor, based on individual needs. | 12 weeks | Improved inhibition naming, affect recognition, and behavioral regulation, brain structure changes: increases in grey matter. |
| Parents and Children Together – uses elements of Alert Program for Self-Regulation and traumatic brain injury strategies. | Created for children ages 6-12, in a clinical setting with a specialist in child and parent groups. | 12 weeks | Improved executive functioning and child emotional problem solving. |

Table 4. Summary of Adaptive Functioning Intervention Strategies

| Adaptive Functioning | | | |
|---|---|---|---|
| Program name | Program Information | Program Format | Outcomes |
| Fire and street safety virtual training - safety skills for fire safety or street safety using a video game. | Created for children ages 3-12, in any setting, with an adult supervisor based on individual needs. Offers animated character that teaches children in small incremental steps. | Game played until children accomplish objectives (<30 min). | Improved ability to generalize knowledge to behavioral setting. |
| Good Buddies – works to develop social skills. Created for children with FASD needs, and parents. Teaches parents to support social skills. | Created for children ages 6-12, in a clinical setting with a specialist in child and parent groups. | 12 weeks. | Increased social skills, decreased hostile attribution in peer entry situations, and behavior problems, and increased child self-concept. |
| Literacy and Language Training – Offers language therapy, phonological awareness, and literacy training. | Created for children ages 9-12, in school setting with a speech and language therapist in small groups. | 9 months, twice a week, 30 min. | Gains in categories of language and literacy. |
| Math Interactive Learning Experience – offers wide variety of services to help children learn. | Created for children ages 3-10, in various settings, with trained individuals. Incorporates parent training, child tutoring, and consultations. | 6 weeks | Increase in math functioning post intervention and 6 months later. |

Table 5. Summary of Supplements and Medication Intervention Strategies

| Supplements and Medication | | | |
|---|---|---------------------------------------|---|
| Program name | Program Information | Program Format | Outcomes |
| Choline supplementation pre- or post-natally. | Choline prescribed by a medical professional, to children, prenatally to 10 years of age. | Frequency and duration as Prescribed. | Improved explicit memory in younger children. |
| ADHD medication - target symptoms consistent with ADHD. | Given to children 5-12 years of age, as prescribed by a doctor. | As prescribed. | Improved hyperactivity/ impulsivity and opposition/defiance. |
| Neuroleptics – medication and social skills training. | Created for children ages 5-11 who take medication, in a clinical setting with a specialist in child and parent groups. | 12 weeks, once a week, 90 min. | Improved self-control, assertion, and problem behaviors those prescribed stimulants showed no change or deteriorated. |
| Nutrients - vitamin A, DHA, folic acid, zinc, vitamin E, and selenium | Still in animal testing stages | N/A | N/A |

Chapter 3: Summary and Conclusion

FASD is not a new phenomenon, and the effects of alcohol have been recorded for centuries (Sullivan, 1899). It wasn't until late 1960 and early 1970s that researchers started to examine closely alcohol effects on a growing fetus and subsequent symptoms associated with it. Seminal studies by Dr. Ulleland, Drs. Kenneth Lyons and David Smith helped the scientific community classify specific facial features and symptoms and identify the damage done prenatally to children due to alcohol use (Jones et al., 1973; Williams & Smith, 2015). Based on the research, the term Fetal Alcohol Syndrome was first used in 1973. The following studies slowly began to provide more detailed answers on the causes, symptoms, and treatments available for people who are affected by alcohol use. Soon enough, FASD became an umbrella term which incorporates several classifications such as FAS, pFAS, SE/NA, ND/AE, and ND-PAE. Depending on the symptoms, severity and exposure to alcohol, medical professionals can determine which classification is applicable.

The prevalence of FASD can be difficult to determine, but on average, it's estimated that 2-4% or 24-48 children per 1000 are affected by FASD (Williams & Smith, 2015, ("Fetal Alcohol Spectrum Disorders in Minnesota", 2017). In Minnesota, approximately 7, 061 babies are born with prenatal alcohol exposure, and 32,900 + children with some type of alcohol exposure are enrolled in K-12 school system ("Fetal Alcohol Spectrum Disorders in Minnesota", 2017). The estimated cost of care for each child with FASD is \$2.44 million, and MN cost equals to \$131 million each year. The expenses of raising a child with FASD are approximately 30 times the cost of preventing the FASD ("Fetal Alcohol Spectrum Disorders in Minnesota", 2017). Based on the information, it's important for Early Childhood educators to be aware of impacts of alcohol on children's health and wellbeing, and familiar enough with interventions to best help them.

Based on the type of FASD, the symptoms can range from mild to severe. Children can experience different effects of alcohol exposure, resulting in abnormal facial features, smooth ridge between the nose and upper lip and physical characteristics such as, small head size, and low body weight and problems with the heart, kidneys, or bones. FASD also causes developmental delays resulting in poor coordination, hyperactive behavior, difficulty with attention, poor memory, learning disabilities and delays, poor reasoning and judgment skills, vision or hearing problems.

FASD symptoms can be divided into two categories: primary features disability and secondary/co-existing conditions. Primary features are related to growth, face and brain development, while secondary/co-existing conditions are a result of these delays in development. Brain development is particularly susceptible to alcohol exposure, affecting central nervous system, the frontal cortex, caudate, hippocampal formation, corpus callosum, and components of the cerebellum, including the anterior cerebellar vermis. Studies have shown that alcohol reduced white matter growth in the brain leading to cognitive developmental delays, such as memory, attention and communication. Likewise, children will have difficult time developing complex language skills. They will have problems with understanding complex language and figures of speech. Many children with FASD will have some type of language impairment to qualify with severe language deficits. These delays then lead to problems with adaptive functioning skills, such as self-care and intellectual abilities. Children's social skills would be impaired and make it difficult to form relationships. There may be symptoms of ADHD, leading to problems in acquiring new skills, and school readiness.

FASD can often impact physical health, leading to autoimmune diseases (Zhang et al., 2012), and an increased vulnerability to the influenza virus (McGill et al., 2009). The researchers found that hearing loss, visual impairment and blindness were remarkably more common in children impacted by FAS than among the general population in the US. Based on the variety of physical and mental delays and disabilities that are associated with FASD, the intervention strategies should be able to address multitude of needs. The research showed that support is particularly important for families with FASD. Caregivers of children with FASD tend to have lower education, socioeconomic status and more health problems. As such, parent education interventions can help create a safer and more nurturing environment for children. Interventions which target parents tend to focus on increasing the knowledge about FASD and its effects, and what parents can do to help their children. Additionally, these programs also build positive behavioral support between children and parents. Some programs also provide resources in the community or offer mentoring programs for parents who need help. Breaking the Cycle program is flexible in that it's based on home visits, while The Coaching Families works with children of all ages. Some of the programs are long term-based interventions while others last a few weeks (Koren, 2013).

Children with FASD often have problems with attention and self-regulation, which is why it's important that they have access to programs and interventions which address these issues. In recent years, programs have been developed based on computer games which help children with attention and memory improvements. Programs such as Pay Attention, CPAT, Alert, GoFAR etc. have shown improved sustained and selective attention, spatial working memory and reading and math fluency skills (Williams & Shelleberger, 1996; Petrenko & Alto, 2017). These interventions are relatively short, and often can be completed in less than 20 hours.

This is usually completed over a period of a few weeks and children sometimes have help from trained professionals who can guide them through the sessions. There has also been evidence that these types of programs promote grey matter growth in the brain in critical regions for self-regulation (Soh et al., 2015; Coles et al., 2018).

Adaptive Functioning Interventions are important for children who struggle with adaptive functioning skills. These programs address safety awareness, social functioning and academic achievements. Some of the programs are simple games which teach children basic safety instructions and are suitable for all ages. The biggest advantage of these types of interventions is that it doesn't require professional assistance, only a parent or an adult to guide a child through the game. Some interventions like Good Buddies work on developing social skills in children through group activities. It also offers training to caregivers, so they can better support their children as they work on peer interaction, and behavior management. A few programs also target academic skills, especially math and language. MILE and LLT were designed for in school interventions and have shown positive improvements in overall reading, literacy and math levels (Petrenko & Alto, 2017; Kable et al., 2015).

Along with early childhood interventions, researchers have been studying if certain types of medications or supplements can help children with FASD. Many children who have been diagnosed with FAS usually have symptoms of ADHD and are prescribed medication to treat it. ADHD medications seem to reduce hyperactivity and impulsivity but not inattention. Currently, there are no medical treatments which can cure FASD but certain substances have been shown to improve children's cognitive skills. In particular, choline (vitamin like nutrient) if administered to 2-5 year old children may have positive impact on explicit memory. Some studies also point to

neuroleptics (tranquilizers) as a possible way to help children improve self-control, and exhibit less problematic behaviors (Wozniak et al., 2015; Petrenko & Alto, 2017).

Overall, the variety of interventions and supplements seem to offer possible strategies to families and children who have FASD. Different programs target different needs and depending on the severity of FASD, it's important to consider all options available. Providing comprehensive intervention in a timely manner can perhaps reduce symptoms and prepare children for more independent life later on. Thankfully, many of the early childhood education programs use some form of already discussed interventions, but it's important to also emphasize the benefits of one-on-one interventions, and the parent education programs.

Chapter 4: Position

FASD is one disorder which is completely preventable and yet, there is not as much research as I previously assumed. While some studies focus on investigating the neurological effects of alcohol consumption on a developing fetus, and the effects later in life, research on children in early childhood settings was rarer. This is interesting in of itself, considering how important it is to address developmental delays and disabilities early on. Perhaps the difficult part is determining if children have FASD in the first place. Unless there are specific facial abnormalities, it can be challenging to determine if a child has some form of FASD. Moreover, the various interventions which target the children seem to mostly focus on general cognitive, adaptive or behavioral delays and if there are indications that it also helps children with FASD, they are promoted in FASD literature. Based on the evidence gathered in the paper, it is obvious that very diverse approaches must be taken to help children who experience FASD symptoms.

The need for such diverse interventions and strategies stems from the evidence that support provided to families is a first step in trying to also help children. Many families with children who have FASD come from lower SES, which makes ‘typical’ interventions problematic, as many families may not be able to dedicate so much time and resources to having their children attend these programs. Parent intervention programs which offer mentoring services, and resources can prepare caregivers and families navigate the challenging field of FASD. If parents acquire wellness skills, they may be able to better connect and bond with their children. Still, it’s equally important to offer interventions in schools, as some children with FASD may not be able to qualify for a diagnosis and receive appropriate help otherwise.

It is interesting to observe the use of modern technology in interventions, such as virtual and computer games, due to their relative inexpensiveness and general applicability in variety of

settings. An interesting area of research should focus more on what types of games and interventions would help children with FASD, and how they can be integrated in classrooms. Still, future research should look into what are the most common interventions and programs administered to children with FASD and then see which programs show best results. It can also be helpful to investigate if there are any variations based on gender, age and environments. Promising research on supplements and medications may yield additional information on how to pair them with early childhood interventions, so future research should address if any supplements can alleviate FASD symptoms or enhance cognitive and adaptive skills.

Overall, it is obvious that there is a great need for more research-based interventions that address the multitude of needs children with FASD have. Based on the evidence that FASD has significant impact on academic and social skills of children, resulting in struggle to live and function independently, it's imperative that more focus is placed in identifying evidence-based interventions targeted for children with FASD. Whether that means adapting current programs, combining two or more interventions, or creating new ones, the emphasis must be placed on helping these children develop skills, so they can function in a society semi-independently. Hopefully, with the emerging neurobiological research, future studies will then progress into early childhood intervention.

References

- Armstrong, E. M. (2003). *Conceiving risk, bearing responsibility: Fetal alcohol syndrome & the diagnosis of moral disorder*. Baltimore: The Johns Hopkins University Press.
- Arnett, A., & Labovitz, S. (1995). "Effect of physical layout in performance of the Trail Making Test". *Psychological Assessment*. 7 (2): 220–221. doi:10.1037/1040-3590.7.2.220
- Åse, F., Ilona, A.R., Mirjam, K., Pekka, S., Eugene, H.H., Sarah, M.N., Marit, K. (2012). Adaptive behaviour in children and adolescents with foetal alcohol spectrum disorders: a comparison with specific learning disability and typical development. *European Child Adolescent Psychiatry* 21 (4), 221e231.
- Astley, Susan (2015). The 4 Diagnoses under the FASD Umbrella. Retrieved October 01, 2017, from <https://depts.washington.edu/fasdpn/htmls/fasd-fas.htm>
- Bernard, K., Dozier, M., Bick, J., Lewis-Morrarty, E., Lindhiem, O., Carlson, E., 2012. Enhancing attachment organization among maltreated children: results of a randomized clinical trial. *Children Development* 83, 623e636.
- Burd, L., Klug, M.G., Martsof, J.T., Kerbeshian, J., 2003. Fetal alcohol syndrome: neuropsychiatric phenomics. *Neurotoxicology Teratol.* 25 (6), 697e705.
- Coles, C. D., Kable, J. A., Taddeo, E., & Strickland, D. (2018). GoFAR: improving attention, behavior and adaptive functioning in children with fetal alcohol spectrum disorders: Brief report. *Developmental neurorehabilitation*, 1-5.
- Cook, J. (2017, May 18). Should pregnant women be allowed to drink alcohol? Retrieved February 12, 2018, www.netdoctor.co.uk/parenting/pregnancy/news/a28215/pregnant-alcohol-guidelines/

- Davis, K., Desrocher, M., & Moore, T. (2011). Fetal alcohol spectrum disorder: A review of neurodevelopmental findings and interventions. *Journal of Developmental and Physical Disabilities, 23*(2), 143-167.
- Doig, J., McLennan, J.D., Gibbard, W.B., 2008. Medication effects on symptoms of attention-deficit/hyperactivity disorder in children with fetal alcohol spectrum disorder. *Child Adolescent Psychopharmacology 18* (4), 365e371.
- Doney, R., Lucas, B. R., Watkins, R. E., Tsang, T. W., Sauer, K., Howat, P., ... & Elliott, E. J. (2016). Visual-motor integration, visual perception, and fine motor coordination in a population of children with high levels of Fetal Alcohol Spectrum Disorder. *Research in developmental disabilities, 55*, 346-357.
- Elwood, R. W. (1995). The California Verbal Learning Test: psychometric characteristics and clinical application. *Neuropsychology Review, 5*(3), 173–201.
- Fetal Alcohol Spectrum Disorders in Minnesota. (2009). Retrieved September 01, 2017, from <https://www.mofas.org/2014/07/fetal-alcohol-spectrum-disorders-in-minnesota/>
- Frankel, F., Paley, B., Marquardt, R., O'Connor, M., 2006. Stimulants, neuroleptics, and children's friendship training for children with fetal alcohol spectrum disorders. *J. Child Adolescent Psychopharmacology 16* (6), 777e789.
- Gautam, P., Nuñez, S. C., Narr, K. L., Kan, E. C., & Sowell, E. R. (2014). Effects of prenatal alcohol exposure on the development of white matter volume and change in executive function. *NeuroImage: Clinical, 5*, 19-27.
- ICD-10-CM Codes. (n.d.). Retrieved from <http://www.icd10data.com/ICD10CM/Codes/Q00-Q99/Q80-Q89/Q86-/Q86.0>

- Howard J. (2017, September 17). Is light drinking while pregnant OK?. Retrieved February 12, 2018, from <https://www.cnn.com/2017/09/11/health/drinking-alcohol-pregnant-study/index.html>
- Jones K.L., Smith D.W, Ulleland C.N., Streissguth A.P. (1973). Pattern of malformation in offspring of chronic alcoholic mothers. *Lancet*. 1 (7815): 1267–1271.
doi:10.1016/S0140-6736(73)91291-9. PMID 4126070.
- Kable, J.A., O'Connor, M.J., Olson, H.C., Paley, B., Mattson, S.N., Anderson, S.M., Riley, E.P., 2015. Neurobehavioral disorder associated with prenatal alcohol exposure (ND-PAE): proposed DSM-5 diagnosis. *Child Psychiatry Human Development* <http://dx.doi.org/10.1007/s10578-015-0566-7>.
- Kable, J.A., Taddeo, E., Strickland, D., Coles, C.D., 2015. Community translation of the math interactive learning experience program for children with FASD. *Res. Developmental Disability* 39, 1e11.
- Kerns, K.A., MacSween, J., Vander-Wekken, S., Gruppuso, V., 2010. Investigating the efficacy of an attention training programme in children with foetal alcohol spectrum disorder. *Dev. Neurorehabilitation* 13 (6), 413e422.
- Koren, G., 2013. Breaking the Cycle (BTC)-20 Years of breaking records in managing addicted mothers and their young children. *J. Popular Therapy Clinical Pharmacology* 21(3), e505ee507.
- Leenaars, L.S., Denys, K., Henneveld, D., Rasmussen, C., 2012. The impact of fetal alcohol spectrum disorders on families: evaluation of a family intervention program. *Community Mental Health J.* 48 (4), 431e435.

- LeWine, H. (2018, January 18). Drinking a little alcohol early in pregnancy may be okay. Retrieved February 12, 2018 from <https://www.health.harvard.edu/blog/study-no-connection-between-drinking-alcohol-early-in-pregnancy-and-birth-problems-201309106667>
- Mattson, S. N., Roesch, S. C., Glass, L., Dewese, B. N., Coles, C. D., Kable, J. A., ... & Jones, K. L. (2013). Further development of a neurobehavioral profile of fetal alcohol spectrum disorders. *Alcoholism: Clinical and Experimental Research*, 37(3), 517-528.
- May, P. A., Blankenship, J., Marais, A. S., Gossage, J. P., Kalberg, W. O., Joubert, B., ... & Robinson, L. K. (2013). Maternal alcohol consumption producing fetal alcohol spectrum disorders (FASD): quantity, frequency, and timing of drinking. *Drug and alcohol dependence*, 133(2), 502-512.
- McGill J., Meyerholz D. K., Edsen-Moore M., Young B., Coleman R. A., Schlueter A. J...Legge K. L. (2009). Fetal exposure to ethanol has long-term effects on the severity of influenza virus infections. *The Journal of Immunology*, 182(12), 7803–7808.
- Merriam-Webster. (n.d.). Central Nervous System. Retrieved from <http://www.merriam-webster.com/dictionary/central%20nervous%20system>
- Merriam-Webster. (n.d.). magnetic resonance imaging. Retrieved from <http://www.merriam-webster.com/dictionary/magnetic%20neural%20imaging>
- Merriam-Webster. (n.d.). socioeconomic status. Retrieved from <http://www.merriam-webster.com/dictionary/socioeconomic%20status>
- Millar, J. A., Thompson, J., Schwab, D., Hanlon-Dearman, A., Goodman, D., Koren, G., & Masotti, P. (2017). Educating students with FASD: linking policy, research and practice. *Journal of Research in Special Educational Needs*, 17(1), 3-17.

- Montgomery, S. (February, 2018). 17 Moms Share The "No-No" Food They Still Ate During Pregnancy. Retrieved February 12, 2018, from <https://www.romper.com/p/17-moms-share-the-no-no-food-they-still-ate-during-pregnancy-8189135>
- Moore E. M., & Riley E. P. (2015). What happens when children with fetal alcohol spectrum disorders become adults? *Current Developmental Disorders Reports*, 2(3), 219–227.
- Nash, K., Stevens, S., Greenbaum, R., Weiner, J., Koren, G., Rovet, J., 2015. Improving executive functioning in children with fetal alcohol spectrum disorders. *Children Neuropsychology* 21 (2), 191e209.
- Nguyen, T.T., 2015. Choline Intervention in Children with Fetal Alcohol Spectrum Disorders (Doctoral dissertation). Retrieved from ProQuest Dissertation & Theses International (3712754).
- O'Connor, M. J., Laugeson, E. A., Mogil, C., Lowe, E., Welch-Torres, K., Keil, V., & Paley, B. (2012). Translation of an Evidence-Based Social Skills Intervention for Children with Prenatal Alcohol Exposure in a Community Mental Health Setting. *Alcoholism: Clinical and Experimental Research*, 36(1), 141-152. diffusion tensor imaging
- O'Connor, M. J., Quattlebaum, J., Castañeda, M., & Dipple, K. M. (2016). Alcohol intervention for adolescents with fetal alcohol spectrum disorders: Project Step Up, a treatment development study. *Alcoholism: Clinical and Experimental Research*, 40(8), 1744-1751.
- 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. *Alcoholism: Clinical and Experimental Research*, 22(9).

Peadon, E., & Elliott, E. J. (2010). Distinguishing between attention-deficit hyperactivity and fetal alcohol spectrum disorders in children: clinical guidelines. *Neuropsychiatric Disease and Treatment*, 6, 509.

Petrenko, C. L., & Alto, M. E. (2017). Interventions in fetal alcohol spectrum disorders: An international perspective. *European journal of medical genetics*, 60(1), 79-91.

Popova, S., Lange, S., Shield, K., Mihic, A., Chudley, A. E., Mukherjee, R. A., ... & Rehm, J. (2016). Comorbidity of fetal alcohol spectrum disorder: a systematic review and meta-analysis. *The Lancet*, 387(10022), 978-987.

Proven, S., Ens, C., & Beaudin, P. G. (2014). The Language Profile of School-Aged Children with Fetal Alcohol Spectrum Disorder (FASD). *Canadian Journal of Speech-Language Pathology & Audiology*, 37(4).

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

Ruiz, M. (2014, October 22). Why I Drank While I Was Pregnant. Retrieved February 12, 2018, from <https://www.cosmopolitan.com/sex-love/news/a32292/why-i-drank-while-i-was-pregnant/>

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.

State-level estimates of alcohol use among women. (2015). Retrieved September 01, 2017, from <https://www.cdc.gov/ncbddd/fasd/data-maps.html>

- Streissguth, A. P., Bookstein, F. L., Barr, H. M., Sampson, P. D., O'malley, K., & Young, J. K. (2004). Risk factors for adverse life outcomes in fetal alcohol syndrome and fetal alcohol effects. *Journal of Developmental & Behavioral Pediatrics*, 25(4), 228-238.
- Sullivan W.C. (1899). "A note on the influence of maternal inebriety on the offspring". *Journal of Mental Science*. 45 (190): 489–503 doi:10.1192/bjp.45.190.489
- Tibbett, T., & Jeffery, M. I. (2016). Smart Justice and FASD in Alaska: From Prevention to Sentence Mitigation. In *Fetal Alcohol Spectrum Disorders in Adults: Ethical and Legal Perspectives* (pp. 169-189). Springer, Cham.
- Vernescu, R.M., 2009. Sustained attention training in children with fetal alcohol spectrum disorder (Doctoral dissertation). Retrieved from Proquest Dissertations & Theses International (NR55383).
- Warren, K. R., Hewitt, B. G., & Thomas, J. D. (2011). Fetal alcohol spectrum disorders: research challenges and opportunities. *Alcohol Research & Health*, 34(1), 4.
- Wells, A. M., Chasnoff, I. J., Schmidt, C. A., Telford, E., & Schwartz, L. D. (2012). Neurocognitive habilitation therapy for children with fetal alcohol spectrum disorders: An adaptation of the Alert Program. *American Journal of Occupational Therapy*, 66(1), 24-34.
- Williams, J. F., Smith, V. C., & Committee on Substance Abuse. (2015). Fetal alcohol spectrum disorders. *Pediatrics*, 136(5), e1395-e1406.
- Williams, M.S., & Shellenberger, S., 1996. *How Does Your Engine Run?: a Leader's Guide to the Alert Program for Self-regulation*. TherapyWorks, Inc.
- Wozniak, J. R., Fuglestad, A. J., Eckerle, J. K., Fink, B. A., Hoecker, H. L., Boys, C. J., ... & Zeisel, S. H. (2015). Choline supplementation in children with fetal alcohol spectrum

disorders: a randomized, double-blind, placebo-controlled trial, 2. *The American journal of clinical nutrition*, 102(5), 1113-1125.

Wyper, K. R., & Rasmussen, C. (2011). Language impairments in children with fetal alcohol spectrum disorder. *Journal of Population therapeutics and Clinical Pharmacology*, 18(2), 364-376.

Young, J. K., Giesbrecht, H. E., Eskin, M. N., Aliani, M., & Suh, M. (2014). Nutrition implications for fetal alcohol spectrum disorder. *Advances in nutrition*, 5(6), 675-692.

Zhang X., Lan N., Bach P., Nordstokke D., Yu W., Ellis L., ...Weinberg J. (2012). Prenatal alcohol exposure alters the course and severity of adjuvant-induced arthritis in female rats. *Brain, Behavior, and Immunity*, 26(3), 439-450