The Effects of Prenatal Alcohol Exposure on Prenatal and Neonatal Development

Reem Deif
AUC Department of Psychology

Abstract

This paper examines the effects of prenatal alcohol exposure (PAE) on prenatal and neonatal development from a bio-psychosocial perspective. PAE has shown to contribute to various developmental problems during the prenatal and neonatal period with effects on a continuum ranging from minimal effect till perinatal death. I examine factors contributing to such impacts such as maternal age, time or exposure and dose. I also look at research examining impacts on the Central Nervous System (CNS), socio-behavioral development, mother-infant attachment, sensorimotor development in addition to cognitive aspects of development throughout lifespan. Although various interventions are available, the final conclusion is that abstinence is the only preventable measure against all the previously mentioned developmental problems.

Prenatal Alcohol Exposure (PAE) contributes to a series of impacts on prenatal and neonatal developmental defects on a continuum ranging from no effect till perinatal death. Different effects of PAE lie on a wide scale of motor and intellectual disabilities which constitute the Fetal Alcohol Spectrum Disorders (FASD); this includes Fetal Alcohol Syndrome (FAS), Partial FAS (PFAS), Alcohol-Related

1 Faculty advisor: Mona Amer
Deif: Effects of Prenatal Alcohol Exposure

Neurodevelopmental Disorders (ARND), and Alcohol-Related Birth Defects (ARBD). Research has investigated effects of different levels of PAE on cognitive, physical and behavioral aspects of development, taking possible risk factors into account such as maternal age, timing and level of exposure. The purpose of this paper is to assess short-term and long-term effects of PAE through reviewing previous studies with focus on FAS as one of the most common disorders under the FASD category. It may also encourage further research on effective interventions for individuals with PAE and related disorders through the examination of related psychosocial issues.

The mechanism of action of alcohol, or ethanol, is illustrated in its chemical composition that allows rapid diffusion of its molecules across the placenta. Ethanol enters the fetal circulation without being metabolized in the placenta and then it enters the fetal liver to be metabolized at a rate that is only 5-10% of the adult functioning rate (Pikkarainen, 1971). Consequently, ethanol accumulates in the amniotic fluid, a protective medium for the fetus. It may also return to the fetal circulation and repeat the same cycle (Gilbert, 2006).

PAE refers to a condition resulting from alcohol consumption by a pregnant human female which might disrupt normal fetal development. Exposure to alcohol during each of the three trimesters of pregnancy affects the fetal development differently. Dosage of alcohol is also considered to determine the severity of PAE-related symptoms; and according to the National Institutes of Health (2010), binge drinking, which is estimated to be
four or more drinks per at one occasion, represents the highest risk.

FAS, which was first identified by Jones and Smith (1973), refers to a range of mental and physical defects attributed to high levels of PAE. Symptoms are categorized into three groups; growth retardation, central nervous system abnormalities and facial defects (i.e.: flat nasal bridge and thin upper lip) (IOM, 1996). Such deficits are most obvious during adolescence (Sampson et al., 1994). Prevalence of alcohol-related disabilities is estimated to be 9.1 per 1000 (O’Learey, 2002) and FAS is still one of the leading teratogenic causes of birth defects worldwide (Welch-Carre, 2005). However, some of the birth defects assumed to be etiologically related to PAE can be attributed to other risk factors which might challenge accurate diagnosis (Aase, 1994). Alcohol consumption also raises the risk of miscarriage by 30% (Hannigan & Armant, 2000).

**Impacts on the Central Nervous System (CNS)**

Some neurobehavioral deficits associated with PAE (i.e.: poor memory, attention and motor functions) are attributed to hippocampal abnormalities. Riley and colleagues (1986) reached that conclusion based on the similarities between the impacts of hippocampal lesions and PAE. Effects of higher levels of PAE are evident in brain imagining that suggests reduced brain volumes in individuals with FAS (See Figure 2) especially the basal ganglia, hippocampus and cerebellum, in addition to less gray matter and white matter (Lebel, Roussotee, & Sowell, 2011). Furthermore, corpus callosum, that is
responsible for the coordination between the right and left brain hemispheres, can be altered by PAE. It develops during the second trimester (Deng & Elberger, 2001) which makes this stage of significantly high vulnerability. Alcohol may also cause delays in the development of the cortical barrels (Margret et al., 2006). This may lower the rates of neuronal generation and cell migration which may explain the decreased CA3 pyramidal neurons count in rats prenatally exposed to alcohol (Miller, 1995).

**Figure 1:** Different prenatal developmental stages of the CNS and other organs. The blue parts of the bar represent the most critical stages (Note that CNS is the longest). Minor defects could occur during the periods covered by the yellow parts of the bars (Moore & Persaud, 1993).
Socio-behavioral Impacts

Damage to the CNS may result in secondary problems including externalizing behaviors (i.e.: impulsiveness), and internalizing behaviors (i.e.: withdrawal). Decreased response inhibition could be linked to abnormal 5-HT (serotonin) functions as suggested by Fryer and colleagues (2007). This neurotransmitter abnormality may be evident in cases of high levels of anxiety and aggression (Jacobson et al. 2006). Examples of externalizing behaviors in individuals with PAE as suggested by research include nicotine, alcohol and drug dependence among individuals aged 18-45 years (Yates...
et al., 1998), and DSM-IV alcohol use disorders among individuals aged 13-21 years (Alati et al., 2006). This is explained in terms of their inability to contemplate consequences of actions and interpret social cues. They also display uneasiness in interpersonal relationships, and therefore they are more likely to suffer from poor adjustment (Streissguth et al., 2004).

A recent study demonstrates a relationship between behavioral problems at the age of 22 as mentioned in self-reports and that alcohol use across pregnancy is more significant than during the first trimester or no use at all. PAE during the third trimester had the highest impact on both externalizing and internalizing behaviors (Day et al., 2013).

Mother-Infant Attachment

Generally, individuals with FAS show relatively low socio-emotional development when IQ is taken into account (Carmichael Olson et al., 1998); this includes higher irritability in infancy as suggested by Coles and colleagues (1991) and poor attachment to the mother due to temperamental style (Kelly et al., 2000). Early neurocognitive deficits during infancy are significant due to their role in the infant-mother interaction (O’Conner & Paley, 2009) which is essential for learning and proper psychological attachment.

Dosage of alcohol could determine the level of negative emotions as concluded by O’Conner, Sigman & Kasari (1992), and mothers of the more affected infants were less likely to act responsively. This resulted in the
development of insecure attachment between the infants and the mothers. A follow-up study identified depressive symptoms among children with higher PAE levels (O’Conner & Kasari, 2000). Negative emotionality associated with PAE has also been linked with internalizing disorders during adolescence such as depression and anxiety (Murray et al., 2006).

**Cognitive Impacts**

Research suggests that PAE has remarkable negative impacts on IQ, executive functioning, verbal, arithmetic skills and learning (Chiodo, Janisse, Delaney-Black, Sokol, & Hannigan, 2009; Mattson, Calarco, & Lang, 2006; Streissguth, Barr, & Sampson, 1990; Vaurio, Riley, & Mattson, 2008) in addition to the acquired risk of hyperactivity, depression and poor communication skills (Crocker, Vaurio, Riley, & Mattson, 2009). Language deficits, both receptive and expressive, and speech delays are common among children with FAS (Abel, 1990). For example, Autti-Rämö and colleagues (1992) suggest that articulation, word comprehension and naming were impacted by prenatal alcohol exposure. Such factors could put children with heavy PAE at risk of poor academic performance (Streissguth, Barr, & Sampson, 1990).

Results of the Neonatal Behavioral Assessment Scale indicated poor performance and decreased arousal among infants prenatally exposed to alcohol in response to various animate and inanimate stimuli (Smith et al., 1986). However, alcohol use during mid-pregnancy has been correlated with lowered habituation (Streissguth,
Barr & Martin, 1983); this means that infants prenatally exposed to alcohol are less able to inhibit responses to repetitive stimulus. Interestingly, individuals with PAE usually show higher reactivity to stressors in adulthood (Glavas et al., 2007).

Both sustained and focused attentions have also proven to be affected by various levels of PAE; for instance, FAS has been correlated with difficulty shifting attention, or poor cognitive flexibility (Kodituwakku et al., 1995). Exposure during the second trimester has the most significant effects on attention (Day et al., 2013). Nevertheless, children with FAS perform better on tasks that require focused attention than children with attention deficit hyperactivity disorder (ADHD) (Coles et al., 1997).

Performance of children with FAS on psychometric tests such as the Tower of California (Mattson et al., 1999), Raven’s Standard Progressive Matrices and Progressive Planning Test (Kodituwakku et al., 1995) reflects poor planning abilities. Performance on the Wisconsin Card Sorting Test (WCST) test for non-verbal problem solving skills was worse among children with FAS who showed “decreased accuracy” (Carmichael Olson, Feldman, Streissguth, & Gonzalez, 1992).

A study examining the effects of FAS on memory recall indicates that children with FAS could recall 17% fewer objects after 24 hours than children in the control group; deficits in spacial memory were the most significant (Uecker and Nadel, 1996). In another study, children with FAS performed poorly on both free and recognition
recalling with high rates of false positives (Mattson et al., 1996). Declarative memory is also influenced by PAE; in a study conducted by Carmichael Olson et al. (1998), children in the experimental group performed as good as children in the control groups; however, they failed to “recall” the sequence of the tasks they performed.

*Impacts on the Sensorimotor System*

A study conducted on 16 children and adolescents with heavy PAE and 16 controls suggests poor odor identification in the experimental group. This can be explained in terms of smaller olfactory bulbs identified in an animal model during a stage equivalent to the third trimester in fetal development (Maier et al., 1999). Poor binocular visual acuity was also linked to PAE in infants, especially those born to mothers aged 30+ (Carter et al., 2005).

PAE has also proven to have impacts on childhood body balance. Generally, defects in balance, a complex process based on sensory inputs and motor outputs, has been etiologically linked to various negative psychosocial outcomes such as anxiety and depressive symptoms as the affected individual fails to maintain control over his/her body (Humphriss, Hall, & Macleod, 2010). Children prenatally exposed to alcohol may also have delayed fine and gross motor skills, and the former are usually more deteriorated (Kalbery et al., 2006). Severe cases may display kinetic tremors when performing a task (i.e.: writing) and some movement disorders including axial ataxia (Marcus, 1987). This
overall impacted functioning of the somatosensory system in individuals with PAE puts them in need of greater and more intense sensorimotor input (Burd et al., 2003) as provided by different rehabilitation programs.

**Interventions**

Fortunately, it is argued that environmental enrichment can improve some of the PAE-related cognitive and neurobehavioral deficits. Major challenges to different types of interventions include ongoing parental use of alcohol and/or other drugs and psychopathology in addition to child neglect and/or abuse. In this sense, an effective intervention should not only address the child/infant’s developmental deficits, but also social and environmental problems that act as risk factors. Interventions should also be comprehensive so that the child, parents and different aspects of the environment are all included. For example, physical therapy and balance exercises are commonly used as an intervention for children with FAS to overcome motor and cognitive deficits besides language and speech therapy (Harris et al., 1993). The child’s cognitive functioning can also be addressed through literacy training, safety awareness, and occupational therapies. Behavioral strategies may focus on the mother-infant interactions through improving the mother’s responsiveness, and thus improving the attachment style. Parent-assisted social skills training help in enhancing the child’s adjustment and related social issues in the presence of a parent figure. Studies examining the effectiveness of such interventions report better outcomes in experimental
groups than in control groups (Paley & O’Connor, 2011).

**Conclusion**

The body of knowledge offered by other researches is significant and therefore this review only covers some of the physiological, cognitive and behavioral impacts of PAE. Recent studies have also been able to identify several risk factors on different levels; individual, maternal and environmental. Nevertheless, no safe dose of alcohol has been identified, and consequently, complete abstinence remains the only means of prevention (IOM, 1996).

Longitudinal studies are still few and most of the studies examined the short-term effects of PAE, (i.e.: till adolescent). More thorough examination of each of these aspects may result in a better understanding of PAE and related disorders and probably more effective interventions for affected individuals.
Deif: *Effects of Prenatal Alcohol Exposure*

**References**


http://dx.doi.org/10.1111/j.1530-0277.1998.tb05909.x


http://dx.doi.org/10.1016/j.jpeds.2005.04.063

http://dx.doi.org/10.1111/j.1530-0277.2008.00878.x

http://dx.doi.org/10.1016/0892-0362(91)90085-B


http://pediatrics.aappublications.org/content/119/3/e733


http://neoreviews.aappublications.org/content/7/6/e292

http://dx.doi.org/10.1053/siny.2000.0027

http://ptjournal.apta.org/content/73/9/608.long


[http://dx.doi.org/10.1017%2FS1355617708080144](http://dx.doi.org/10.1017%2FS1355617708080144)

[http://dx.doi.org/10.1016/j.adnc.2005.04.007](http://dx.doi.org/10.1016/j.adnc.2005.04.007)