# Potential effects of alcohol consumption on prenatal development

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Since 1973, when fetal alcohol syndrome (FAS) was first identified, a significant volume of research has drawn conclusions that prenatal alcohol exposure (PAE) has an effect on the biological basis of development (Templeton et. al., 2006). Alcohol is a major teratogen which passes through the placenta to the foetus during the prenatal period. Alcohol exposure interferes with fetal nerve cell development and connections, causing cell death of neurons (Gohlke et. al., 2008) and altering the brain??™s structure (Chen et.

al., 2003; O??™Connor, 2002). Earlier research focused on the relationship between heavy alcohol consumption and FAS, while more recent research has also considered low to moderate intake. FAS has a set diagnostic criteria based on three areas but research has broadened out to cover the wider range of harms created by maternal alcohol use, rather than solely within the FAS criteria, and fetal alcohol syndrome disorders (FASD) are more commonly referred to. FASD is preventable but has devastating consequences for cognitive, behavioural and psychosocial functioning (McGee et al, 2008) which will be discussed below.

The key areas of research considered here relate to growth, cognitive status, psychiatric health, and socio-emotional functioning. Brain pathology indicates that the area affected by PAE is the cerebellum, which has motor, cognitive and emotional functions. The vermis, between the cerebellum??™s hemispheres, was found to be underdeveloped in 100% of the PAE cohort in a study of MRI findings (Autti-Ramo et. al., 2002). The authors identified that risk factors other than alcohol exposure could have caused or contributed to the same damage. However, they considered the findings relevant as ??? in

a study by Paradiso and coworkers (1997) of normal volunteers, vermis abnormality was found to be related to IQ, verbal memory, and motor dexterity.??? (Autti-Ramo et.

al., 2002, p. 103). Other brain areas are cited in PAE research. McGee et.

al (2008) refer to Schonfield et. al. (2005) who attribute lower moral maturity and higher rates of delinquency to central nervous system dysfunction (the CNS is contained within the cranial cavity).

Abnormal neuro-anatomical networks are identified by Greenbaum et. al. (2009) as adversely affecting social cognition and emotional processing. Growth was the focus of studies of children aged 6 (Cornelius et. al., 2002) and 14 (Day et. al., 2002), where the effects of PAE had been identified at birth.

Both studies, drawing on their own findings and other research, identified that length, weight and head circumference were reduced at birth. Height remained lower than average at ages 6 and 14 in both studies, However Cornelius et. al (2002) concluded that only height was still affected at age 6, while Day et. al (2002) found that 14 year olds were consistently smaller in all three areas. They also offered different perspectives on when the damage occurred, with Cornelius et. al. (2002) reporting the 2nd and 3rd trimesters, and Day et.

al. (2002) indicating the 1st trimester as most significant. Both studies found that the effect on height was apparent even where alcohol exposure had

been low, that is less than one drink daily. Day et. al. (2002) make reference to Samson et.

al. (1994) who did not find an association of PAE with size at 14 years. Day et. al. (2002) indicate they studied a lower income group and additional stressors (medical, economic, psychiatric, social, legal) may have made growth deficits more significant. In relation to cognitive status, Kahlberg et.

al (2006) summarised that ??? studies have shown that the average intelligence scores of children with prenatal alcohol exposure generally fall 2 standard deviations below the mean??? (Kahlberg et. al., 2006, p. 2038).

This bears out Willford et. al. (2004) who found ???…. there is a consistent pattern of long-term impairment in learning and memory function as a result of light to moderate alcohol exposure??? (Willford et.

al., 2004, p. 503). Willford et. al. (2004) identified that at aged 14 the deficits were connected to verbal and non-verbal learning, and memory. Deficits in verbal learning were linked to PAE in the 1st and 2nd trimesters, with reduced memory performance resulting from PAE in the 2nd and 3rd trimesters. This study did not identify a link between alcohol exposure and visual-spatial learning and memory, while the authors report that other studies have made this association and it may depend on the complexity and type of tasks used.

In 2006 Willford et. al. studied the effect of moderate PAE on the cognitive ability of 10 year olds and found the largest deficits were from 2nd trimester exposure, though in this study they identified a greater significance amongst

African-American children rather than Caucasians. The authors concluded that the race differences appeared to be genetic as they had considered social/ environmental effects.

However, the research used the Stanford-Binet Intelligence Test, fourth edition, and this has been identified as having a racial/ ethnic bias (Vincent, 1991). Cognitive status in terms of social cognition and emotional processing has also been researched and there is evidence of deficits which may be related to abnormal neuro-anatomical networks (Greenbaum et. al., 2009) and neuro-behavioural deficits (Kahlberg et.

al., 2006). The issues are identified as ??? externalizing behavior problems and deficient social skills??™ (Greenbaum et. al., 2009, p. 1664) and fine motor delays (Kahlberg et.

al., 2006). Psychiatric health has also been identified as affected by PAE. O??™Connor et. al. (2001) used a depression scale to study 6 year olds and found that PAE children, particularly girls, were at greater risk of depression in early childhood. They considered other substances ingested during pregnancy and only alcohol had this impact. In a later study O??™Connor et.

al. (2002) reported that 87% of a sample of PAE 5 to 13 year olds had psychiatric diagnoses, with a high incidence of mood disorders. 35% of the sample children had early onset Bipolar Disorder. The authors stated that this may be attributed to structural changes in the brain which have been identified in PAE children, and underdevelopment of the same structures in adult brains has been associated with primary and secondary mood

disorders. Flynn and Chermack (2008) also indicate a high interrelation of PAE with lifetime psychosocial and psychiatric risk factors.

O??™Connor et. al. (2001) recommend that that PAE should be considered when looking at risks for mental disorders. Socio-emotional functioning is the final area considered from the research.

McGee et. al. (2008) studied the ??? executive functioning??™ of adolescents ??“ the basis of complex cognitive, emotional and social skills.

They found that PAE adolescents were more likely to be deficient in the skills related to social problem solving (identifying problem, developing potential solutions, implementing the most effective solution, inhibiting less effective responses, and monitoring effect on other people. They had a low frustration tolerance and were less able to regulate their behaviour and control their emotions. Further to this Crocker et.

al. (2009) concluded that adaptive ability does not improve with age in children with FASD and socialisation is particularly affected. All studies depended on self-reporting of alcohol use, though attempts were made to increase validity of information. Most did not include partner alcohol-use patterns (important given possible links between partner violence and risk of PAE). The follow-up interview rates were not 100% and possibly excluded women with the most systematic difficulties.

Women who did not seek prenatal care were not included. Prenatal alcohol exposure is linked to development deficits over many areas. The effects depend on the stage at which PAE occurs and long term effects vary.

However interventions are required from the early stages of pregnancy as this may be when the most significant harm is caused (Day et.

al., 2002). There is evidence that alcohol exposure needs to be only light for negative outcomes and continued efforts and education are required to prevent drinking during pregnancy (O??™Connor, 2001; Willford, 2006). The highest risks are amongst women likely to have repeat pregnancies and increased substance use (Cornelius et. al., 2002) and this knowledge could facilitate the identification and targeting of risk factors. Families where children have FASD issues require more support, have higher rates of mortality, and are more likely to be involved in child custody issues (Templeton et al, 2006). REFERENCESAutti-Ramo, I.

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