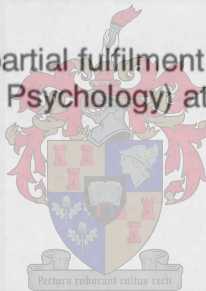


THE NEUROPSYCHOLOGICAL EFFECTS OF PRENATAL EXPOSURE TO ALCOHOL

LEILANIE CASHANDRA PHILLIPS

An assignment presented in partial fulfilment of the requirements for the
degree of Masters in Arts (Clinical Psychology) at the University of Stellenbosch.



Supervisor: Dr. J. Wait
December 2004



Declaration

I, the undersigned, declare that the work contained in this assignment is my own original work and that I have not previously in its entirety or in part submitted it any university for a degree.

A large white rectangular box redacting the signature of the undersigned.

Signature

A large white rectangular box redacting the date of the declaration.

Date

SUMMARY

The objective of this thesis is to review and synthesize the scientific literature on cognitive and neuropsychological deficits associated with children who were exposed to alcohol prenatally and to highlight possible areas of future attention.

High incidences of Fetal Alcohol Syndrome has been reported especially in patients from low socio-economic areas. The highest reported incidence is found in the Western Cape province in South Africa. The devastating part of FAS is that its affects are entirely preventable. Alcohol is a physical and a behavioural teratogen. Prenatal alcohol exposure causes structural damage to the central nervous system and the brain that is vulnerable throughout the pregnancy. A dose-response association exist as exposure to heavier amounts of alcohol can cause more harm. The timing and pattern of alcohol consumption also plays a role. To date though, no "safe" level of alcohol consumption during pregnancy can be advocated.

Various neuropsychological decrements are found in individuals with fetal alcohol syndrome or alcohol related neuro-developmental deficits as evaluated on standardized tests. Mental retardation is commonly found and even individuals with normal IQ's still display other learning disabilities. IQ's remain stable over the life span. Along with impaired intellectual functioning they also struggle with mathematical tasks especially as their complexity increases.

Speech and language development is also delayed in individuals with FAS. There is little variation in the path and display poor language comprehension. Attentional deficits are also noted and especially impact on academic functioning. Clinically, children often present with ADHD but in-depth studies have revealed that neurobiologically there is some differences as children with FAS struggle more with encoding and shifting of attention as opposed to other patients with ADHD.

Difficulties with visual-spatial functioning has also been found. Verbal learning and memory are also impaired in individuals with FAS. Their poor verbal learning are influenced by their shallow level of encoding. Problems with fine motor skills are also noted.

It also appear that all executive functions are impaired. They demonstrate poor planning skills, initiation, cognitive shifting, slow information processing, their thinking is concrete and they have poor self-regulatory skills. Behavioural problems include impulsivity, hyperactivity, aggressiveness, poor social skills and impaired judgement.

Early intervention is thus essential to lessen the impact of neuro-psychological deficits on functional adaptation. A sensitive battery of neuro-psychological tests are also required to identify all the impairments in affected individuals and to plan more focussed intervention strategies.

OPSOMMING

In hierdie tesis word 'n oorsig aangebied van literatuur wat betrekking het op die disfunksie van kinders wie se moeders tydens swangerskap alkohol misbruik het. Leemtes asook moontlike areas van toekomstige navorsing, is bespreek.

'n Hoë voorkoms van fetale alkohol sindroom (FAS) word gerapporteer, pasiënte uit die lae SES gebiede. Die hoogste voorkoms word gerapporteer in die Wes-Kaapse provinsie in Suid Afrika. Wat die probleem meer tragies maak, is die feit dat dit heeltemal voorkombaar is. Alkohol is 'n teratogeen wat fisieke, neurologiese en gedragsimplikasies het. Blootstelling aan alkohol voor geboorte veroorsaak strukturele veranderinge in die sentrale senuweestelsel en die brein. Blootstelling tot hoër volumes van alkohol veroorsaak noodwendig meer skade. Die spesifieke stadium van alkohol-inname tydens die swangerskap, en die moeder se drinkpatroon, speel 'n rol in die neurosielkundige uitkomst. Tot op hede kon geen veilige alkoholvlak tydens swangerskap vasgestel word nie.

Verskeie neurosielkundige uitvalle is gevind in kinders met FAS en ook kinders met alkohol-verwante neurologies ontwikkelings probleme, volgens neurosielkundige toetsing. Verstandelike gestremdheid kom algemeen voor in kinders met FAS. Kinders met FAS wat oor normale intellektuele vermoëns beskik ervaar leerprobleme. Die intellektuele inkortings bly stabiel oor die lewenspan. Kinders met FAS ondervind erge probleme met wiskunde, veral wanneer die werk moeiliker raak.

Die spraak-en taalontwikkeling wat kinders met FAS ervaar sluit in beperkte taalbegrip en intonasie. Hulle kort aandagspan affekteer veral hulle akademiese funksionering. Die aandagsteuring van kinders met FAS en kinders met aandagstekort-hiperaktiwiteit versteuring verskil neuro-biologies. Verdere verskille bestaan ook aangesien kinders met FAS spesifiek sukkel met swak enkoderingsvermoë en om kognitiewe aanpassings te maak.

Visueel-ruimtelike vermoë van kinders met FAS is ook benadeel. Hulle sukkel ook met verbale leer en hulle geheue is ook ingekort. Die inkortings dui op 'n oppervlakkige enkoderingsvermoë. Probleme met fyn-motoriese vaardighede is ook gevind, volgens toetseing.

Toetse wat gemik is om uitvoerende funksies te evalueer, het verskeie uitvalle aan die lig gebring. Probleme in abstrakte redenering, beplanning, impulsiwiteit, self-regulering, en die inisiëring en prosessering van informasie. Gedragsprobleme soos swak sosialiseringvaardighede, aggressiwiteit, swak oordeel en hiperaktiwiteit.

Die wye neurosielkundige uitvalle wat voorkom in kinders met FAS noodsaak vroeë intervensie om die langtermyn-impak daarvan te verminder. Hiervoor word 'n sensitiewe battery neurosielkundige toetse benodig wat al die kognitiewe uitvalle kan identifiseer.

ACKNOWLEDGEMENTS

Grateful acknowledgement is made to the following:

- My Heavenly Father for all His blessings.
- My parents, who sacrificed so much to help me realize my dream.
- Dr. Wait, for all his patience, understanding, and skillful editing. Thank you for never giving up on me and for going the extra mile.
- My siblings, family and friends who always encouraged and supported me.
- Ashley, who constantly inspired me and who played an instrumental part in the final product.

“It is as natural to die as to be born; and
to a little infant, perhaps, the one is as
painful as the other”

Sir Francis Bacon, 1561-1626, *Essays*, ‘Of Death’

CONTENTS

Declaration	i
Summary	ii
Opsomming	iv
Acknowledgement	vi
Quote	vii
1. INTRODUCTION	1
2. RATIONALE	1
3. THE INCIDENCE OF FAS	3
4. THE DIAGNOSIS OF FAS	4
5. ALCOHOL TERATOGENESIS	7
6. "SAFE" LEVEL OF DRINKING	8
7. STRUCTURAL DAMAGE TO THE CENTRAL NERVOUS SYSTEM AND BRAIN	10
8. NEUROPSYCHOLOGICAL DEFICTS IN INDIVIDUALS OF FAS/ARND	12
8.1. Intellectual functioning	13
8.2. Mathematical Skills	16
8.3. Attention and Concentration	17
8.4. Motor Abilities	21
8.5. Visual–Spatial abilities	22
8.6. Speech and Language abilities	23

8.7. Verbal learning and Memory	25
8.8. Executive Functioning	27
8.8.1. Initiation	28
8.8.2. Information Processing	29
8.8.3. Self-regulation, Self- monitoring and Impulsivity	29
8.8.4. Abstract Reasoning	30
8.8.5. Planning and Cognitive shifting	30
8.9. Behavioural Problems	31
8.9.1. Social Skills	32
8.9.2. Judgement	33
8.9.3. Aggression	33
8.9.4. Impulsivity and Hyperactivity	34
9. DISCUSSION	34
10. LIMITATIONS	39
11. FUTURE RESEARCH	40
12. REFERENCES	43

LIST OF TABLES

Table 1: Tests available to delineate Neuro-Developmental Dysfunctions in Children	12
Table 2: IQ's of patients with FAS	13

1. INTRODUCTION

Maternal drinking during pregnancy can lead to many adverse effects on the developing fetus. Fetal alcohol syndrome is described as the leading cause of mental handicap in the Western world (Abel, 1984, 1998a, 1998b; Centre for Disease Control and Prevention, 2002, 2003a, 2003b). It is also entirely preventable. Prenatal alcohol exposure cause structural damage to the central nervous system and brain. The impact on cognitive functioning and behaviour are devastating as it persist over time and negatively affect the quality of life of exposed individuals.

In 1973, a cluster of birth defects resulting from prenatal alcohol exposure was formally recognised as a clinical entity called fetal alcohol syndrome (FAS) (Clarren & Smith, 1987; Jones & Smith, 1973). In 1978, Hayden and Nelson described the first three cases of FAS in South Africa.

The diagnosis of FAS remains much the same as originally proposed by Jones and Smith (1973). The current diagnostic criteria, however, also makes provision for those individuals that exhibit some but not all the criteria of FAS (will be discussed later).

2. RATIONALE

Despite the increased awareness and publicity on fetal alcohol syndrome and the detrimental effects of alcohol on the fetus, these effects often go undetected in

the mental health field, and is cited as one of the most under treated of the life long developmental disabilities in psychiatry and related mental health fields (Lockhart, 2001).

Changes in the brain structure could negatively affect behavioural effects (Mattson, Schoenfeld & Riley, 2001). However, most studies have only narrowly looked at intelligence testing and neglecting the widespread neuro-psychological deficits present in affected children. Neuro-psychological tests broaden our insight into the specific cognitive deficits associated with FAS and alcohol related neuro-developmental deficits (ARND). It can potentially help to identify affected children sooner (especially those without the physical malformations). Knowledge about what areas are specifically affected or whether all cognitive functions are impaired is important as it can prompt more focussed and efficient intervention strategies that can improve the prognosis of affected individuals.

The area of FAS is wide and complex but in this review will specifically emphasise the neuro-psychological impact of prenatal alcohol exposure. Firstly, the epidemiology, diagnosis, and factors that influence the outcome of children exposed to alcohol before birth will be reviewed. A brief discussion of alterations in brain structures resulting from prenatal alcohol exposure will be presented. Then, the various areas of cognitive functioning and behaviour will be presented. They include intellectual functioning, speech and language, attention and

concentration, visual-spatial functioning, verbal learning and memory, motor abilities, mathematical skills, executive functions and behaviour.

3. THE INCIDENCE OF FAS

The exact prevalence of FAS or prenatal alcohol exposure is unknown. It is, however, considered to be underestimated because of the difficulty in making the diagnosis, and the reluctance of health care professionals to label mothers and their children (Gardner, 2000; Stoler & Holmes, 1999). Another reason could be that severely affected children probably do not attend school, where many researchers obtain their samples and conduct their studies.

Abel (1998a) estimated the overall incidence of FAS at 0.97/1000 (0.097%) live births and 43/1000 among babies of heavy drinkers. More recently, based on three population studies, Sampson, Streissguth and Bookenstein (1997) estimated the incidence of FAS to be between 2.8/1000 and 4.8/1000 live births and the incidence of a combination of FAS and ARND to be at least 9.1/1000 live births. All races are susceptible but a higher prevalence was detected in poor areas (Canadian Paediatric Society, 2002; Viljoen, 1999b).

An increase of 56% between 1997 and 1999 of FAS was reported in South Africa (Viljoen, 1999b). The incidence of FAS in the Western Cape province is the highest reported anywhere in the world (May, Brooke, Gossage, & Croxford, 2000; Viljoen, 1999b). The highest prevalence of FAS worldwide has been

reported among first grade children in the wine-growing regions of the Western Cape province of South Africa, but in other parts of the country the similar numbers were obtained (Centres for disease control and prevention, 2003b). Viljoen (1999a) conducted a prospective survey about alcohol intake. Pregnant women in rural and urban areas in the Western Cape province of South Africa were interviewed. The sample comprised of 636 pregnant women. Forty two percent of the women admitted to alcohol use during their pregnancy; 23.7% admitted to moderate, heavy and binge drinking. The percentage of women consuming even limited amounts of alcohol during pregnancy in the Western Cape was substantially higher than the percentage found in other surveys (42.8% v 25% in USA). Of the 80 000 babies born in the Western Cape province annually, up to 1600–3200 have FAS (Baleta, 1998). These statistics were obtained from poor and lower socio-economic populations in the Western Cape. In Soweto an incidence of 2.2% was reported, 1.2% in Lenasia South, 3.7% in Westbury (Viljoen, 1999a). Fetal alcohol syndrome will cost the Western Cape province roughly R40 billion annually (Viljoen, 1999b). Therefore, FAS must be regarded as a serious and preventable cause of morbidity (Hayden & Nelson, 1987) and an urgent health problem.

4. THE DIAGNOSIS OF FAS

Fetal alcohol syndrome refers to a specific constellation of findings; however, over the years it is imprecisely and loosely used. As the awareness of the problem grew, more subtle effects and milder cases have been described leading

to the terms of fetal alcohol effects (FAE). The acronym FAE have been commonly used to signify those offspring with some but not all of the features of FAS (Aase, Jones & Clarren, 1995). There are several controversies and debates around the use of these terms but an exploration around these issues is beyond the scope of this thesis.

It is believed that FAS represents only one end of a continuum of effects that can be produced by alcohol exposure in utero. Initially children with FAS were thought to be born only to mothers who are chronic alcoholics and drank heavily during pregnancy (Van der Leeden, Van Dongen, Kleinhout & Phaff, 2001). These children typically manifest a pattern of physical anomalies, growth deficiencies and central nervous system (CNS) dysfunctions that are unquestionably of prenatal origin and are diagnosable at birth. More recently, alcohol exposure prenatally has been associated with a variety of other neuro-developmental problems, and the terms alcohol-related neuro-developmental disorder and alcohol-related birth defects (ARBD) have been proposed to identify affected infants (Streissguth, et al., 1994). In an effort to provide more clarity and coherence to research findings regarding diagnostic issues, the Institute of Medicine (IOM) of the National Academy of Sciences in Washington decided on five categories of alcohol-related disabilities that are summarized as follows (Jacobson & Jacobson, 2002).

1. FAS, with confirmed maternal alcohol exposure: evidence of a characteristic pattern of facial abnormalities, such as a small palpebal

fissure, thin upper lip, flattenediltrum and midface; evidence of growth retardation; and neuro-developmental disabilities such as microcephaly, structural brain anomalies, or neurologic hard signs;

2. FAS, without confirmed maternal alcohol exposure: the use of the same criteria as in (1) without the confirmation of maternal alcohol use;
3. Partial FAS, with confirmed maternal alcohol exposure; some components of the characteristic facial anomalies, evidence of growth retardation, central nervous system (CNS), neuro-developmental abnormalities, a complex pattern of behaviour or cognitive abnormality that is inconsistent with developmental level and cannot be explained by familial background or environment;
4. Alcohol-related birth defects (ARBD): the presence of congenital anomalies, malformations or dysplasias arising as a consequence of confirmed or unconfirmed prenatal alcohol exposure;
5. Alcohol-related neuro-developmental disorder (ARND): evidence of CNS neuro-developmental abnormalities such as decreased cranial size, structural brain anomalies, presence of neurological hard or soft signs, a complex pattern of behaviour or cognitive abnormalities inconsistent with developmental level, which cannot be explained by familial background or environment.

There is no single symptom specific to FAS. The diagnosis is likely only if various characteristics are present and combined with a history of maternal alcohol abuse before and during pregnancy (Lovell, 1995).

5. ALCOHOL TERATOGENESIS

Alcohol is a behavioural and physical teratogen. This was demonstrated by animal and human research (Osofsky & Fitzgerald, 2000). The teratogenic effects of alcohol have been documented by a large body of experimental animal research that parallel findings from clinical and epidemiological human studies (Streissguth & Kanter, 2000). According to Streissguth, Bookenstein and Barr (1994) any agent that disrupts the normal development of the fetus is a teratogen. It also causes several birth defects in the offspring ranging from growth deficiencies functional deficits and most severe, death. Alcohol reportedly causes more damage to the developing fetus than marijuana, heroine or cocaine (McCormick, 2003).

Alcohol is described as the largest etiological factor for FAS. The pathogenesis is multifactorial. Prenatal maternal smoking, malnutrition during pregnancy, narcotics or benzodiazepines use can adverse affects on the central nervous system (CNS) and the brain (Autti-Ramo, Autti, Korkman, Kettunen, Salonen, Valanne, 2002). Unfavourable living conditions can also negatively affect the postnatal development of such infants. Therefore, it is difficult to partition out the impact of prenatal alcohol exposure from the impact of postnatal environmental

factors. Evaluations done closer in time to the insult will thus provide a more accurate picture of the teratogenic effect and the interactions of the various factors mentioned above (Stoler & Holmes, 1999).

The effects of alcohol also occur across a continuum of prenatal exposure according to a dose-response relationship (Streissguth, Barr, Sampson & Bookenstein, 1994). Streissguth, Randals and Smith (1991) reported that heavy drinking (most sources mentions more than five drinks per day) and the timing and pattern of alcohol use will cause more debilitating developmental problems of a structural and physical nature. At the most serious end of the continuum fetal alcohol syndrome will occur. Lower levels of alcohol exposure often emerge as problems in behaviour, adaptive functioning and learning problems, to mention but a few (Lovell, 1995).

6. "SAFE" LEVEL OF DRINKING

Controversy surrounds the question of a "safe" drinking level during pregnancy. It is unclear why heavy drinking in some women causes no harm to their babies while lesser alcohol intake by other mothers negatively affected their offspring. Individual differences like their sensitivity levels, nutritional and physical status of mothers have been proposed.

Some researchers mention one drink per week while others suggests more flexible amounts. The Canadian Paediatric Society (2002) have found that 15ml

of alcohol or less daily will cause no harm but higher levels of alcohol intake in mothers older than 30 increases the risk of developmental abnormalities. When mothers drank five drinks per occasion at least once a week, FAS or ARND was mostly the result. The minimum quantity of alcohol required to produce adverse fetal consequences is still unknown and, therefore, it is clinically prudent to advise abstinence during pregnancy (Abel, 1998a).

The amount of alcohol consumed in pregnancy is, however, not the only important factor of note. Other contributing factors exist. More significantly, in alcohol related fetal anomalies are the peak blood alcohol level reached. Binge drinking may therefore be more harmful to the developing fetus than more steady patterns of alcohol consumption (Abel & Hannigan, 1996; Committee on Substance Abuse and Committee on Children with disabilities, 1993; Gardner, 1997; Olsen, Streissguth, Sampson, Barr, Bookenstein, & Thiede, 1997).

Children do not need to have all the criteria of FAS to have significant difficulties. According to Gusella and Fried (1984), even light drinking can have adverse effects on the child's verbal language and comprehension skills. Research conducted on the neurology of prenatal exposure to alcohol showed that children of mothers who drank but who did not have a diagnosis of FAS have many of the same neurological abnormalities as children who had been diagnosed with FAS (Mattson & Riley, 1998).

7. STRUCTURAL DAMAGE TO THE CENTRAL NERVOUS SYSTEM AND BRAIN

Since the first case of an infant born to a chronic alcoholic mother was documented (Jones & Smith, 1973), post-mortem investigations have been performed. Experimental models of FAS have now revealed several important relationships between analogs of human prenatal exposure and subsequent neurological impairment (Autti-Ramo, et al., 2002; Mattson & Riley, 2000b). Prenatal alcohol exposure can cause changes in brain structure and anomalies in brain functions (Osofsky & Fitzgerald, 2000).

Harmful effects on the brain can occur at any stage as the brain and the central nervous system develops through the entire pregnancy. Various regions of the brain can be affected by alcohol exposure depending on which areas are developing at the time of alcohol consumption. Heavy drinking early in the pregnancy cause facial, musculo-skeletal and organ abnormalities, as seen in children with FAS (Gardner, 1997). Growth, cognitive and behavioural impairment are associated with alcohol consumption in the second and third trimester. Fetal growth is significantly associated with alcohol consumption in the first eight weeks and third trimester of pregnancy. Most commonly it affects the head and brain size (Mattson & Riley, 2000a).

Mattson and Riley (2000a) showed with the use of imaging techniques that prenatal alcohol exposure affects the size of the cerebral cortex that is involved

with higher cognitive functioning. The cerebellum that controls coordination, movement, behaviour and memory are sometimes damaged (Clarren & Smith, 1987). The corpus callosum, the communication pathway that unites the left (rules and logic) and right (impulses and feelings) hemispheres might be smaller in people with FAS. In some cases, it is nonexistent. Abnormalities in the corpus callosum explain the comprised information processing that is seen in children exposed to alcohol before birth. The hippocampus that is involved with memory is also affected (Gardner, 2000). The basal ganglia, and especially the caudate that governs voluntary movement, perseveration and some cognitive functions related to perception of time, thinking and spatial memory are affected. The basal ganglia also play a role in abilities that is required to execute goal directed tasks and to switch modes. The cerebellar vermis (the vermis connects the two halves of the cerebellum) is believed to be involved in attention and gross motor functions such as balance and it is sensitive to the indirect effects of alcohol (Mattson, Schoenfeld, & Riley, 2001).

The most noteworthy damage probably occurs in the prefrontal cortex, which controls the executive functions. Several studies have highlighted affects on the neurotransmitters, especially if exposure occurred early in the pregnancy, as they can cause specific neuro-psychiatric conditions, mental retardation, and other cognitive disorders (Lockhart, 2001). As so many brain regions are affected, FAS children display a variety of decrements. Cognitive difficulties are one of the

most devastating effects due to its long-term consequences (Committee on Substance Abuse and Committee on Children with Disabilities, 2000).

8. NEUROPSYCHOLOGICAL DEFICITS IN INDIVIDUALS WITH FAS / ARND

Several neuro-psychological tests have been found sensitive to detect neuro-psychological deficits in children prenatally exposed to alcohol as outlined in Table 1. These tests will be discussed in more detail under the various headings following Table 1.

Table 1: Test available to delineate Neuro-Developmental Dysfunctions in children with FAS:

Tests used to measure intelligence may include:

- Bayley Scales of Infant Mental and Motor Developmental (Bayley) resulting in a Mental Developmental Index
Wechsler Scales – yield Intelligence Quotient (IQ)
- Wechsler Preschool and Primary Scales of Intelligence (WPPSI) and WPPSI- R (revised).
 - Wechsler Intelligence Scale for Children (WISC) and WISC- R (revised).
 - Wechsler Adult Intelligence Scales (WAIS)

Tests used to measure attention and hyperactivity may include:

- Winconsin Card Sorting Test (WCST)
Freedom from distractibility subtest (WISC- III)
Parent and teacher rating scales (Conners PRS-R; Conners TRS-R)
CBCL/6-18, CBCL/ 6-18 TRF

Tests used to measure learning and memory may include:

- California Verbal Learning Test (CVLT).

Tests used to measure language may include:

- Word Span Tests
Word Comprehension Tests
Naming tasks
Reading Tests

Tests used to measure motor abilities may include:

- WISC – R
Grooved Pegboard
Beery Developmental Visual and Motor Integration Test (VMI)

Tests used to measure social skills and behaviour may include:

- Vineland Adaptive Behaviour Scale (VABS)
Child Behaviour Check List (CBCL)

Tests used to measure visual – spatial and Gestalt abilities may include:

- Frostig Developmental Test of Visual Perception
Bender Visual-Motor Gestalt Test

Tests used to measure executive functions may include:

- WCST
CVLT
Wechsler Intelligent Scales
-

(Adapted from Canadian Paediatric Society, 2002: p.166)

8.1. Intellectual Functioning

Prenatal exposure to alcohol can severely impact on cognitive functioning (Autti - Ramo, 2002; Lynch, Coles, Corley & Falek, 2003; Rydelius, 1997). Mental handicap is one of the main features and sequelae seen in exposed children, although not a criterion for the diagnosis. Prenatal alcohol exposure is regarded as the most common non-genetic cause of mental handicap (Weinberg, 1997). Surprisingly though only a few studies are based on standardized intelligence tests and reports the frequency distribution of the intelligent quotient (IQ). This makes it difficult to compare the results of studies as different intelligence tests from different countries were used. The reliability and inter-correlation coefficients of these tests also vary. The lack of comparability and the paucity of data could be reasons why results on this matter differ so vastly (American Academy of Pediatrics, 1993, 1998; Committee on Substance Abuse and Committee on Children With Disabilities, 2000). Therefore, only studies where standardized tests (as demonstrated in Table 1) were used as shown in Table 2, and will be reviewed.

Tabel 2: IQ's of patients with FAS

Study	<i>n</i>	Age range	IQ	
			Mean (IQ)	Range (IQ)
Streissguth et al.(1991)	52	12-40	68	20-105
Steinhaussen et al.(1996)	25	3-15	89	86-115
Autti-Ramo et al.(2002)	17	12-14	84	64-103
Kaemingk et al. (2003)	20	6-16	66	55-89

There is a lot of variability found in the IQ ranges of individuals with FAS as demonstrated in Table 2. The IQ's ranges from severely retarded to normal (Olsen et al., 1997). Although some FAS children achieved normal IQ's, they may still demonstrate learning disabilities. Even in the absence of specific facial abnormalities, which are crucial for the diagnosis of FAS, cognitive deficits were still evident. The Mental Development Index (MDI) of the Bayley Scales of Infant Development, a standardized measure of infant development, has been used to assess infants and toddlers (Testa et al., 2003). This test has been found sensitive enough to discriminate between exposed children and controls (Canadian Paediatric Society, 2002; Van der Leeden, 2001).

Spohr and Steinhausen (1996) tested 70 children and found that 34.3% of the children obtained IQ's in the normal range (86 - 115), 34.3% obtained IQ's in the borderline range (71 - 85) and the rest had varying degrees of mental handicap. Streissguth, Aase, Clarren, & Randals (1991) studied 61 participants with FAS/FAE that ranged from 12 to 40 years of age. The Wechsler Adult Intelligence Scale—Revised or Wechsler Intelligence Scale for Children-Revised was used. The average IQ was 68, which place them in the mild mentally disabled range. The range of IQ scores varied from 20 (severely retarded) to 105 (average). In a South African study a mean IQ of 65 was reported (Viljoen, 1999b).

In a follow-up study done by Spohr (1996) it was found that 30% of children started their schooling in a standard primary school, 40% went to a school for pupils with learning problems, and 30% to a school for the mentally challenged. At the end of their schooling (when they were adolescents), 55% of the sample was at schools for children with learning disabilities and 30% at schools for the mentally handicapped. This change for the worse was generally found among the children with ARBD and milder forms of FAS. This study also found that changes in the home environment could not improve the intellectual functioning and academic achievements of exposed children. These findings are not in agreement with previous studies (Spohr & Steinhausen, 1987) that reported compensatory environmental and educational interventions could result in some "biological catch-up maturation" and better developmental outcomes for adolescents and adults with FAS. Steinhausen and Spohr (1984) reported an improvement in intellectual abilities in children in their follow-up study. Upon closer investigation of this study results, however, a high attrition rate was found. Formal testing on standardised intellectual tests were also done on only 13 of the 28 children.

On standardised intelligence tests, exposed children perform poorer on the verbal subscale and demonstrate a relative intact performance on non-verbal competencies. Streissguth et al. (1991) reported that the average performance scale IQ was about 10 points higher than the average verbal scale IQ scores for both FAS and FAE groups in their cohort study.

A longitudinal study highlighted that intelligence remains stable over the life span (Streissguth, Barr et al., 1994). In a test-retest study the relative stability of IQ's into mid-adolescence was demonstrated (Streissguth, Randals, & Smith, 1991). Over an eight year period, group means also remained stable. No significant differences between IQ scores of children with FAS or FAE was found. It thus seems that brain damage due to prenatal alcohol exposure causes long term effects. These findings were supported by cross-sectional and longitudinal data (Steinhaussen, Wilms & Spohr, 1993).

8.2. Mathematical Skills

Deficits in mathematical abilities are the most consistent learning disability mentioned in studies (Jacobson & Jacobson, 2002). In a longitudinal study conducted on white middle class, 7-year old children the Wide Range Achievement Test was used to assess mathematical skills of children with FAS. Difficulties with abstractions like time and space, cause and effect were found. They were also unable to generalize from one situation to the next. Exposed children were unable to calculate with numbers greater than 10 (Spohr, Wilms & Steinhaussen, 1994).

Lower scores on standardised intelligence tests have been reported consistently. In comparison, reading and writing abilities are relatively intact (Committee on Substance Abuse and Committee on Children With Disabilities, 2000; Kerns et al., 1997; Osofsky & Fitzgerald, 2000). This finding seems to be irrespective of

their level of cognitive functioning level. Mathematical impairments were especially demonstrated in adolescents and adults (Streissguth et al., 1994).

In a study performed on young children it was found that mathematical skills were not as impaired as in adolescents and adults (Janson et al., 1995). This could suggest that more complex mathematical skills that are supposed to develop at later stages, fail to develop in children with FAS.

Difficulties with mathematical skills also influence independent living of children and adults, as they are unable to make decisions about finances and their lives. It also makes it difficult for them to enter to the labour market (Steinhaussen, Wilms, & Spohr, 1993).

8.3. Attention and Concentration

Attention deficits have been found in children exposed to low and heavy levels of alcohol prenatally (Committee on Substance Abuse and Committee on Children with Disabilities, 2000; Lockhart, 2001; Sokol, 2000; Streissguth & Kanter, 2000). Maternal alcohol use and smoking during early pregnancy were significantly related to decreased attention and difficulties with sustained attention in the children (Autti-Ramo, 2002). This has been confirmed by a large-scale prospective, longitudinal and population-based study that promotes greater generalizability (Streissguth, Barr, et al., 1994).

Tests that can be used to test for attention and concentration problems are demonstrated in Table 1. They include the WISC-R digit span, Winconsin Card Sorting Test (WCST), teacher and parent rating scales (Canadian Paediatric Society, 2002). The attention problems scale of the Child Behaviour Checklist (CBCL), the Freedom from Distractibility index from the Wechsler Individual Scale for Children–Third Edition (WISC-III) and the Conners PRS-R have been found sensitive enough to distinguish exposed children from non–exposed controls (Lee, Mattson, & Riley, 2004).

Impairments in attention are dose dependant, suggesting that higher alcohol consumption leads to more impairment in attention (Coles, Platzman, & Raskin - Hood). The effects of alcohol on attention, are detectable as early as two days after birth and is still evident at 4 years. Alcohol exposure in utero has the strongest effect on processes such as reaction time, latency to respond, attention and speed, suggesting impairments in the central processing of information (Streissguth et al., 1991).

Kerns, Don, Mateer and Streissguth (1997) found that exposed children with an average IQ performed in normal limits on basic attention tasks. On tasks that required higher levels of mental control and processing efficiency they, however, struggled. Researchers in this area believe it to be due to the sensitivity of the cerebellum, a structure that is located at the back of the brain and that is mainly

involved with movement and cognitive processes like attention (Coles, et al., 1997).

Over the years a possible relationship between prenatal exposure to alcohol and attention deficit hyperactivity disorder (ADHD) have been postulated. An association between ADHD and prenatal alcohol exposure has been supported by prospective studies of alcohol-exposed children with FAS or ARND (Streissguth et al., 1991). Oesterheld et al. (1998, as cited in Lockhart, 2001) reports that 54,5% of the 22 subjects with FAS that he tested with the Conners scales exhibited symptoms of ADHD. A 2.5 fold increased risk for ADHD when a child was exposed to alcohol before birth has also been mentioned (Mick, Biederman, Faraone, Sayer & Kleinman, 2002).

Nanson and Hiscock (1990) compared 20 children with FAS/FAE with 20 children with ADHD and 20 normal controls. The study indicated that although the children with FAS/FAE were intellectually more impaired, their attention problems were similar and could thus benefit from the same interventions that proved efficient for ADHD.

Coles, Platzman, Lynch and Friedes (2001), on the other hand, believed that attention problems were not secondary to hyperactivity. It was found that children parentally exposed to alcohol and those children diagnosed with ADHD demonstrated a different pattern of neurological deficits.

Coles, Platzman and Raskin-Hood (1997) tried to establish the specific aspects of attention that was impaired in children with FAS. Four factors of attention namely focussing, sustaining, encoding and shifting were investigated. Focussing was measured with the coding subtest of the WISC-R, shifting with the Winconsin Card Sorting Test, sustaining with the computerised vigilance procedure known as Continuous Performance Test and encoding with the number and arithmetic subtests from the Kaufman Assessment Battery for Children. Results indicated that alcohol exposed children struggled more with encoding (learning new material) and shifting (utilizing flexibility in problem solving) of attention whereas children with ADHD experienced difficulties with focussing and sustained attention.

Initially it was thought that children with FAS and those exposed to alcohol before birth will respond to the same treatments that are traditionally used for ADHD but O'Malley, (1994) and Clarren (1995) characterizes FAS children with an atypical attention deficit disorder. According to them, these children do not respond to methylphenidate. Studies on this matter, however, have been inconclusive (Autti-Ramo, 2002) and more research is required to replicate or confirm these results as it has implications for treatment. More in-depth studies are also needed to gain a better understanding of the underlying neuro-biology and its relationship with the clinical diagnosis of ADHD (Lockhart, 2001). A developmental progression approach with regard to the multitude of factors that influence behaviour, social behaviour and emotions is also proposed (Coles et al., 2001).

8.4. Motor Abilities

Motor deficits are found in children exposed to alcohol before birth and it appears that fine motor skills are more affected than gross motor functions (Committee on Substance Abuse and Committee on Children with Disabilities, 2000; Osofsky & Fitzgerald, 2000). Similar results were found in a meta-analytical study on 12-13 month old infants (Testa, Quigley & Eiden, 2003) and animal studies (Weinberg, 1998).

The Beery, VMI and Grooved Pegboard Test have been found sensitive enough to identify motor problems in children with FAS as demonstrated in Table 1. Janzen et al. (1995) used the VMI to evaluate fine motor skills in preschool children. In this race matched controlled study, delayed motor development was found in children exposed to alcohol before birth.

The Grooved Pegboard Test was also used to assess fine motor skills, as it has been proven sensitive enough to differentiate between children with or without FAS. Children with FAS performed significantly slower on both hands, compared to the non-exposed controls. These results support previous findings that fine motor functioning is impaired in children with FAS (Olsen et al., 1997).

Other problems with motor functioning include body tremors, weak grasp, poor hand-eye coordination and uncoordinated motor patterns (Gardner, 2000). Decreased muscle tone, ataxia, hemiplegia and finger tapping speed difficulties

have also been mentioned (Weinberg, 1997). The speed and accuracy of motor tasks were decreased (Olsen et al., 1997).

Other studies produced contrasting evidence and did not find deficits in motor functions. A South African study found that motor functioning in children with FAS was mainly preserved (Viljoen, 1999a). The lack of effects could be due to the poor sensitivity (validity) of the gross and fine motor assessments, used in specific studies. No significant impairments were found in fine motor functioning of preschool children with FAS when tested on the Grooved Pegboard Test. So, perhaps the Grooved Pegboard Test is not suited or sensitive enough when used on young children (Janzen et al., 1995).

8.5. Visual-Spatial Functioning

Visual-spatial functioning relates to the ability to see and understand objects and their spatial relationships. Difficulties in perception and remembering of spatial relationships and recalling of visual details were found in children prenatally exposed to alcohol (Olsen et al. 1997; Uecker & Nadel, 1996) and supported by animal studies (Autti-Ramo, 2002; Committee on Substance Abuse and Committee on Children with Disabilities, 2000).

The Frostig Developmental Test of Visual Perception and the Beery Developmental Test of Visual-Motor-Integration (VMI) have been found sensitive

enough to highlight visual spatial problems in children exposed to alcohol before birth (Canadian Paediatric Society, 2002).

Results of one study revealed that visual-perceptual skills, as tested by the Frostig Developmental Test are even more affected than intellectual functioning. A 12-month delay was found in 47% of the subjects and 68% had a 6-month delay whereas no delays were found in the control group. The FAS group struggled particularly with hand-eye coordination and visual form-perception (Sokol, Black-Delaney & Nordstrom, 2003).

Significant differences were also found between exposed pre-school children and control when tested on the VMI. An average score of 54 was obtained in the exposed group and the control group had an average of 95. When a visual-spatial test without a motor component was administered, no significant differences were found. It thus seems that visual-spatial processes may be intact in young children with FAS (Janzen, Nanson, & Block, 1995).

It appears that not all visual-spatial functions are impaired. Kaemingk, Mulvaney and Halverson (2003) found that their ability to recognize faces was preserved.

8.6. Speech and Language Abilities

Speech and language difficulties have been noted in several studies (Autti-Ramo, 2002; Committee on Substance Abuse and Committee on Children with

Disabilities, 2000). There does not exist a significant relationship between language deficits and cognitive abilities. This suggests the presence of two independent deficits rather than global impairment in children with FAS (Chiriboga, 2003).

Naming, word span, word comprehension and reading tests can be employed to identify specific language or speech problems (Canadian Pediatric Society, 2002). Studies on language functioning of children with FAS/ARBD suggest specific patterns of deficits but there is a paucity of studies in this very important area. Shaywitz and Caparulo (1981) described two case studies with specific references to the language and speech delays of children with FAS. It was found that both the children had difficulty with language comprehension. Their spontaneous speech also contained more semantic errors. Based on formal assessments their speech was also described as monotonous, retarded and forced. They were unable to use language appropriately, to initiate and continue social discourse and used shortened sentences in comparison to their age counterparts. Overall, it appears that communication abilities of children prenatally exposed to alcohol were not age appropriate (Streissguth et al., 1991).

A study on language acquisition in socio-economically disadvantaged children prenatally exposed to alcohol yielded different results. The relationship between maternal alcohol use during pregnancy and language development was statistically insignificant. These findings demonstrate the importance for controls,

matched for race and socio-economic status, to ascertain possible differences between children with FAS and their peers (Abel, 1984).

Language skills are important for the development of ego development and self-regulation. Further studies are needed in this important area (Canadian Paediatric Society, 2002). Language difficulties of children with FAS can further hamper their behaviour and social relationships. Language impairments impacts in all spheres of life and makes children with FAS vulnerable to exploitation and unintentional abuse (Chiriboga, 2003).

8.7. Verbal Learning and Memory

Individuals exposed to alcohol in utero display verbal learning memory difficulties (Committee on Substance Abuse and Committee on Children with Disabilities, 2000; Mattson et al., 2001; Sokol et al., 2003; Weinberg, 1998) as indicated by human and animal studies (Coles et al., 2001). A meta-analytical study also confirmed impairments in verbal learning memory as well as subjective reports from mothers of children with FAS (Tessa, Quigley & Eiden, 2003).

Streissguth (1990) illustrated impaired spatial memory and integration and poor verbal memory by means of neuropsychological testing. On the California Verbal Learning Test (CVLT), a test of verbal memory and learning, children with FAS struggled to recall a list of words after the initial presentation. After a 20-minute delay, they recalled fewer words than the control group. Their learning curves

were normal but overall learning was suppressed. Children with FAS made more errors and perseverations. Retention for learned words after a distraction list fell in the average range over both long and short delays. Recognition was below average as these children demonstrated significantly poorer discrimination of list from non-list words and significantly more false-positive answers (Kaemingk et al., 2003; Kerns et al., 1997). They made little use of semantic clustering which is indicative of a shallow level of encoding and difficulty employing effective learning strategies (Jacobson et al., 2002).

Mattson (1992) tried to establish the impact of IQ on verbal memory. An attempt was made to control for effects of IQ in a study with two boys with mental handicap and FAS. On a test of word-list learning, the two boys demonstrated impaired immediate and delayed recall, excessive perseverations and source confusion. On a recognition trial, they made an increased number of false positive errors. Similar results were obtained among children with prenatal alcohol exposure but without a diagnosis of FAS (Mattson, Riley, Gremling, Delis & Jones, 1998). It thus seems that irrespective of the level of cognitive functioning, verbal memory was still impaired (Mattson, 1992).

In a qualitative study with the (foster) parents children with FAS, a mother described her child's memory problems as follows *"it's like an on/ off switch in her brain. She wakes up one day and she cannot even remember how to pick up a spoon and self-feed, and yet, the night before she was managing rice and soups*

and everything else without dripping" (Gardner, 2000). This finding further highlights the impairments in learning and short-term memory.

8.8. Executive Functioning

Several studies have highlighted decrements in executive functioning (EF) (Mattson et al., 2001; Schonfeld, Mattson, Lang, Delis & Riley, 2001) and reported that all areas are affected (Autto-Ramo, 2002; Mattson et al., 1999). Executive functioning (EF) skills encompass all skills that enable us to make decisions, consider different options about how to obtain the desired object, initiate and sustain a complex sequence of actions to obtain the object, and self-monitor and self-correct, if the chosen plan of action is not working. These high order cognitive processes are required for daily living skills, independent functioning and self-perception (Lezak, 1995; Mattson, Goodman & Caine, 1999). Weinberg (1997) cited that the learning problems, as well as the behaviour problems highlighted in various studies resemble those resulting from frontal lobe damage.

Mattson and Riley (2000b) employed a non-exposed control group matched with the prenatal alcohol exposed group for age, sex, verbal IQ, socio-economic status and ethnicity. It was found that the abovementioned factors were not responsible for clinically significant behaviour scores and concurred that prenatal exposure to alcohol was a definite etiological factor for the impairments in executive functioning.

Kodituwakku, Kalberg and May (2001) have gone further and made a distinction between cognitive-based and emotion-related executive functioning. Cognitive based EF refers to deliberate and purposeful actions taken to solve a specific problem or to initiate a solution. Emotion-related EF, in contrast, refers to actions that are selected based on the compensation and the punishment experienced previously in similar situations. Cognitive based EF was measured by the Wisconsin Card Sorting Test (WCST), where the subject sort cards by different dimensions. This measures the subject's ability to make cognitive shifts. On the other hand, emotion-related EF was measured by visual discrimination reversal tests, where the subject learns must constantly adjust their responses as associations change. Research results indicated impairment in both types of EF in children with FAS and those exposed to moderate amounts of alcohol (7-13 drinks per week).

8.8.1. Initiation

Children affected by prenatal exposure to alcohol experience difficulties in self-activation to work, to organize and to prioritise. Further difficulties lies in sustained effort and the modulation and managing of frustrations and emotions (Mattson et al., 1999). It is noticed, that these children perform better on neuro-psychological tests than one would expect based on their performance in school or work related areas of their lives. This could be due to the structured nature of executive functioning tests or neuro-psychological tests in general (Lezak, 1995).

8.8.2. Information Processing

In a study conducted on 13-month old infants, who have been exposed to alcohol in utero, it was found that these children displayed decreased speed of information processing (Jacobson, Jacobson, Sokol, Martier, Ager & Kaplan-Estrin, 1993) which is indicative of problems with internal ordering. Studies on adolescents yielded similar results as exposed teenagers struggled to make complex decisions (Streissguth et al., 1994; Olsen et al., 1997), and seem unable to figure out solutions by themselves. They also struggled on tasks that required focussing and encoding (Olsen, et al., 1992).

8.8.3. Self-regulation, Self-monitoring and Impulsivity

Difficulties with self-regulation and self-monitoring have been documented (Olsen et al., 1997). Individuals with FAS made more perseverative designs on the Ruff Figural Fluency Test (RFF), a test for non-verbal fluency. It is hypothesized that reductions in the rate of design fluency are a result of inflexible processing such as seen in frontal lobe damage (Kerns et al., 1997). Poor regulation and modulation of emotions and social behaviour are also reported by mothers of FAS children (Gardner, 2000; Streissguth et al., 1992).

Clinical and anecdotal reports frequently note symptoms of impulsivity, poor attention span and restlessness in children with FAS and ARBD as measured on vigilance task tests and self reports by teachers and parents (Weinberg, 1997). Reports of poor sexual inhibitions especially in social settings have also been

mentioned (Steinhausen, 1996). Streissguth and Barr et al. (1991) noted weak problem solving skills. Children with FAS require constant feedback and supervision due to their impulsive nature and poor self-regulatory skills. Deficits in language and other executive functions may all play a role in failures of self-regulation (Tarter, Hedgedus, Goldstein, Shelly, & Alterman, 1997).

8.8.4. Abstract Reasoning

Children with FAS or those who have been exposed to alcohol in utero experience difficulties with abstract reasoning and comprehension of complex relations. Persistent deficits in central cognitive processing have been noted (Mattson et al., 1999). Exposed individuals seemed unable to relate one question to another and to apply consequences from past actions to guide current behaviour or decisions (Kodituwakku et al., 2001). For example, in studies that looked at the justice system it was apparent that affected individuals did not comprehend the consequences of providing the police with an inculpatory statement (David, 2000; Lynch et al., 2003). Problems with concept formation have also been noted (Olsen et al., 1997).

8.8.5. Planning and Cognitive Shifting

Cognitive shifting or flexibility refers to the ability to attend to multiple criteria simultaneously and to shift attention during a task. Children and adults displayed impaired attention shifting (Mattson et al., 1999; Olsen, Feldman, Streissguth & Gonzalez, 1992). They found it very difficult to switch from naming animals to

naming furniture and back to animals again. Toddlers frequently struggle to make transitions when they play (Autti-Ramo, 2002).

With regard to planning, children exposed to alcohol before birth demonstrated poor performances as tested by the Tower of Hanoi (Jacobson & Jacobson, 2002) as well as in a clock drawing test (Spohr et al., 1994).

8.9. Behavioural Problems

Olsen et al. (1997) found a relationship between the amount of alcohol use in utero and the severity of behaviour problems. The Center for Disease Control and Prevention (2000) found that between 1993 and 1997, 45.8% of children with FAS were referred due to conduct problems. A wide range of behavioural deficits were observed in children with FAS. The etiology of these behavioural difficulties is complex as a variety of environmental factors must also be considered.

Babies with FAS are described as irritable. Preschool children with FAS struggle to make transitions (Gardner, 1997). In middle school-aged children hyperactivity and impulsivity are the most common behavioural traits mentioned (Janzen et al., 1995). In a population based cohort study it was found that when mothers regularly binged on alcohol, their offspring were more inclined to present with antisocial personality disorder/traits and school problems (Mattson et al, 2001).

The Vineland Adaptive Behaviour Scale (VABS) has been used to measure the behaviour of children with FAS. Their best performance was on the daily living skills that were on a 9 year level and socialization skills were on a 6 year level. According to the VABS, 68% of the participants presented with significant behaviour problems. The behaviour problems include dependency, inflexible, bullying, and anxiety. Delinquent behaviour like lying and stealing was also found (Streissguth et al., 1991).

The Child Behaviour Checklist (CBCL) has also been found sensitive enough to differentiate between children with and without FAS. On average 47.6% children with FAS exhibited behaviour problems, compared to 17.9% behaviour problems noted in the control group. Findings from this study yielded similar results like hyperactivity, impulsivity, delinquent behaviour and self-mutilation was found in 40% of the patients (Janzen et al., 1995).

Streissguth, Barr, Kogan and Bookenstein (2000) found that 90% of patients in their study had mental health problems, 60% left school, 60% had trouble with the law and 50% had drug problems. Recurring inappropriate sexual behaviour was also found in 50% of the patients.

8.9.1. Social Skills

Children with FAS have poor social skills. Kelly, Day and Streissguth (2000) found that infants have difficulty attaching to the mother. They tend to withdraw

socially and their affect is inappropriate (Shaywitz et al., 1981). Exposed children struggle to construe social cues and have poor interpersonal relationships. They also struggle to adapt to new situations (Streissguth et al., 1991). Similar behaviour was seen in animal studies. They concluded that prenatal alcohol exposure caused social deficits (Kelly et al., 2000).

8.9.2. Judgement

Children with FAS and those who have been exposed to alcohol before birth display poor judgement on tests and in their personal domain (Gardner, 2000; Kerns et al., 1997; Steinhausen et al., 1993; Weinberg, 1997). Their impaired judgement could possibly explain problem behaviour like stealing and lying that are often reported in individuals with FAS (Gardner, 2000; Lynch et al., 2003).

8.9.3. Aggressiveness

Gardner (2000) cited that some children can become extremely aggressive. A foster mother described her child as "volatile and extremely aggressive". They displayed destructive behaviour and high pain tolerance. Continued supervision for these children is indicated into adolescence and adulthood in order for them to gain some sense of independence. Mattson and Riley (2000) found elevated scores on externalising scales that include aggressiveness when the Child Behaviour Checklist (CBCL) was administered. A control group was used. It was

concluded that socio-economic factors and verbal IQ did not cause the behaviour problems of exposed children.

8.9.4. Impulsivity and Hyperactivity

Signs of hyperactivity and impulsivity has been of the most consistent descriptions given to individuals with FAS (Coles et al., 1997) and are often diagnosed with ADHD as discussed earlier in this thesis. Impulsivity and hyperactivity severely impacts on academic and social functioning (Olsen et al., 1997).

9. DISCUSSION

The teratogenic characteristics of alcohol have been well established. There is conclusive evidence from epidemiological and experimental studies to support this (Abel & Hanningan, 1996). Prenatal alcohol exposure has numerous structural and functional effects on the developing fetus. The brain is particularly vulnerable. The pattern and the severity of these effects depend on the dose, timing, pattern and duration of the alcohol exposure.

It seems that the type of cognitive and neuro-psychological deficits do not differ significantly between people with FAS and those exposed to lower levels of alcohol and who do not meet the criteria of FAS. It thus seems that neuro-psychological deficits occur even in the absence of physical features associated with FAS (Mattson et al., 1998) and that alcohol has a specific rather than global

or diffuse effect on the brain. The severity of the effects is dose dependant implying that higher alcohol levels leads to more serious cognitive impairments.

Many studies done on children with FAS have focussed on their levels of cognitive functioning. The range of IQ's vary from severely disabled to normal (Olsen et al., 1997). Even when a normal IQ was obtained, affected individuals still demonstrated other learning difficulties. On standardized intelligence tests, children with FAS performed better on the non-verbal subscale than the verbal one.

The level of cognitive functioning remains stable over time and impacts on various areas of life. Most children exposed to heavy alcohol prenatally attend special schools and exhibit many behavioural problems as well. They include aggressiveness, poor impulse control, hyperactivity, problems with social interactions and delinquent behaviour.

Children with FAS display difficulties with language comprehension and make more syntactic-semantic mistakes in their spontaneous speech. They use language inappropriately, hyper-articulate and lack emotion when they express themselves. Difficulties in speech and language impede on how they are received and treated in social settings. Speech difficulties also influence ego development. Problems with language and speech also leave them vulnerable to exploitation and abuse by others.

A short attention span and concentration difficulties have also been reported as tested by the WCST, CBCL, Freedom from distractibility subtest (WISC-III) and parent and teacher rating scales. There is also an association between attention deficit hyperactivity disorder and prenatal exposure to alcohol. Although still controversial, recent studies indicate an atypical ADHD although clinical presentations are similar. Children prenatally exposed to alcohol struggle with encoding and cognitive shifting whereas children with ADHD experience problems with focussing and sustained attention. Based upon these results it seems that different treatment strategies would be required.

Impairments in hand-eye coordination and visual form-perception have been described as measured by the Beery and Frostig Developmental Test of visual perception. Affected individuals experience difficulty with copying tasks and struggles to remember spatial relationships. Mathematical decrements are the most consistent reported finding across all the studies as problems with basic and more complex mathematical calculations were found.

Specific impairments in verbal learning memory have been demonstrated by the CVLT. Overall, learning was poor and they made significantly more errors. Their approach to verbal tasks reflects a shallow level of encoding as well as poor learning strategies that necessarily influences academic functioning. Their ability to retain words after a distraction task was intact. This suggests that these children can learn and retain skills and information after several repetitions.

Generally, it seems that fine motor skills are more affected than gross motor functioning as indicated on the Grooved Pegboard Test, Beery and VMI Test.

All areas of executive functioning are reportedly impaired. This includes problems in self-monitoring, cognitive shifting and initiation as measured by the CVLT. Their ability to think abstractly are affected and they demonstrate planning difficulties. They are impulsive, aggressive and demonstrate poor insight and judgement. Poor socialization skills were also noted as one of the key behavioural difficulties experienced by children prenatally exposed to alcohol.

The results of several studies indicated that alcohol use by pregnant women even in the absence of self reported problems of alcohol abuse can have long-term behavioural effects on children. Longitudinal and population-based studies also confirmed this (Streissguth, Barr, Sampson & Bookenstein, 1994). Aronson and Hagberg (1998) followed up 24 children from the age of 12 to 14. Results from this study confirmed that behavioural and neuro-psychological deficits persist after childhood.

For children prenatally exposed to alcohol several functional impairments increase over time. Streissguth et al. (1991) found that a small number of adults (less than 10%) with FAS could function independently in terms of housing and income. This is further influenced by their lack of social skills (difficulty perceiving social cues) and hyperactive and inattentive nature, as these symptoms were still

evident during adolescence (Streissguth et al., 1996) and adulthood (Gardner, 2000). As independent functioning are compromised, continuous supervision is required at work and at home.

An early diagnosis of FAS is imperative as early intervention can reduce the impact of neuro-psychological deficits (Streissguth, 2000) and thus improve the overall quality of life for individuals with FAS or those with related difficulties. However, successful detection of prenatal alcohol effects relies on a carefully constructed method utilizing a multifactorial approach to exposure. An assessment should include a battery of focussed and sensitive neuro-psychological tests (as outlined in Table 1).

Questions about prenatal exposure to substances including alcohol should be a routine part of the psychiatric intake interview for patients of all ages. A complete alcohol history should be taken. The possibility of teratogenic alcohol effects and thus some organic basis for the problem behaviour should be entertained in the diagnosis as this influences treatment and prognosis.

It is imperative that we challenge schools and communities to provide appropriate remedial experience and shelter for these patients in order to prevent discrimination, dysfunctional lives, increased incidence of psychopathology, chronic mental illness, and homelessness. Health and social service professionals should be educated and trained on how to identify affected

individuals and appropriately intervene with women at risk for alcohol-exposed pregnancies. Interventions should target the various deficits as highlighted by neuro-psychological evaluations. Programmes aimed at prevention of alcohol use/abuse during pregnancy appeared to be of high priority, particularly in areas such as the Cape Province in the RSA.

10. Limitations:

Several limitations have been highlighted in the literature. The stigma around FAS leads to under-diagnosing of the syndrome by many medical workers, as there is a reluctance to label. This reluctance is one reason why the exact prevalence cannot be estimated and current statistics probably reflect underestimations (Stoler & Holmes, 1999).

Several studies have suggested that some individual anomalies are associated with lower levels of drinking, commonly referred to as light or moderate drinking. In nearly every instance, these findings are attributable to either lumping children whose mothers were heavy drinkers (defined as consuming five drinks or more per occasion) or alcohol abusers (defined as drinking five drinks or more regularly, e.g. two or more times a week) together with those of moderate drinkers. This gives a skew picture and makes it difficult to figure out the impact that lower levels of alcohol consumption has on the developing fetus (Abel, 1998).

Many studies have lacked control groups. Studies are often too small, due to the stigma associated with FAS and this limits the generalizability of study results. Clinical studies suffer from ascertainment biases that plague all clinical studies that depend on referrals. Where retrospective study designs were utilized, the possibility of recall bias exist. Mothers who abused alcohol while pregnant might be less inclined to report this or to accurately report the amount of alcohol consumed because of guilt. Retrospective studies also make it difficult to ascertain how much of the behaviour problems are a direct result of the prenatal alcohol exposure or due to adverse postnatal environments. Therefore, when we discuss and infer problems due to alcohol use, the vast heterogeneity of the population must always be kept in mind and deterministic predictions should be avoided.

11. Future Research

Several areas render closer inspection. Research in mental health treatment is of vital importance. Mental health professionals need to be better informed about the impact of prenatal exposure to alcohol as that could have implications on examinations and treatment plans.

The question of an actual threshold remains unanswered because of the methodological difficulties involved. An actual threshold will prove valuable when educating pregnant women.

More research is needed to broaden our understanding about the areas of the brain that are affected by alcohol abuse, and how these brain changes affect daily behaviour. Better-designed studies are required to establish brain and behaviour correlations. Researchers can then work more effectively to design means of intervention and perhaps prevention. Further evaluations into the duration of neuro-psychological deficits are necessary. There is a need to find tests that is sensitive enough to detect neuro-psychological and cognitive deficits earlier and more accurately. Standardized tests should also be used, as this is the only manner in which a variety of cognitive impairments can reliably be measured and documented.

Few studies have been conducted on preschool children and adults with FAS. Future studies on the neuro-psychological performance of children with FAS should include both cognitively impaired and normal control groups.

Too little has been done on how prenatal alcohol exposure effect attachment behaviour, emotional development and the psychopathology that are often seen in this population. These areas of study are important as it has implications for these children in terms of their personal, academic, social and behaviour development.

Research into preventative measures and strategies are needed as early intervention can avoid or minimize the secondary disabilities that many of these people face.

12. References

- Aase, J. M., Jones, K.L., & Clarren, S.T. (1995). Do we need the term "FAE"? *Pediatrics*, 95 (8), 428-430.
- Abel, E.L. (1984). Prenatal effects of alcohol. *Drug and Alcohol Dependence*, 14(1), 1-10.
- Abel, E. R. (1998). Prevention of Alcohol abuse-related birth effects- 1. Public education efforts. *Alcohol and Alcoholism*, 33(4), 411-416.
- Abel, E. R. (1998). Prevention of alcohol abuse-related birth effects- II. Targeting and Pricing. *Alcohol and Alcoholism*, 33(4), 417-420.
- Abel, E. R., & Haningan, J. H. (1996). Risk factors and pathogenesis. In H.L. Spohr & H. C. Steinhausen (Eds). *Alcohol, Pregnancy and the developing child*. Australia: Cambridge University Press.
- Achenbach, T. M., Eidelbrock, C. (1991). *Manual for the Child Behaviour Checklist/4-18 and 1991 child behaviour profile*. Burlington, VT: University of Vermont.
- American Academy of Pediatrics. (1993). Fetal Alcohol Syndrome and Fetal Alcohol Effects. *Pediatrics*, 91(5), 1004-1006.
- American Academy of Pediatrics. (1998). Identification of children with Fetal Alcohol Syndrome and opportunity for the referral of their mothers for primary prevention-Washington, 1993-1997. *Mortality and Morbidity Weekly Report*, 47(40), 861-864.
- Armstrong, E. M., & Abel, E. L. (2000). Fetal Alcohol Syndrome: The origins of a moral panic. *Alcohol and Alcoholism*, 35(3), 276-282.

- Aronson, M., & Hagberg, B. (1998). Neuropsychological disorders in children exposed to alcohol during pregnancy: A follow-up study of 24 children to alcoholic mothers in Goteberg, Sweden. *Alcoholism: Clinical and Experimental Research*, 22(2), 321-324.
- Astley, S. J., & Clarren, S. T. (2000). Diagnosing the full spectrum of Fetal Alcohol-Exposed individuals: Introducing the 4-digit diagnostic code. *Alcohol and Alcoholism*, 35(4), 400-410.
- Autti-Ramo, I, Autti, T, Korkman, M., Kettunen, K., Salonen, G., & Valanne, G. (2002). MRI findings in children with school problems who had been exposed prenatally to alcohol. *Developmental Medicine & Child Neurology*, 44(1), 98-106.
- Autti-Ramo, I. (2002). Fetoel alcohol syndrome- multifaceted condition. *Developmental Medicine and Child Neurology*, 44(1), 141-144.
- Baleta, A. (1998). Fetal alcohol syndrome rife in South Africa. *Lancet*, 352(9124), 295.
- Beery, K. (1982). *Developmental Test of Visual Motor Integration*. Chicago: Follett.
- Canadian Pediatric Society. (2002). Fetal Alcohol Syndrome. *Pediatric Child and Health*, 7(3), 161-174.
- Centers for Disease Control and Prevention. (2002). Defining the National for Fetal Alcohol Syndrome and Other Prenatal Alcohol- Related Effects. *Morbidity and Mortality Weekly Report*, 51(RR-14), 9-12.

- Centers for Disease Control and Prevention. (2003a). Motivational Intervention to Reduce Alcohol-Exposed Pregnancies- Florida, Texas, and Virginia, 1997-2001. *Morbidity and Mortality Weekly Report*, 52(19), 441-444.
- Centers for Disease Control and Prevention. (2003b). Fetal Alcohol Syndrome- South Africa, 2001. *Morbidity and Mortality Weekly Report*, 52(28), 660-662.
- Chandler, L.S., Richardson, G.A., Galagher, J.D., & Day, N.L. (1996). Prenatal exposure to alcohol and marijuana: Effects on motor development of preschool children. *Alcoholism: Clinical and Experimental Research*, 20(7), 455-461.
- Chiriboga, C. A. (2003). Fetal Alcohol and Drug Effects. *Neurologist*, 9(6), 267-289.
- Clarren, K. S., & Smith, D. W. (1978). The Fetal Alcohol Syndrome. *The New England Journal of Medicine*, 298(19), 1063-1067.
- Coles, C. D., Platzman, K.A., & Raskin-Hood, C. L. (1997). A comparison of children affected by prenatal alcohol exposure and attention deficit hyperactivity disorder. *Alcoholism: Clinical and Experimental Research*, 20(1), 150-161.
- Coles, C. D. (1997). Fetal alcohol syndrome, ADHD require different treatment. *Alcoholism & Drug Abuse Weekly*, 9(25), 6.
- Coles, C. D., Platzman, K.A., Lynch, M. E., & Friedes, D. (2001). Auditory and visual sustained attention in adolescents exposed to alcohol. *Alcoholism: Clinical and Experimental Research*, 24(2), 210-225.

- Coles, C.D. (2001). Fetal alcohol exposure and attention: Moving beyond ADHD. *Alcohol Research and Health*, 25(3), 199-204.
- Connor, P. D., & Streisguth, A. P. (1996). Effects of prenatal exposure to alcohol across the life span. *Alcohol Health and Research World*, 20(3), 170-175.
- Croxford, J., & Viljoen, D. (1999). Alcohol Consumption by Pregnant Women in the Western Cape. *South African Medical Journal*, 89(9), 962-965.
- David, S. (2000). How can court deal with FAS? *Medical Post*, 36(21), 512-520.
- Delis, D. C., Freeland, J., Kramer, J. H., & Kaplan, E. (1988). Integrating clinical assessment with cognitive neuroscience: Construct validation of the CVLT. *Journal of Consulting and Clinical Psychology*, 56(1), 123-130.
- Egeland, G. M., Perham-Hester, K. A., Gessner, B. D., Ingle, D., Berner, J. E., & Middaugh, J.P. (1998). Fetal Alcohol Syndrome in Alaska, 1977 through 1992: An Administrative Prevalence derived from Multiple Data Sources. *American Journal of Public Health*, 88(5), 781-786.
- Gardner, J. (1997). Fetal Alcohol Syndrome - Recognition and Intervention. *The American Journal of Maternal/Child Nursing*, 22(6), 318-322.
- Gardner, J. (2000). Living with a child with fetal alcohol syndrome. *The American Journal of Maternal/Child Nursing*, 25(5), 252-257.
- Goodlett, C.R., & Horn, K.H. (2001). Mechanisms of alcohol induced damage to the developing nervous system. *Alcohol Research and Health*, 25(3), 175-184.
- Grant, D. A., & Berg, E. A. (1980). *The Wisconsin Card Sorting Test*. San Antonio: Psychology Corporation.

- Gusella, J. L., & Fried, P. A. (1984). Prenatal exposure to alcohol. *Neurobehavioural Toxicology and Teratology*, 6(1), 13-17.
- Hayden, M. R., & Nelson, M. M. (1978). The Fetal Alcohol Syndrome. *South African Medical Journal*, 16(3), 571-578.
- Jacobs, E. A. (2000). Fetal Alcohol Syndrome and Alcohol-Related Neurodevelopmental Disorders. *Pediatrics*, 106(2), 358-361.
- Jacobson, J.L., & Jacobson, S.W. (2002). Effects of prenatal alcohol exposure on child development. *Alcohol Research and Health*, 26(4), 282-286.
- Jacobson, J. L., Jacobson, S. W., Sokol, R. J., Martier, S. S., Ager, J. L., & Kaplan-Estrin, M. G. (1993). Teratogenic effects of alcohol on infant development. *Alcoholism: Clinical and Experimental Research*, 17(1), 174-183.
- Janzen, L. A., Nanson, J.L., & Block, G.W. (1995). Neuropsychological Evaluation of Preschoolers with Fetal Alcohol Syndrome. *Neurotoxicology and Teratology*, 17(3), 273-279.
- Jones, K. L., & Smith, D. W. (1973). Recognition of the fetal alcohol syndrome in early infancy. *Lancet*, 2(5), 999-1001.
- Kaemingk, K.L., Mulvaney, S., & Halverson, P.T. (2003). Learning following prenatal alcohol exposure: performance on verbal and visual multitrial tasks. *Archives of Clinical Neuropsychology*, 18(1), 33-47.
- Kearns, K.A., Don, A., Mateer, C. A., & Streissguth, A. P. (1997). Cognitive deficits in nonretarded adults with Fetal Alcohol Syndrome. *Journal of Learning Disabilities*, 30 (6), 685-694.

- Kelly, S. J., Day, N., & Streissguth, A. P. (2000). Effects of prenatal alcohol exposure on social behaviour in humans and other species. *Neurotoxicology and Teratology*, 22(2), 143-149.
- Kodituwakku, P. W., Kalberg, W., & May, P. A. (2001). The effects of alcohol exposure on executive functioning. *Alcohol Research and Health*, 25(3), 192-198.
- Lee, K. T., Mattson, S. N., & Riley, E. P. (2004). Classifying children with heavy prenatal alcohol exposure using measures of attention. *Journal of the International Neuropsychological Society*, 10(2), 271-278.
- Lezak, M. D. (1995). *Neuropsychological Assessment* (3rd Ed.). New York: Oxford University Press.
- Lockhart, P. J. (2001). Fetal alcohol spectrum disorders for mental health professionals – A brief review. *Current Opinion in Psychiatry*, 14(5), 463-469.
- Lovell, J. (1995). Fetal Alcohol Syndrome. *Modern Medicine of South Africa*, 19(2), 75-77.
- Lynch, M. E., Coles, C.D., Corley, T., & Falek, A. (2003). Examining delinquency in adolescents differentially prenatally exposed to alcohol: The role of proximal and distal risk factors. *Journal of Studies on Alcohol*, 64(5), 678-682.
- Mattson, S. N., Riley, E. P., Gramling, L., Delis, D. C., & Jones, K.L. (1998). Neuropsychological comparison of alcohol exposed children with or

without physical features of fetal alcohol syndrome. *Neuropsychology*, 12 (1), 146-153.

Mattson, S. N., Goodman, A. M., & Caine, C. (1999). Executive functioning in children with heavy prenatal alcohol exposure. *Alcoholism: Clinical and Experimental Research*, 23(5), 1808-1815.

Mattson, S. N., & Riley, E. P. (2000). Parents ratings of behaviour in children with heavy prenatal alcohol exposure and IQ-matched controls. *Alcoholism: Clinical and Experimental Research*, 24(2), 226-231.

Mattson, S. N., & Riley, E. P. (2000). Neurobehavioural and Neuroanatomical effects of heavy prenatal exposure to alcohol. *Neurotoxicology and Teratology*, 25(1), 3-14.

Mattson, S. N., Riley, E. P., Jernigan, T. L., Ehlers, C. C., Delis, D. C., Jones, K. L., Stern, C., Johnson, K. A., Hesselink, J. R., & Bellugi, Y. (1992). Foetal Alcohol Syndrome: A case report of neuropsychology, MRI, and EEG assessments of two children. *Alcoholism: Clinical and Experimental Research*, 16(5), 1001-1003.

Mattson, S. N., Schoenfeld, A.M., & Riley, E.P. (2001). Teratogenic effects of alcohol on brain and behaviour. *Alcohol Research and Health*, 25(3), 185-192.

May, P. A., Brooke, L., Gossage, P., Croxford, J., Adams, C., Jones, K. L., Robinson, L., & Viljoen, D. (2000). Epidemiology of Fetal Alcohol Syndrome in a South African community in the Western Cape Province. *American Journal of Public Health*, 90(12), 1905-1912.

- Mick, E., Biederman, J., Faraone, S. V., Sayer, J., & Kleinman, S. (2002). Case-control study of Attention-Deficit Hyperactivity Disorder and Maternal Smoking, Alcohol use, and drug use during pregnancy. *Journal of Academic Child and Adolescent Psychiatry, 41*(4), 378-385.
- Nanson, J. L., & Hiscock, M. (1990). Attention deficits in children exposed to alcohol prenatally. *Alcoholism: Clinical and Experimental Research, 14*(11), 656-661.
- Niccols, G. A. (1994). Fetal alcohol syndrome: Implications for psychologists. *Clinical Psychology Review, 14*(2), 91-111.
- O` Connor, M. J., & Whaley, S. E. (2003). Alcohol use in pregnant low-income women. *Journal of Studies on Alcohol, 64*(6), 773-778.
- Olsen, H. C., Sampson, P. D., Streissguth, A. P., & Bookenstein, F. L. (1992). Prenatal exposure to alcohol and school problems in late childhood: A longitudinal prospective study. *Development and Psychopathology, 4*(3), 341-359.
- Olson, H. C., Streissguth, A. P., Sampson, P. D., Barr, H. M., Bookenstein, F. L., & Thiede, K. (1997). Association of prenatal alcohol exposure with behavioural and learning problems in early adolescence. *Journal of American Academic Child and Adolescent Psychiatry, 36*(9), 1187-1194.
- Osofsky, J. D., & Fitzgerald, H. E. (2000). Prenatal and postnatal exposure to parental alcohol use and abuse. *Handbook of Infant Mental Health, 4*(4), 125-157.

- Ruff, R. (1988). *Ruff Figural Fluency Test: Administration Manual*. San Diego: Neuropsychological Resources.
- Rydellius, P. (1997). Annotation: Are children of alcoholics a clinical concern for child and adolescent psychiatrists of today? *Journal of Child Psychology and Psychiatry*, 38(6), 615-624.
- Sampson, P. D., Streissguth, A. P., & Bookenstein, F. L. (1997). Incidence of fetal alcohol syndrome and prevalence of alcohol related neurodevelopmental disorder. *Teratology*, 56(2), 317-326.
- Shaywitz, S. E., Caparula, B. K., & Hodgson, E. S. (1981). Developmental language disability as a consequence of prenatal exposure to ethanol. *Pediatrics*, 68(6), 850-855.
- Schonfeld, A. M., Mattson, S. N., Lang, A. R., Delis, D. C., & Riley, E.D. (2001). Verbal and nonverbal fluency in children with heavy prenatal alcohol exposure. *Journal of Studies on Alcohol*, 62(2), 239-245.
- Sokol, R. J., Black-Delaney, V., & Nordstrom, B. (2003). Fetal Alcohol Spectrum Disorder. *Journal of the American Medical Association*, 290(22), 2996-2999.
- Spohr, H. L., Willms, J., & Steinhausen, H. C. (1994). The fetal alcohol syndrome in adolescence. *Acta Paediatrica, Supplement (404)*, 19-26.
- Spohr, H. L., & Steinhausen, H. C. (1987). Follow-up studies of children with fetal alcohol syndrome. *Neuropediatrics*, 18(1), 13-17.
- Spohr, H. L., & Steinhausen, H. C. *Alcohol, Pregnancy and the developing child*. (1996). Great Britain: Cambridge University Press.

- Steinhaussen, H. C. (1996). Psychopathology and cognitive functioning in children with fetal alcohol syndrome. In H.L. Spohr and H.C. Steinhaussen (Eds). *Alcohol, Pregnancy and the Developing Child*. Cambridge: University Press.
- Steinhaussen, H.C., Gobel, D., & Nestler, V. (1984). Psychopathology in the offspring of alcoholic parents. *Journal of the American Academy of Child and Adolescent Psychiatry*, 23(4), 465-471.
- Steinhaussen, H.C., Willms, J., & Spohr, H. L. (1993). Long-term psychopathological and cognitive outcome if children with fetal alcohol syndrome. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32(5), 990-994.
- Steinhausen, H. C., Willms, J., Metzke, C. W., & Spohr, H. (2003). Behavioural phenotype in foetal alcohol syndrome and foetal alcohol effects. *Developmental Medicine and Child Neurology*, 45(3), 179-183.
- Stoler, J. M., & Holmes, L. B. (1999). Under-recognition of prenatal alcohol effects in infants of known alcohol abusing women. *Journal of Pediatrics*, 135(5), 430-436.
- Streissguth, A. P., Randals, S. P., & Smith, D. F. (1991). A test-retest study of Intelligence in patients with Fetal Alcohol Syndrome: Implications for care. *Journal of Academic Child and Adolescent Psychiatry*, 30(4), 584-587.
- Streissguth, A. P., Aase, J. M., Clarren, S.K., & Randals, S. P. (1991). Fetal Alcohol Syndrome in adolescents and adults. *Journal of the American Medical Association*, 265(15), 1961-1967.

- Streissguth, A.P., Barr, H. M., Sampson, P. D., & Bookstein, F.L. (1994). Prenatal alcohol and offspring development: the first fourteen years. *Drug and alcohol dependence*, 36(2), 89-99.
- Streissguth, A. P., Barr, H., Kogan, J., & Bookenstein, F. L. (2000). Primary and Secondary disabilities in Fetal Alcohol Syndrome. In A. Streissguth & J. Kanter (Eds.) *The Challenge of Fetal Alcohol Syndrome*. London: University of Washington Press.
- Streissguth, A. P., & Kanter, J. (2000). *The Challenge of Fetal Alcohol Syndrome*. London: University of Washington Press.
- Tarter, R. E., Hedgedus, A. M., Goldstein, G., Shelly, D., & Alterman, A. I. (1997). Adolescent sons of alcoholics: Neuropsychological and personality characteristics. *Alcoholism: Clinical and Exeprimental Research*, 20(2), 216-221.
- Testa, M., Quigley, B. M., & Das Eiden, R. (2003). The effects of prenatal alcohol exposure on infant mental development: A meta-analytical review. *Alcohol and Alcoholism*, 38(4), 295-304.
- Van der Leeden, T., Van Dongen, S., Kleinhout, M., & Phaff, J. (2001). Infants exposed to alcohol prenatally: outcome at 3 and 7 years of age. *Annals of Tropical Peadiatrics*, 21(2), 127-132.
- Viljoen, D. (1999a). Fetal Alcohol Syndrome. *South African Medical Journal*, 89(9), 958-960.

- Viljoen, D. (1999b). Update on the activities of the Foundation for Alcohol Related Research (FARR). *Urban Health and Developmental Bulletin*, 2(1), 19-20.
- Wechsler, D. (1974). *Manual for the Wechsler Intelligence Scale for Children-Revised*. San Antonio: Psychological Corporation.
- Wechsler, D. (1981). *Manual for the Wechsler Adult Intelligence Scale- Revised*. San Antonio: Psychological Corporation.
- Wechsler, D. (1991). Wechsler Intelligence Scale for children – 3rd edition. San Antonio: Psychological Corporation.
- Weinberg, N. Z. (1997). Cognitive and behavioural deficits associated with prenatal alcohol use. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36(9), 1177-1185.