Developmental delay of infants and young children with and without fetal alcohol spectrum disorder in the Northern Cape Province, South Africa

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Abstract
Objective: To describe the extent and nature of developmental delay at different stages in childhood in a community in South Africa, with a known high rate of Fetal Alcohol Spectrum Disorder (FASD). **Method:** A cohort of infants, clinically examined for FASD at two time periods, 7-12 months (N= 392; 45 FASD) and 17-21 months of age (N= 83, 35 FASD) were assessed using the Griffiths Mental Developmental Scales (GMDS). **Results:** Infants and children with FASD perform worse than their Non-FASD counterparts over all scales and total developmental quotients. Mean quotients for both groups decline between assessments across subscales with a particularly marked decline in the hearing and language scale at Time 2 (scores dropping from 110.6 to 83.1 in the Non-FASD group and 106.3 to 72.7 in the FASD group; P=0.004). By early childhood the developmental gap between the groups widens with low maternal education, maternal depression, high parity and previous loss of sibling/s influencing development during early childhood. **Conclusion:** The FASD group show more evidence of developmental delay over both time points compared to their Non-FASD counterparts. Demographic and socio-economic factors further impact early childhood. These findings are important in setting up primary level psycho-educational and national prevention programmes especially in peri-urban communities with a focus on early childhood development and FASD.

Keywords: Developmental Delay; Fetal Alcohol Syndrome (FAS); Griffiths Mental Developmental Scales (GMDS)

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Introduction

Developmental delays in early childhood are often associated with later more permanent neurocognitive deficits. Identifying these delays during infancy is complicated. However, their early detection provides important opportunities for invaluable referrals and intervention, significantly decreasing secondary disabilities in later childhood. Without early detection and intervention for developmental delays the vast majority of children with delays will experience some degree of learning disability limiting their life opportunities.[1] The most common cause of developmental delay worldwide is FAS (Fetal Alcohol Syndrome).[2] Some of the highest prevalence rates of FASD (Fetal Alcohol Spectrum Disorder) have been documented in South Africa, affecting up to 10.9 per 1000 children in heavily affected areas.[3-6] Few studies have assessed developmental delay within impoverished communities in South Africa. Fewer still, have focused on the impact of prenatal alcohol exposure on early childhood development.

FAS is the most severe disorder within the Fetal Alcohol Spectrum, the umbrella term encompassing all disorders associated with prenatal alcohol exposure including, full FAS, Partial FAS and the subtler, often misdiagnosed, behavioural and neurodevelopmental aspects of Alcohol Related

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Neurodevelopmental Disorder (ARND) and Alcohol Related Birth Defects (ARBD).[7] Previous studies document cognitive dysfunction amongst children prenatally exposed to alcohol as being in the borderline range with many showing difficulties in solving problems, shifting attention between tasks, poor memory recall and executive functioning.[8-15] Findings from a study conducted in South Africa on children aged 7 years of age suggests that children with a FASD diagnosis perform lower on higher order cognitive competencies.[16] About two thirds of children with FASD require special services for learning problems in schools, yet limited special service resources are available in South Africa.[17] Public sector psychiatric services are poorly developed in smaller towns and rural areas in South Africa while the child and adolescent sub-speciality remains chronically under-resourced.[18] Further, the integration of mental health care services within primary health care lags.[19] With scarcity of psychiatric specialists in peri-urban communities, limited child mental health services are provided by primary health care staff. Also, there is very little integration of psychiatric services between the departments of health, education or social services. Although previous studies relating to delays during childhood differ in their sampling, research designs and measurements, they consistently confirm the existence of an association between socio-economic circumstances and early development.[20-25] Adverse conditions in developing countries with high rates of poverty and maternal depression after childbirth, are high and compromise childhood developmental outcomes. A key contributor to overall child wellbeing is maternal mental health with maternal depression impacting the early mother-infant relationship and overall child development.[26-29] The bonding relationship said to commence immediately after birth is greatly affected by the child's state as well as the mothers wellbeing. Better infant developmental outcomes have been linked to an increased sense of security in the key attachment relationship. Much is known about the cognitive effects of prenatal alcohol exposure, yet little is known of the early developmental delays and mediating factors amongst infants and young children. This lack of information may partly be attributed to the difficulties in making a FASD diagnosis in infancy.[30] The aim of the present study was to describe the extent and nature of developmental delay over two time periods (7-12 months and 17-29 months) and highlight the impact of FASD and external variables on early childhood development as measured by the infant scales of the GMDS, with important implications for sub specialities within psychiatry.

Methods

Sample

The sample consisted of a cohort of infants born in a one year period between 2002 and 2003 in the public hospital in De Aar. Infants were identified from birth records collected from the hospital (n=500). Potential participants, living in the town (many women in surrounding rural areas deliver in De Aar and return home thereafter), were invited to participate. Of the initial larger sample of infants (younger than 12 months) who received a clinical diagnosis at Time 1 less than half returned for their second assessment during early childhood (older than 12 months) at Time 2. Sample attrition was anticipated due to the longitudinal nature of the study and the characteristics of the population. However, it was further complicated by; the passive follow up of Non-FASD children, families moving out of town, refusal to take part in the second assessment and deaths of mothers or children.

Procedure

Once permission had been obtained from the Department of Health to access birth record data, potential participants were visited at their homes and invited to participate in the study. Infants whose parents/guardians consented to participate received a clinical examination and a developmental assessment. Mothers of infants with possible prenatal exposure were actively followed up and a maternal interview and Beck Depression Inventory was administered.

Identification of FASD group

Two specialist clinicians with dysmorphology training used the Institute of Medicine (IOM) criteria, to diagnose FASD amongst infants and children at Time 1 and 2.[7] A FAS diagnosis was made when infants and children had the characteristic facial phenotype, of small palpebral fissures, midface hypoplasia, smooth philtrum and/or thin vermillion border; with the presence of growth deficiency (on or below the 10th percentile for height and/or weight and/or head circumference) and observed neurological abnormalities. A PFAS diagnosis required at least two of the three facial features, as stated above, with growth deficiency and the presence of neurological abnormality. The clinical diagnosis of FAS, but not PFAS is considered distinctive even in the absence of a history of maternal alcohol consumption in pregnancy.[31,32] The FASD group for this study was defined as the combination of FAS and PFAS diagnosed cases. Infants without a confirmed FAS or PFAS diagnosis formed the Non-FASD comparison group. It is anticipated that some infants in the Non-FASD group had subclinical effects of prenatal alcohol exposure, which may have influenced the developmental performance, but were insufficient to warrant a clinical FASD diagnosis.

Developmental Assessment

The level of cognitive and motor development of infants and children, at both time periods, was assessed using the GMDS.[33,34] These scales, developed in the United Kingdom, have been extensively used in South Africa.[35-48] They show good test-retest reliability and are able to predict long term development.[49] The first five scales assess development from infancy to middle childhood (age 8) with the higher order, sixth scale included at three years of age. Items of the GMDS were adapted for the population and test instructions were translated into Afrikaans and then back translated. Translators were used with caregivers and children who were Xhosa speaking.

Due to the age of the sample being younger than 2 years, general quotients obtained from the subscales were used, as was accepted practise at the time of the study. Measurements for children older than 2 years are now based on percentiles, due to their higher validity.[50] Quotients for each subscale were derived independently and averaged to give a total developmental quotient. Each subtest is uniquely scaled, the mean (μ=100) and standard deviations of 16 were used. Higher scores indicate better performance. If developmental indices could not be determined due to low raw test scores an index score of 50 (the lowest standard score) was assigned. Using the GMDS developmental delay categories, we define developmental delay amongst all of the subscales and total developmental quotient as 2 standard deviations below the expected mean (</=68) and scores of between 1 and 2 standard deviations below the mean (69-83) were described as borderline developmental delay.

Table I: Characteristics of study participants in De Aar, South Africa by infant FASD status			
Variables	*Non-FASD n/N (%)*	*FASD n/N (%)*	*P**
Maternal Variables			
Age, years			
15-19	46/348 (13)	1/43 (2)	
20-24	110/348 (32)	4/43 (9)	
25-29	86/348 (25)	9/43 (21)	
30-34	77/348 (22)	21/43 (49)	
35-60	29/348 (8)	8/43 (19)	<0.001**
Ethnicity			
Black	111/353 (31)	6/45 (13)	
Mixed ancestry	241/353 (68)	39/45 (87)	
Other	1/353 (0.3)	0/45	0.039*
Marital status			
Living together	26/345 (8)	10/45 (22)	
Married/Engaged	79/345 (23)	8/45 (18)	
Single	240/345 (70)	27/45 (60)	<0.006*
Maternal body mass index			
18.5-24.9	3/30 (10)	8/35 (23)	
25-29.9	20/30 (67)	21/35 (60)	
≥30	6/30 (20)	3/35 (9)	0.268
Level of education			
Incomplete primary school	30/325 (9)	15/40 (38)	
Primary school completed	32/325 (10)	10/40 (25)	
Incomplete high school	166/325 (51)	14/40 (35)	
High school completed	92/325 (28)	1/40 (3)	
Tertiary	5/325 (2)	0/40	<0.001**
Maternal occupation			
Unemployed	267/343 (78)	39/45 (87)	
Temporary employed	29/343 (9)	3/45 (7)	
Full time employed	47/343 (14)	3/45 (7)	0.354
Parity			
1	131/343 (38)	3/45 (7)	
2	103/343 (30)	12/45 (27)	
3	54/343 (16)	15/45 (33)	
4+	55/343 (16)	15/45 (33)	<0.001**
Death of a child			
Yes	43/340 (13)	15/45 (33)	<0.001**
Maternal alcohol use during pregnancy†			
1 week	6/39 (15)	1/36 (3)	
2-3 weeks	2/39 (5)	5/36 (14)	
Every week	5/39 (13)	25/36 (69)	
Never	26/39 (67)	5/36 (14)	<0.001**
Maternal depression (Becks Inventory Score)			
Normal (0-10)	72/309 (23)	8/40 (20)	
Mild (11-19)	93/309 (30)	4/40 (10)	
Clinical (20-29)	80/309 (26)	7/40 (18)	
Moderate (30-39)	42/309 (14)	10/40 (25)	
Severe (40+)	22/309 (7)	11/40 (28)	<0.001**
Infant Variables			
Gender			
Male	178/354 (50)	17/45 (38)	
Female	176/354 (50)	28/45 (62)	0.114
Low birth weight (<2.5kg)			
<2.5kg	48/326 (15)	26/42 (62)	
≥2.5kg	278/326 (85)	16/42 (38)	<0.001**
FASD diagnosis	347/392 (89)	45/392 (12)	

Note: FASD=Fetal Alcohol Spectrum Disorder; † = average number of weeks mother drank during pregnancy; p<0.05*; p<0.001**

Maternal Interview

Maternal interviews were used to measure demographic, socio-economic variables and alcohol use within groups. Trained interviewers completed a structured questionnaire with participating mothers, previously developed and tested on local populations.[51] History of alcohol consumption was acquired from mothers of FASD children and an equal number of controls using a using a timeline follow-back method.[52,53] Shorter interviews were used with guardians if mothers were deceased or untraceable. The Beck Depression Inventory was administered to mothers of participating infants to assess cognitions associated with depression.[54,55] Participating mothers received an honorarium in the form of a food voucher and a laminated photo of themselves and their child.

Referrals to local professionals and services, including speech and hearing therapists, occupational therapists and physiotherapists were made with infants and children who presented with FASD or other developmental delays. Other medical conditions detected during clinical examinations of the infants and children were referred to local and regional medical services, as required. Mothers with depressive symptoms were referred to the psychiatric nurse for evaluation and a support group, while those with alcohol dependence were linked with specialist programmes being run through local non-governmental organisations.

The study was nested within a larger prevention study that was conducted in De Aar and the neighbouring town of Upington. The focus of this study was on the De Aar sample. The University of the Witwatersrand Research Committee study reviewed and approved the project (M01-11-20).

Statistical analysis

Clinical data, GMDS mean quotients, infant characteristics, maternal demographic and socio-economic variables were analysed using Intercooled Stata 10.0 (Stata Corporation, College Station, TX). Univariate comparisons for categorical variables were tested using a chi-square test or chi-square test for trend. For continuous variables, the Student's t test and Wilcoxon rank-sum test were used for comparing data with normal and non-normal distributions, respectively. Because standard growth curves were not available for children in South Africa, Centres for Disease Control growth reference curves were used.

Results

About 10% of infants examined at 7-12months of age (n=392) were diagnosed with either FAS or PFAS, forming the FASD group (45/392). Of those, 21% (81/392) returned for their second assessment with 35 diagnosed with FASD during early childhood. More than two thirds of the sample (n= 398, 70.4%) were of mixed ancestry, with 29.4 % classified as black (117/398) (Table I). Demographic data in Table I indicates that almost two thirds of infants (26/42; 61.9%) in the FASD group were of low birth weight (<2.5kg). Mothers of children with FASD were more likely to be older (P<0.001) and single (P<0.006), although most mothers in the community were unmarried. Particularly low levels of education were reported amongst mothers of FASD children, with 35% having no formal secondary schooling. Fifteen of the mothers within the FASD group (33.3%) had more than four children with the same percentage of mothers having lost a child. Twenty five mothers with a FASD diagnosed child (69%) reported drinking alcohol, on average, every week during their pregnancy. Finally, depression was very common amongst women in the community, with 27.5% of the mothers in the FASD group (11/40) having severe depression, and 30% of the mothers in the Non-FASD group (93/309) showing mild depression.

Student t tests were conducted to compare the developmental means between groups over the GMDS subscales. Table II describes the descriptive statistics between groups obtained for each subscale, as well as the total developmental quotient over both time points. Total developmental quotients of the FASD group were significantly lower than the Non-FASD group at both Time 1 (mean=97.7, SD =14; P<0.001) and Time 2 (mean=75.1, SD=14.7; P<0.001). All subscale mean scores for the FASD group were lower than their counterparts at Time 1, with 4 of the 5 subscales showing significant differences, with gross motor functioning being the most marked. No significant differences were found between infant groups on the eye and hand subscale (P=0.37). Both groups performed within the average to above average range (90-119) over all scales on the GMDS categories at Time 1. By Time 2 greater differences between groups emerged. Means at Time 2 dropped over all subscales for both groups when compared to Time 1, although the FASD continue to perform significantly worse over all domains, dropping in their performance from average or above average to within the borderline range (70-84) for all scales. By Time 2 the greatest decrease in performance for both groups occurs on the hearing and language subscale.

Table II: Mean developmental quotients and standard deviations comparing infants with and without fetal alcohol spectrum disorder

GMDS Subscales Mean (SD)	*Time 1 7-12 months*			*Time 2 17-29 months*		
	Non-FASD (n=347)	*FASD (n=45)*	*P**	*Non-FASD (n=48)*	*FASD (n=35)*	*P**
Locomotor	100 (13.4)	89.8 (17.9)	<0.001**	90.1 (15.4)	78.3 (17.6)	<0.001**
Personal-Social	109.4 (15.7)	102.3 (16.7)	0.004*	97.8 (18.8)	79.0 (20.9)	<0.001**
Hearing - Language	110.6 (11.4)	106.3 (12.7)	0.02*	83.1 (15.6)	72.7 (16.7)	0.004*
Eye - Hand Coordination	100.9 (15.7)	98.6 (18.4)	0.37	86.8 (13.5)	75.4 (18.3)	<0.001**
Performance	96.9 (14.5)	92.3 (19.4)	0.05*	82.3 (14.5)	70.6 (16.6)	<0.001**
Total developmental quotient	103.8 (11.2)	97.7 (14.0)	<0.001**	87.8 (12.1)	75.1 (14.7)	<0.001**

Note: GMDS=Griffiths Mental Developmental Scales; SD=Standard deviation; FASD=Fetal Alcohol Spectrum Disorder, p<0.05*; p<0.001**

Table III: Association between maternal and infant characteristics on developmental delay in infancy and early childhood regardless of fetal alcohol syndrome diagnosis

Variables	*Time 1 n/N (% delayed)*				*Time 2 n/N (% delayed)*			
Maternal Variables	*Locomotor*	*Personal-Social*	*Performance*	*Total DQ*	*Locomotor*	*Personal Social*	*Performance*	*Total DQ*
Age, years								
20-24	3/114 (3)	1/114 (1)	3/114 (3)	0/114	3/17 (18)	1/17 (6)	3/17 (18)	2/17 (12)
25-29	2/93 (2)	1/93 (1)	2/93 (2)	1/93 (1)	2/19 (11)	2/19 (11)	3/19 (16)	2/19 (11)
30-34	1/97 (1)	0/97	5/96 (5)	1/97 (1)	6/29 (21)	8/29 (28)	10/29 (35)	7/29 (24)
35-60	1/35 (3)	1/35 (3)	3/34 (9)	1/35 (3)	1/11 (9)	2/11 (18)	2/11 (18)	3/11 (27)
P*	0.753	0.523	0.176	0.491	0.747	0.278	0.398	0.518
Marital status								
Living together	3/36 (8)	1/36 (3)	2/36 (6)	1/36 (3)	2/6 (33)	2/6 (33)	2/6 (33)	2/6 (33)
Married/Engaged	0/86	0/86	3/84 (4)	0/86	2/18 (11)	4/18 (22)	4/18 (22)	5/18 (28)
Single	4/266 (2)	2/266 (1)	8/266 (3)	2/266 (1)	9/55 (16)	9/55 (16)	14/55 (26)	9/55 (16)
P*	0.006*	0.278	0.724	0.278	0.445	0.557	0.863	0.410
Level of education								
No primary school	1/45 (2)	1/45 (2)	5/45 (11)	1/45 (2)	2/15 (13)	3/15 (20)	4/15 (27)	4/15 (27)
Primary school completed	2/42 (5)	1/42 (2)	1/42 (2)	1/42 (2)	5/13 (39)	5/13 (39)	6/13 (46)	5/13 (39)
No high school	3/179 (2)	1/179 (1)	5/178 (3)	1/179 (1)	3/36 (8)	4/36 (11)	7/36 (19)	4/36 (11)
High school completed	1/92 (1)	0/92	2/92 (2)	0/92	0/8	0/8	1/8 (13)	0.8
P*	0.680	0.513	0.073	0.513	0.032*	0.073	0.223	0.062
Parity								
1	2/133 (2)	1/133 (1)	2/133 (2)	1/133 (1)	2/14 (14)	0/14	1/14 (7)	0/14
2	2/115 (2)	1/115 (1)	3/114 (3)	1/115 (1)	2/21 (10)	2/21 (10)	6/21 (29)	3/21 (14)
3	2/68 (3)	0/68	3/68 (4)	0/68	6/23 (26)	9/23 (39)	10/23 (44)	8/23 (35)
4+	1/70 (1)	1/70 (2)	5/69 (7)	1/70 (1)	3/20 (15)	4/20 (20)	3/20 (15)	5/20 (25)
P*	0.892	0.818	0.172	0.818	0.505	0.015*	0.054*	0.065
Death of a child								
Yes	0/58	0/58	4/58 (7)	1/57 (2)	5/19 (26)	7/19 (37)	5/19 (26)	7/19 (37)
P*	0.260	0.463	0.111	0.376	0.206	0.015*	0.848	0.028*
Maternal alcohol use during pregnancy†								
1-3 weeks	1/14 (7)	1/14 (7)	1/14 (7)	1/14 (7)	2/6 (33)	2/6 (33)	4/6 (67)	3/6 (50)
Every week	3/30 (10)	3/30	3/30 (10)	1/30 (3)	5/19 (26)	5/19 (26)	6/19 (32)	6/19 (32)
Never	0/30	1/30 (3)	0/30	0/30	2/11 (18)	4/11 (36)	3/11 (27)	4/11 (36)
P*	0.219	0.381	0.219	0.381	0.774	0.836	0.227	0.715
Maternal depression								
Normal (0-10)	1/80 (1)	1/80 (1)	3/80 (4)	0/80	2/14 (14)	2/14 (14)	3/14 (21)	3/14 (21)
Mild (11-19)	0/97	0/97	1/97 (1)	1/97 (1)	1/14 (7)	1/14 (7)	2/14 (14)	1/14 (7)
Clinical (20-29)	3/87 (4)	1/87 (1)	2/87 (2)	1/87 (1)	2/16 (13)	2/16 (13)	6/16 (38)	2/16 (13)
Moderate (30-39)	0/52	0/52	3/52 (6)	0/52	1/13 (8)	2/13 (15)	2/13 (15)	2/13 (15)
Severe (40+)	2/33 (6)	1/33 (3)	4/33 (12)	1/33 (3)	5/12 (42)	3/12 (25)	4/12 (33)	5/12 (42)
P*	0.094	0.502	0.049*	0.543	0.109	0.784	0.493	0.205
Infant Variables								
Low birth weight								
<2.5kg	4/74 (5)	3/74 (4)	4/74 (5)	3/74 (4)	4/38 (11)	6/28 (21)	7/38 (18)	7/28 (25)
P*	0.014	<0.001**	0.178	<0.001**	0.237	0.553	0.185	0.342

Note: Time 1= 7-12 months of age, Time 2= 17-29 months of age; Total DQ= Total developmental quotient; † = average number of weeks mother drank during pregnancy; p<0.05*; p<0.001**

Table III presents the maternal characteristics associated with developmental delay of the whole population regardless of FASD diagnosis. At Time 1 relationships between marital status and lower locomotor scores (P<0.006) emerged, while low birth weight was significantly associated with delays on the personal and social (P<0.001) and total developmental quotient (P<0.001). Maternal depression further influenced the performance scale (P<0.049). With age, more socio-economic variables contribute to early childhood delay. Low maternal education (P=0.032) is associated with delayed scores on the locomotor subscale during early childhood, while having a higher parity and having lost a sibling (P<0.015) shows delay on the personal and social and performance scales. Gross motor functioning and overall performance are the only two subscales to be consistently influenced by socio-economic variables at both time points, although variation of the socio-economic variable in question exists.

Discussion

Although socio-economic factors like poverty and prenatal maternal alcohol consumption place children at a greater risk for developmental delay, the risk to a larger extent is dependent upon the micro-environment of the child.[56] Overall results from the study indicate that the FASD group show more developmental delay when compared to their Non- FASD counterparts over all developmental subscales. Aside from these anticipated delays associated with prenatal alcohol exposure, the FASD group performed relatively well during their infant assessment, however, showed marked delay during early childhood. With age, the developmental delay between groups becomes more evident and the apparent influence of socio-economic factors more marked. Our findings concur with previous research showing that children from low-income families tend to score within the normal range on cognitive assessments during early infancy but when they remain in disadvantaged environments for the first two years of life, all developmental domains become more significantly deprived.[57-60] Regardless of prenatal alcohol exposure, social factors have been shown to be amongst the strongest predictors of poor developmental outcome with children living in poverty at greater risk for cognitive delays.[61-63]

Results from the current study suggest that maternal marital status is linked to delayed gross motor development during infancy. While by early childhood, levels of maternal education play a larger role. Although specific income indicators were not collected in the current study, maternal level of education and occupation are generally correlated with income.[63] Most researchers concur that maternal education levels are amongst the most reliable predictors of childhood development with similar associations between low maternal educational levels and gross motor functioning been reported in a study amongst black South African children 13 to 16 months of age.[48] On the other hand, researchers using the Bailey Scales of Infant Development failed to find a link between low maternal education levels, their findings suggest that the quality of the home environment affects the gross motor delay of low birth weight infants aged 18 months to 5 years.[64] The current study identified links between high parity and the death of a sibling with delay over developmental scales during early childhood. These factors are often associated with the quality of the home environment, maternal health and the child's relationship with caregivers.

A previous study found high rates of antenatal depression in South Africa, with depression during pregnancy highly associated with antenatal alcohol use.[65]

Research further suggests that mothers with major depression from low income communities are less involved, less sensitive and more negative when interacting with their infants, affecting maternal-child rearing behaviours, impeding cognitive stimulation and directly affecting the maternal-infant bond.[66,67] Because children are less receptive to attachment after three years of age it is imperative that early bonding problems be identified within the first nine months of an infant's life, which may decrease the emergence of many later dysfunctions.

Findings from the current study confirm the association between developmental delay during infancy, on the performance scale measuring manipulation skill, speed and precision of work and maternal depression. The impact of socio-economic factors from infancy through to early childhood on developmental domains fortifies the idea that children develop in relation to their environment, not in isolation to it.[68]

Conclusion

Around 200 million children under the age of 5 in developing countries do not reach their potential.[49] Yet, only a small percentage of research addresses childhood development from impoverished backgrounds.[69] Previous key psychiatric interventions have attempted to use depression and HIV as the port of entry into a family, however this proved difficult, particularly within peri-urban areas with limited mental health services for diagnosis.[70]

With high rates of adverse social factors and alcohol abuse amongst impoverished communities, frontline primary health care staff should make specific efforts to identify high risk families especially among women attending antenatal clinics. Doing this is an essential step for ensuring that women at high-risk receive not only combined interventions for alcohol use, depression and other co-morbid psychiatric disorders associated with alcohol abuse but possibly preventing the birth of a child with FASD. Similarly, early recognition of children with FASD may significantly decrease secondary disabilities associated with Fetal Alcohol Syndrome. Indirectly this type of intervention would strengthen important factors within the micro environment, Firstly, that of the emotional state of the mother and her responsiveness to her child. Secondly, the social support she receives from others and thirdly, the protection offered to the child with particular reference to alcohol use and abuse.[56]

Caution is needed in using and generalising the study findings. The relatively homogenous racial composition of the sample does not reflect South Africa more broadly. Inclusion of only infants from predominately a rural area further limits the study's generalisability. The sample size was small, with limited power to detect differences between groups. The low response rate of Non-FASD children at 17-21 months and possible presence of subclinical effects of alcohol within this group might incur developmental bias. Finally, some limitations stem from the use of the GMDS tool, which tends to measure general ability in completing a given task, but may have low sensitivity for detecting minor developmental delays especially during infancy.[16,48]

Despite these limitations, the current study sheds light on early development of children with and without FASD in an impoverished community in South Africa. Furthermore, it

describes socio-economic factors, present in the community which appear to contribute to the nature and extent of developmental delay. Addressing developmental delay in peri-urban communities is complicated by the lack of specialists and competing demands on primary health care staff. Nevertheless, these findings highlight the importance of high-quality national psycho-educational and prevention programmes that are multidisciplinary and focused on increasing the ability of staff at primary health facilities to identify high-risk families and to provide preventive interventions, especially in antenatal clinics, as well as early intervention services.

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