Original Article

Pregnancy characteristics of women giving birth to children with fetal alcohol syndrome in Far North Queensland

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Background: Fetal alcohol syndrome (FAS) has been identified as a major cause of impairment to normal physical and intellectual development among Indigenous children in Far North Queensland; however, little is known of the pregnancy characteristics of mothers of those children diagnosed with FAS or of interventions that might assist in lowering the prevalence of the syndrome.

Aim: To review the pregnancy records of women whose infants were subsequently diagnosed with FAS by the Paediatric Outreach Service (POS) of the Cairns Base Hospital, and to determine how such women might be identified prospectively in pregnancy and offered intervention to reduce alcohol consumption.

Methods: A retrospective case–control study involving all children diagnosed with FAS by the POS between 1994 and 2006; maternal pregnancy records were accessed and details obtained.

Results: Mothers of cases were older, of higher parity, smoked more cigarettes, attended fewer antenatal visits and experienced more antenatal and delivery complications than mothers of controls. The average gestational age at booking was not statistically significant between the two groups. There was a significant difference between the two groups in self-reported alcohol consumption both before and during pregnancy and in numbers of women who decreased alcohol consumption once the diagnosis of pregnancy was known to them.

Conclusions: There is the potential to identify prospectively women presenting for antenatal care who are heavy drinkers and risk FAS in their infants, using the self-reported information about alcohol intake already being collected by our service; such women may then be offered specific interventions to try to reduce alcohol consumption in pregnancy.

Key words: alcohol, antenatal, fetus, pregnancy, prenatal.

Fetal alcohol syndrome (FAS) is the term first used in 1973 to describe a set of physical, developmental and central nervous system defects in children associated with chronic maternal alcohol consumption during pregnancy.[1] The features necessary for a clinical diagnosis of FAS are growth restriction before and/or after birth, morphological abnormalities (short palpebral fissures, flat mid-face, short nose, indistinct philtrum and thin upper lips) and central nervous system deficits (intellectual impairment, microcephaly, behavioural problems, hyperactivity, impulsivity and inattention).[2,3] Heavy alcohol use in early pregnancy has a strong association with the neurological malformations and facial characteristics of FAS, whereas heavy use in later pregnancy is associated with cognitive and behavioural problems and retarded growth.[4,5] There is growing evidence associating lesser levels of alcohol use in pregnancy with a spectrum of effects in offspring; however, well-defined criteria for these have yet to be defined.[2–6]

FAS and alcohol-related birth defects represent an entirely preventable cause of permanent physical, mental and developmental deficiencies among children.[7–9] In Australia, an incidence of 0.02 per 1000 live births in non-Indigenous children and 2.76 per 1000 live births in Indigenous children was noted in 2000 in Western Australia.[9] A 2003 study from the

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Northern Territory found a prevalence of 1.7 per 1000 Indigenous live births for FAS and 4.7 per 1000 Indigenous live births for lesser disorders.[10] The Australian Paediatric Surveillance Unit conducted the first national prospective study from 2001 to 2003, identifying a national birth prevalence of 0.06 per 1000 live births.[11] No national Indigenous prevalence was reported, however, 65% of mothers in this study were identified as Indigenous.[1,2,4,5,7]

A prevalence of 1.5% for 'fetal alcohol spectrum disorders' was noted among Aboriginal children in communities in Far North Queensland reviewed by the Paediatric Outreach Service (POS) of Cairns Base Hospital (CBH) in 2001–2006.[12] These communities also receive antenatal care services from the CBH Flying Remote Obstetric and Gynaecological Service (FROGS) and the majority of births to women from these communities take place in CBH. As reported elsewhere, all women booking for antenatal care with our service are asked about alcohol intake prior to pregnancy and after the diagnosis of pregnancy has become known to them.[13] It was decided to review the case notes of all mothers of children diagnosed with FAS by the POS to determine whether maternal high-risk factors for FAS could be identified using our current antenatal care procedures, or whether tools shown to be useful elsewhere might be appropriate.[14,15] If women at risk can be identified early in pregnancy, it may be possible to alter behaviour to reduce alcohol consumption and hence reduce the incidence of FAS.

Methods

The database Medical Director of the POS was searched to identify all children with a diagnosis of FAS. Although the POS was established in 1994, electronic records were not introduced until 2001 so only cases who have been seen at least once since 2001 were included in the study (although the diagnosis may have been made prior to this). All children had the diagnosis of FAS made by a paediatrician from the POS. For each case, a control was found in the database matched for month of birth, sex, community, Indigenous status and *without* any congenital abnormalities or features that could be associated with FAS or fetal alcohol spectrum disorder. Controls were immediately excluded if they were seen only once, had a diagnosis of fetal alcohol effect (FAE), were a sibling (same natural mother) to a child with FAS or FAE, if they had already qualified as a control, and were a twin or born very preterm (≤ 26 weeks). Low birthweight, behavioural problems, speech problems, developmental delay or failure to thrive were insufficient reasons for exclusion as these characteristics are not unique to FASD. Search criteria were expanded to +/–12 months from the case birth date to identify suitable controls. Where several children matched all selection criteria, that with the closest birth date was selected. Where no matches were identified, the search was expanded to (in decreasing rank) different Indigenous status, opposite sex then closest community visited by the POS. Each of the two cases who were twins was matched with a singleton control.

A review of CBH maternal records was then conducted (for both cases and controls), noting mother's age, parity, self-reported alcohol and cigarette consumption both prepregnancy and after the diagnosis of pregnancy was known to the woman, antenatal and intrapartum complications and method of delivery. From the infants' charts, details of gestation at birth, birthweight, admission to the special care baby unit (SCBU), and perinatal complications were obtained. Visits were made to the relevant Indigenous communities as required to review the charts of children whose details in the database or CBH notes were incomplete/inadequate and to access the antenatal information for mothers in case this information was incomplete in the CBH charts; where a child was born outside the region, relevant information was obtained from the hospital of birth. Details of any later-born siblings of cases delivered in CBH were also recorded, including any evidence of fetal spectrum disorder short of FAS itself.

Self-reported alcohol consumption was classified as none, mild, moderate or heavy according to the criteria shown in Table 1, and consistent with findings reported from CBH in 2006.[13] These data were blinded as to case/control status and tested for consistency by a medical staff member of the Alcohol, Tobacco and Other Drug Use Service (ATODS) at CBH. Characteristics of mothers and children were compared using the paired *t*-test for continuous variables, Pearson χ^2 and the Wilcoxon signed rank tests for non-parametric variables. Permission for the study was granted by the Ethics Committee of CBH.

Results

There were 60 cases of FAS identified among the 3868 children in the POS; 59 were included for analysis as one case was over 18 years of age. There were 54 mothers of cases and 56 mothers of controls (some women had more than one child among both cases and controls); 43 of the cases were born at CBH and only four outside the region, 45 controls were born in CBH and only three outside the region.

Table 1 Criteria for breakdown of alcohol use

Category	Alcohol range	Examples
None	0 mg/week	'None' 'No' 'Nil'
Mild	< 20 mg/week	'2 beers /week'; '4 cans occasionally socially'
Moderate	20–80 mg/week	'6 light at weekend'/'6 light/week'
Heavy	> 80 mg/week	'1 carton beer/day/week/weekend'; '12 pack Friday night'; '24 beers/week'; 'a lot, many bottles/cans every day'; heavy Friday or Saturday nights; 'drunk weekends'
Not stated	–	Nothing stated 'Social' 'Mild' 'Yes' 'Occasional'

Children were aged one to 17 years and 60% were male. There was no significant difference in Indigenous status with 58 cases and 53 controls identifying as Indigenous – the POS is largely confined to the Indigenous communities of the region.

Mothers of cases had a mean age at delivery of 28 years compared to 23 years for controls and cases were also of higher parity – 35 cases had a parity of ≥ 2 (59.3%) compared to 18 (30.5%) controls. The average gestational age at booking was 18 weeks for cases and 15 weeks for controls which was not statistically significant ($P = 0.072$). Only three (5.1%) case mothers and one (1.7%) control had no antenatal care at all; 29 (49.2%) of cases and 41 (69.5%) of controls attended eight or more antenatal visits in total. The nature and number of antenatal and delivery complications are recorded in Table 2. The most common pregnancy complications in both groups were intrauterine growth restriction (IUGR) as defined by the treating obstetrician, anaemia and active syphilis; there was a significant increase in the incidence of IUGR consistent with the diagnosis of FAS among case mothers compared to controls but no other significant differences. Among the delivery complications, incidences of fetal distress and of preterm births were significantly increased to case mothers.

Self-reported alcohol consumption during pregnancy is shown in Table 3. Alcohol-use information before pregnancy was available for 42 cases (71.2%) and 44 controls (74.6%). Information about alcohol use once the diagnosis of pregnancy was known was available for 45 case mothers (76.3%) and 47 control mothers (79.7%). Among the cases 32 women (76.2%) and among the controls 13 (29.5%) women self-reported as heavy drinkers before pregnancy; this difference was significant ($P = 0.003$). Heavy alcohol use once the diagnosis of pregnancy was known was reported by 31 cases (68.9%) and only eight controls (17%) – these results were also significant ($P = 0.000$). The number of controls reporting a decrease in alcohol use once the diagnosis of pregnancy was known to them was significant ($P = 0.001$), whereas among the cases there was no such significant decrease.

There were similar results for cigarette smoking as shown in Table 3. Smoking information before pregnancy was available for 40 (67.8%) of case and 53 (89.8%) of control mothers. Information on smoking during pregnancy was available for 43 (72.3%) cases and 57 (96.6%) controls. Before pregnancy, 19 (47.5%) case mothers and 16 (30.2%) control mothers smoked > 20 cigarettes per day. During pregnancy, almost half the case mothers (46.5%) smoked > 20 cigarettes per day compared to seven (12.3%) of control mothers. There was a highly significant decline in the rates of smoking among controls once the diagnosis of pregnancy was known; this did not occur among the cases. Trends of alcohol and smoking use are shown in Figure 1.

Delivery gestations ranged from 30 to 41 weeks among cases and 27–43 weeks among controls. Two cases from (separate) sets of twins (and their controls) were excluded from analysis of delivery gestation and subsequent birth measurements because of known differences in the normal ranges compared to singleton pregnancies. Of the remaining 57 pairs, 16 (28.1%) of cases were preterm compared to only six (10.5%) controls. The mean delivery gestation of cases was 37 weeks, compared to 39 weeks for controls representing a highly significant difference in gestational age ($P = 0.00$).

To determine what known variables could account for the difference between the gestational ages at delivery,

Table 2 Pregnancy and delivery complications

	Case mothers		Control mothers		Significance		
Complication	Frequency	%	Frequency	%	χ^2 value	*P*-value	d.f.
Pregnancy complications							
Any pregnancy complication	45	76.3	24	40.7	16.47	0.00	1
GDM	5	8.5	3	5.1	0.54	0.46	1
Anaemia	8	13.6	7	11.9	0.08	0.78	1
Active syphilis	8	13.6	3	5.1	2.51	0.11	1
PIH or PE	4	6.8	4	6.8	0.00	1.00	1
APH	0	0.0	2	3.4	2.03	0.15	1
Multiple pregnancy	2	3.4	0	0.0	2.03	0.15	1
TPL	1	1.7	2	3.4	0.34	0.56	1
PROM	4	6.8	1	1.7	1.88	0.17	1
IUGR	27	45.8	5	8.5	20.75	0.00	1
Appendicitis	1	1.7	0	0.0	1.01	0.32	1
Other infection	2	3.4	0	0.0	2.03	0.15	1
Undiagnosed pregnancy	1	1.7	0	0.0	1.01	0.32	1
Unknown	0	0.0	0	0.0	na	na	na
Delivery complications							
Any delivery complication	35	59.3	15	25.4	13.38	0.00	1
Preterm delivery	18	30.5	5	8.5	9.13	0.00	1
Fetal distress	16	27.1	4	6.8	8.67	0.00	1
Meconium liquor	13	22.0	8	13.6	1.45	0.23	1
Resuscitation	6	10.2	5	8.5	0.10	0.75	1
Twin pregnancy	2	3.4	0	0.0	2.03	0.15	1
Chorioamnionitis	2	3.4	0	0.0	2.03	0.15	1
Complication not stated	1	1.7	0	0.0	1.01	0.32	1

APH, antepartum haemorrhage; GDM, gestational diabetes mellitus; na, not applicable; PE, pre-eclampsia; PIH, pregnancy-induced hypertension; PROM, premature rupture of membranes (defined as rupture for > 24 h before delivery); TPL, threatened preterm labour; IUGR, intrauterine growth restriction (that was recorded in the mothers chart during pregnancy); other infection (significant infection requiring hospitalisation; these were pneumonia and pyelonephritis); resuscitation (only that including bag/mask ventilation and/or intubation).

linear regression analysis against maternal age, smoking and alcohol was performed. Only maternal age and smoking were correlated to gestational age at delivery not alcohol, as shown in Table 4.

The birthweights of cases were on average 720 g lighter than those of controls ($P < 0.01$, $n = 57$). The range of birthweights was 1080–3480 g for cases and 1200–4600 g for controls. The majority of cases (32, 56.1%) were born with low birthweights (1500–2499 g) compared to the controls (10, 17.5%). Removing the confounding influence of gestational age (adjusted birthweight) did not alter the significant difference between birthweights in the groups. Low birthweights were strongly correlated to both alcohol and smoking in both groups as shown in Table 4. There was a highly significant difference between the two groups in rates of admission to SCBU or to neonatal intensive care unit (NICU). Thirty-four cases (62.7%) were admitted to SCBU and two (3.4%) to NICU, compared to 17 (27.1%) controls admitted to SCBU. Among the cases, there were eight diagnosed in the neonatal period with a congenital anomaly (as recorded on the maternal and child perinatal data sheet) but only two of these had a diagnosis of FAS despite all infants being assessed by paediatric staff. Among the controls, there were four diagnoses of congenital anomalies in the neonatal period.

One of the 54 case mothers had three children diagnosed with FAS, all included in the study as cases, and three mothers had two FAS-affected children. Of the remaining case mothers, 21 gave birth in CBH to 29 children *without* FAS following the birth of the FAS-affected child, however, among these 29 three children were noted as showing signs of 'fetal spectrum disorder' including facial features, IUGR and retarded intellectual development. For all

Table 3 Self-reported alcohol consumption and smoking rates

		Case mothers		Control mothers		Between groups	
		n	%	*n*	%	*N*	*P*
Before pregnancy	None	5	11.9	14	31.8		
Alcohol	Mild	1	2.4	7	15.9		
	Moderate	4	9.5	10	22.7	31	0.00
	Heavy	32	76.2	13	29.5		
	Total	42	100.0	44	100.0		
During pregnancy	None	10	22.2	30	63.8		
Alcohol	Mild	0	0.0	4	8.5		
	Moderate	4	8.9	5	10.6	36	0.00
	Heavy	31	68.9	8	17.0		
	Total	45	100.0	47	100.0		
Within groups	*N*	38		42			
	P	0.58		0.00			
Before pregnancy	None	3	7.5	15	28.3		
Smoking	≤ 10/day	11	27.5	16	30.2		
	11–20/day	7	17.5	6	11.3	36	0.00
	> 20/day	19	47.5	16	30.2		
	Total	40	100.0	53	100.0		
During pregnancy	None	3	7.0	27	47.4		
Smoking	≤ 10/day	12	27.9	18	31.6		
	11–20/day	8	18.6	5	8.8	42	0.00
	> 20/day	20	46.5	7	12.3		
	Total	43	100.0	57	100.0		
Within groups	*N*	38		53			
	P	0.08		0.00			

Table 4 Correlations between alcohol, smoking and mother's age for delivery gestation and adjusted birthweight

	Delivery gestation			Adjusted birthweight		
	r	*P*	*N*	*r*	*P*	*N*
Delivery gestation	1	–	114	–	–	–
Adjusted birthweight	–	–	–	1	–	114
Alcohol Yes/No	0.06	0.56	88	–0.38	**0.00**	88
Smoking Yes/No	–0.25	**0.01**	96	–0.45	**0.00**	96
Mother age at delivery	–0.22	**0.02**	114	–0.27	**0.00**	114
Heavy alcohol	–0.01	0.91	76	–0.45	**0.00**	76
Heavy smoking	–0.32	**0.02**	54	–0.44	**0.00**	54

Numbers in bold indicate significant figures.

three children, the mother had self-reported heavy alcohol intake during the relevant pregnancy.

Discussion

These 59 cases represent the most severe manifestations of maternal alcohol consumption on the fetus. FAS is a permanent disorder that significantly impacts on the child's future prospects of a healthy and fulfilling life, and places a heavy financial burden on health and social services. Only cases of FAS were included in our study because no clinically useful diagnostic criteria for fetal spectrum disorders exist; however, as FAS is reportedly less common than

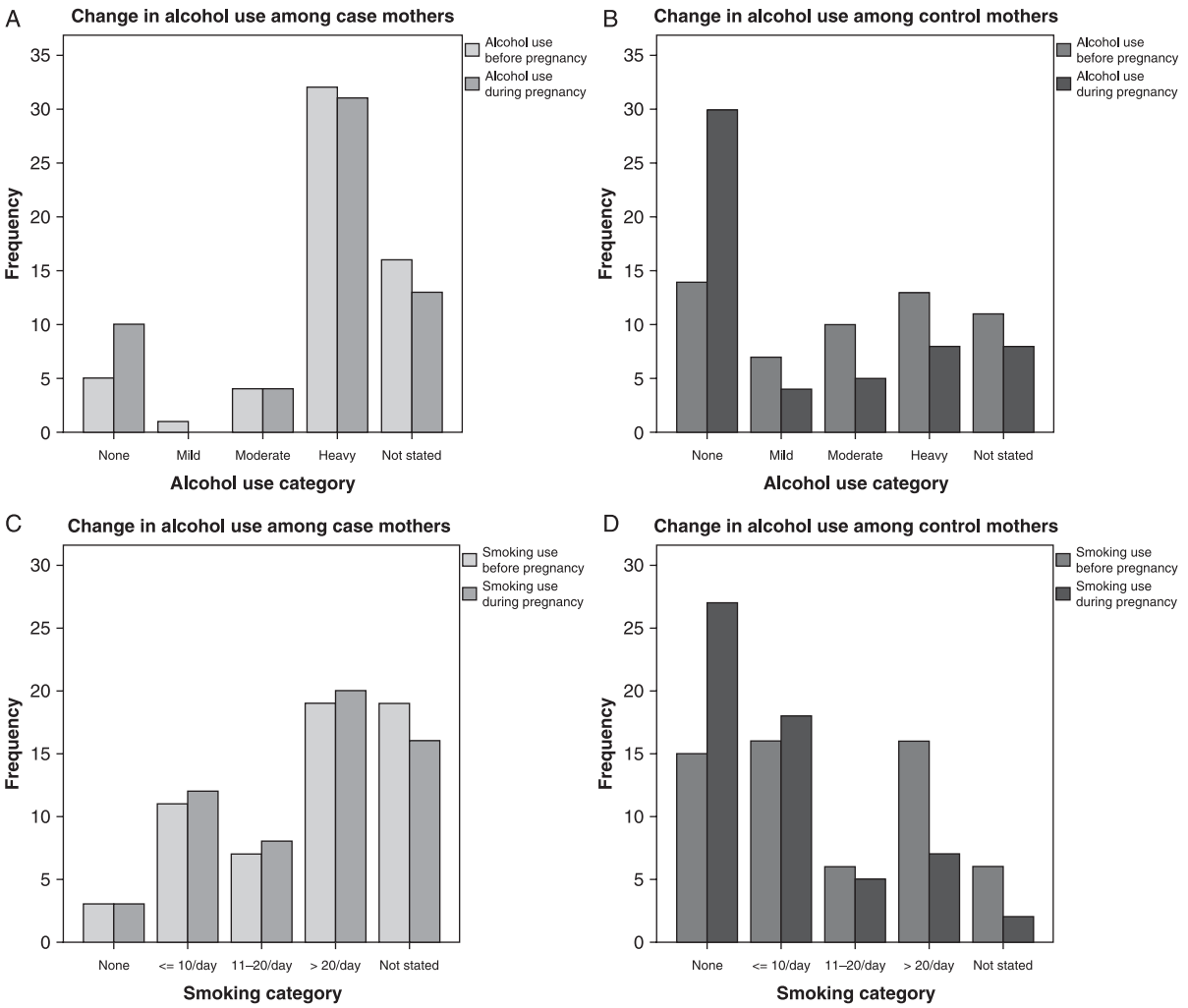

Figure 1 Change in patterns of reported alcohol consumption and smoking between the groups.

less-serious alcohol-related disorders it is likely that there are many more cases of fetal alcohol damage among the POS population, and this is confirmed by our limited observations of the later-born siblings of the cases. We believe that the delay noted in diagnosis of FAS among our cases following birth reflects the fact that developmental and intellectual criteria requiring time to assess are essential to the diagnosis; there may also be reluctance on the part of doctors to label a child with a diagnosis having the negative implications of FAS.

It is widely recognised that self-reported alcohol intake may be underestimated, and generally it is not considered a very reliable method of assessing alcohol intake during pregnancy.[13–16] Moreover, the lengthy timeframe for collection of information and the number of persons recording this information potentially limited the validity of the study. Prior to commencing the study we had anticipated assessing the use of tools such as AUDIT (Alcohol Use Disorder Identification Test, ten questions developed by the World Health Organization) or T-ACE (a similar four-part questionnaire, including one question on alcohol tolerance) to identify women at risk of giving birth to infants with FAS.[14,15]

However, it appears that among the population we care for, a majority of women *do* advise health professionals of 'heavy' alcohol use and that this information is recorded in some form. Thus it seems that the current method of non-judgmentally asking women at booking and later antenatal visits about their alcohol consumption can identify prospectively a large proportion of women at risk of the birth of a child with FAS; it is clear also that, as reported elsewhere, older women and women of higher parity are at greater risk, possibly because chronic alcoholism

and associated poor nutrition are also contributing factors.[7,8] Our findings suggest that standardising the questions already used and categorising the answers as shown in Table 1 will identify women who are heavy drinkers at an average of 18 weeks gestation. Since the morphological and neurological characteristics of FAS result from alcohol exposure in early pregnancy, these features will be unchanged unless women present for booking earlier in pregnancy and accept offered interventions, so strategies to encourage women to present earlier must also be adopted.

That some controls still had heavy intakes, and that most later-born siblings of cases were not diagnosed with FAS, suggests that other factors are involved in producing the full syndrome; this has been noted elsewhere and dietary and genetic factors, among others, have been postulated as important.[17–19] The ten cases with no alcohol consumption reported during pregnancy are most likely due to either a failure to disclose or because the abstinence occurred too late in pregnancy. The association between heavy alcohol consumption and heavy cigarette consumption has also been well reported and it is clear that future strategies must be directed at reducing both alcohol and cigarettes in this population.[7,8] It is also notable that our series included two sets of dizygotic twins among the cases, of whom only one in each set had a diagnosis of FAS.

Alcohol and substance abuse in the communities in which our study was conducted are the result of complex social, environmental, genetic and political interactions. Addressing these issues is not simple and researchers and health policy makers should not focus on alcohol as the sole factor in prevention strategies for FAS. Interventions must focus on the determinants of risk for FAS-affected pregnancies which include limited education, poor nutrition, societal tolerance of alcohol, low socioeconomic status, physical and sexual abuse and other substance abuse. Communities in Far North Queensland have themselves recognised the need to reduce supply and demand of alcohol by implementing alcohol bans and in some communities government restrictions on alcohol use now also apply. Several communities are currently the focus of efforts by the Cape York Institute (led by Mr Noel Pearson) to implement responsible alcohol use through the establishment of a Families' Responsibility Commission.[20,21] Community involvement and leadership is essential if alcohol consumption is to be decreased, thereby preventing FAS.

However, CBH provides a regular and consistent service to the women of these communities during pregnancy, specialist obstetricians, midwives, diabetes educators and ATODS educators all playing a role. Thus, we do appear well placed to try to identify prospectively women at risk of children with FAS and to offer interventions to decrease alcohol consumption in pregnancy. It has been suggested that early 2D ultrasound scanning and the provision of photos help women identify with their fetuses and change behaviour including decreasing alcohol intake.[22–24] The initiative of provided digital photos of the fetus to pregnant women in outreach clinics has been well received. We are now proposing to encourage women more actively to attend for early ultrasound as well as providing non-judgmental counselling aimed at reducing both alcohol and cigarette intake in pregnancy. It will be important to evaluate over time how the rates of FAS in these communities change; hopefully with the combined efforts of both community members and medical services it will be possible to reduce the incidence of this devastating condition in Far North Queensland.

References

1 Jones KL, Smith DW, Ulleland CN, Streissguth P. Pattern of malformation in offspring of chronic alcoholic mothers. *Lancet* 1973; **301**: 1267–1271.
2 O'Leary CM. Fetal alcohol syndrome: Diagnosis, epidemiology, and developmental outcomes. *J Paediatr Child Health* 2004; **40**: 2–7.
3 Hoyme HE, May PA, Kalberg WO *et al.* A practical clinical approach to diagnosis of fetal alcohol spectrum disorders: Clarification of the 1996 Institute of Medicine Criteria. *Pediatrics* 2005; **115**: 39–47.
4 American Academy of Pediatrics. Committee on Substance Abuse and Committee on Children with Disabilities. Fetal alcohol syndrome and alcohol-related neuro-developmental disorders. *Pediatrics* 2000; **106**: 358–361.
5 Riley EP, McGee CL. Fetal alcohol spectrum disorders: An overview with emphasis on changes in brain and behavior. *Exp Biol Med* 2005; **230**: 357–365.
6 Astley SJ, Clarren SK. Diagnosing the full spectrum of fetal alcohol-exposed individuals: Introducing the 4-digit diagnostic code. *Alcohol Alcohol* 2000; **35**: 400–410.
7 Floyd RL, O'Connor MJ, Sokol RJ *et al.* Recognition and prevention of fetal alcohol syndrome. *Obstet Gynecol* 2005; **106**: 1059–1064.
8 Abel EL, Hannigan JH. Maternal risk factors in fetal alcohol syndrome: Provocative and permissive influences. *Neurotoxicol Teratol* 1995; **17**: 445–462.
9 Bower C, Silva D, Henderson TR *et al.* Ascertainment of birth defects: The effect on completeness of adding a new source of data. *J Paediatr Child Health* 2000; **36**: 574–576.
10 Harris KR, Bucens IK. Prevalence of fetal alcohol

syndrome in the top end of the Northern Territory. *J Paediatr Child Health* 2003; **39**: 528–553.
11 Elliott EJ, Payne JM, Morris A, Haan E, Bower CA. Fetal alcohol syndrome: A prospective national surveillance study. *Arch Dis Child* 2007 [Epub ahead of print].
12 Rothstein J, Heazlewood R, Fraser M. Health of Aboriginal and Torres Strait Islander children in remote Far North Queensland: Findings of the paediatric outreach service. *Med J Aust* 2007; **186**: 519–521.
13 Rimmer C, de Costa C. A retrospective review of self-reported alcohol intake among women attending for antenatal care in Far North Queensland. *Aust N Z J Obstet Gynaecol* 2006; **46**: 229–233.
14 Russell M. New assessment tools for risk drinking during pregnancy: T-ACE, TWEAK, and others. (Special issue: Alcohol-related birth defects.) *Alcohol Health Res World* 1994; **18**: 55 (7).
15 Russell M, Martier SS, Sokol RJ *et al.* Detecting risk drinking during pregnancy: A comparison of four screening questionnaires. *Am J Public Health* 1996; **86**: 1435–1439.
16 Morrow-Tlucak M, Ernhart CB, Sokol RJ *et al.* Under-reporting of alcohol use in pregnancy: Relationship to alcohol problem history. *Alcohol Clin Exp Res* 1989; **13**: 399–401.
17 Streissguth AP, Bookstein FL, Barr HM *et al.* Risk factors for adverse life outcomes in fetal alcohol syndrome and fetal alcohol effects. *J Dev Behav Pediatr* 2004; **25**: 228–238.
18 Astley SJ, Bailey D, Talbot C, Clarren SK. Fetal alcohol syndrome (FAS) primary prevention through FAS diagnosis: I. Identification of high-risk birth mothers through the diagnosis of their children. *Alcohol Alcohol* 2000; **35**: 499–508.
19 Elliott EJ, Bower C. FAS in Australia: Fact or fiction? *J Paediatr Child Health* 2004; **40**: 8–10.
20 Cape York Institute for Policy and Leadership. *Welfare Reform, 2007*. [Accessed 1 August 2007.] Available from URL: www.cyi.org.au/welfare.aspx.
21 Pearson N. An abyss beyond the bottle. *The Weekend Australian*. 14–15 July 2007: 28.
22 Timor-Tritsch IE, Platt ID. Three dimensional ultrasound experience in obstetrics. *Curr Opin Obstet Gynecol* 2002; **14**: 569–575.
23 Sedgmen B, McMahon C, Cairns D *et al.* The impact of two-dimensional versus three-dimensional ultrasound exposure on maternal-fetal attachment and maternal health behavior in pregnancy. *Ultrasound Obstet Gynecol* 2006; **27**: 245–251.
24 Lapaire O, Alder J, Perkert R *et al.* Two dimensional versus three dimensional ultrasound in second and third trimester of pregnancy. *Arch Gynecol Obstet* 2007; **276**: 475–479.

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