

Fetal alcohol syndrome prevention in Washington State: evidence of success

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Summary

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Fetal alcohol syndrome (FAS) is a permanent birth defect syndrome caused by maternal consumption of alcohol during pregnancy. It is characterised by growth deficiency, central nervous system damage/dysfunction, and a unique cluster of minor facial anomalies. To assess the effectiveness of fetal alcohol syndrome prevention efforts, one must be able to estimate accurately the prevalence of fetal alcohol syndrome over time in population-based samples. With the establishment of the Washington State Fetal Alcohol Syndrome Diagnostic and Prevention Network of clinics, the development of the Fetal Alcohol Syndrome Facial Photographic Analysis Software, the creation of the Fetal Alcohol Spectrum Disorders (FASD) 4-Digit Diagnostic Code, the establishment of the Foster Care Fetal Alcohol Syndrome Screening Program, and the collection of Pregnancy Risk Assessment Management System data on maternal use of alcohol during pregnancy, the tools, methods and infrastructure for assessing the effectiveness of fetal alcohol syndrome primary prevention efforts in Washington State are in place. A cross-sectional study was conducted to determine whether the prevalence of fetal alcohol syndrome among children in a foster care population, born between 1993 and 1998, decreased with the documented decrease in prevalence of maternal use of alcohol during pregnancy from 1993 and 1998 in Washington State.

The prevalence of maternal drinking during pregnancy in Washington State declined significantly ($P < 0.001$) from 1993 to 1998 as did the prevalence of fetal alcohol syndrome among foster children born 1993–98 ($P < 0.03$). These observations support the likelihood that fetal alcohol syndrome prevention efforts in Washington State are working successfully.

Introduction

Fetal alcohol syndrome (FAS) is a permanent birth defect syndrome caused by maternal consumption of alcohol during pregnancy. It is characterised by growth deficiency, central nervous system (CNS) damage/dysfunction, and a unique cluster of minor facial anomalies.^{1,2} It is also the leading known cause of mental retardation/developmental disabilities in the western world³ and is entirely preventable. The prevalence of FAS is estimated to be 1–3 per 1000 live births⁴ in the US general population, but has been documented to be as high as 15 per 1000 in some high-risk populations.⁵

To prevent FAS, maternal alcohol consumption during pregnancy must be avoided. Prevention efforts

span a broad continuum from public health education and policy^{6–13} to direct intervention targeted to high-risk women.¹⁰ To assess the effectiveness of FAS prevention efforts, one must be able to estimate accurately, consistently and efficiently the prevalence of FAS over time in population-based samples. Accurate estimates of prevalence, in turn, require accurate diagnostic methods. With the establishment/development of the Washington State FAS Diagnostic and Prevention Network (FAS DPN) of clinics in 1993,^{14–17} the computerised FAS Facial Photographic Analysis Software in 1995,^{18–20} the FASD 4-Digit Diagnostic Code in 1997,^{2,21,22} the Foster Care FAS Screening Program in 1999,⁵ and ongoing collection of Pregnancy Risk Assessment Monitoring System (PRAMS) data by

Washington State since 1993,^{23–25} the methods and infrastructure for assessing the effectiveness of FAS primary prevention efforts through FAS screening, diagnosis and surveillance are now in place in Washington State.

The purpose of this cross-sectional study was to determine if the prevalence of FAS among children in a foster care population, born between 1993 and 1998, decreased with the documented decrease in prevalence of maternal use of alcohol during pregnancy from 1993 to 1998 in Washington State.

Methods

Prevention efforts in Washington State

Fetal alcohol spectrum disorder was identified at the University of Washington in 1970 by Ulleland and Smith¹ spawning two major clinical/research programmes, the FAS DPN¹⁴ and the Fetal Alcohol and Drug Unit,²⁶ which have made significant contributions over the past three decades to screening, diagnosis, education and prevention of FASD. Washington State prevention efforts have reflected the full continuum of strategies from public health education and training^{6,8,11,12,27} to direct intervention with high-risk women.^{28–30} Although prevention efforts have been ongoing since the early 1970s, a substantial increase in effort began in 1992 with the implementation of FAS prevention projects sponsored by the Centers for Disease Control,^{16,17,31,32} establishment of the Parent–Child Assistance Program,²⁹ and a legislative mandate in 1995³³ for statewide expansion of the FAS DPN and establishment of the FAS Interagency Work Group to ensure coordination of efforts across key state agencies including family advocacy groups.³⁴

Surveillance of maternal drinking during pregnancy in Washington State

PRAMS is a CDC-sponsored, ongoing, population-based surveillance system designed to monitor self-reported maternal behaviours that occur before, during and after pregnancy.^{23–25} Washington State is one of 31 states participating in PRAMS and the Washington State Department of Health has collected PRAMS data since June 1993. Each month, 1 in 40 live births are randomly selected from the Washington State birth certificates. Over-sampling by race other than Caucasian is conducted to increase the reliability of estimates for these racial groups. The data are then weighted to

reflect the true racial profile of the state. At two to 6 months postpartum, the sampled mothers ($n = 2000$ /year) are sent an explanatory letter and a self-administered PRAMS questionnaire. Non-respondents are sent a second questionnaire by mail and then multiple attempts to follow up are conducted by telephone. In the first year of data collection (1993), data were collected over 9 rather than 12 months with a response rate of 61%. For all other years, data were collected over 12 months with an average response rate of 70%.

The PRAMS questionnaire currently includes 66 questions (52 core questions and 14 state-specific questions). The core questions address obstetric history and risk factors, maternal feelings about timing of pregnancy, maternal economic status, birth control, prenatal care, folic acid awareness, prenatal behaviours and experiences (cigarette smoking, alcohol use, psychosocial stress during the 12 months prior to delivery, and physical abuse before and during pregnancy), prenatal hospitalisation, labour and delivery, and infant health. Two core questions documenting alcohol use include: 'During the 3 months before you got pregnant, how many alcoholic drinks did you have in an average week?' and 'During the last 3 months of your pregnancy, how many alcoholic drinks did you have in an average week?' Reported use of alcohol 3 months prior to pregnancy is deemed the most accurate measure of early pregnancy use because women often do not know they are pregnant until the second month and typically do not change their drinking patterns until they know they are pregnant.^{35,36} A drink was defined as: one glass of wine, one wine cooler, one can or bottle of beer, one shot of liquor or one mixed drink. Women were asked to select from the following choices: I didn't drink then; 1–3 drinks per week, 4–6 drinks per week, 7–13 drinks per week, 14 or more drinks per week; I don't know.

To date, prevalence estimates for maternal use of alcohol 3 months prior to pregnancy and during the third trimester of pregnancy are available from 1993 to 1998 in Washington State from 12 388 women.^{23–25}

Prevalence of FAS in a Washington State foster care population

In 1999, the FAS DPN implemented FAS screening and diagnosis of all children entering the Foster Care Passport Program (FCPP) in King County, Washington using the FAS Facial Photographic Analysis Software^{5,19,20} and the FAS 4-Digit Diagnostic Code.^{2,21}

Details on the performance of the screening tool and the prevalence of FAS among the first 600 children screened have been published previously.⁵ Briefly, all children, who were legally dependent with the State of Washington and enrolled in the Region 4 FCPP on or after 1 March 1999 in King County, Washington were eligible to participate in the screening. To be enrolled in the FCPP, a child had to be: (a) legally supervised by the Department of Social and Family Services; (b) 0–12 years of age at the time of enrolment, but may remain in the programme after their 12th birthday; (c) dependent and (d) in out-of-home placement.

To screen for FAS, a photographer from the FAS DPN was sent to the home of the foster child to take standardised, digital facial photographs with an internal measure of scale placed in the photo. A child screened positive for FAS if they had all three of the following facial features: palpebral fissure lengths two or more standard deviations below the mean, a smooth philtrum (Likert rank 4 or 5 on the 5-point Lip-Philtrum Guide) and a thin upper lip (Likert rank 4 or 5 on the 5-point Lip-Philtrum Guide) (Fig. 1).^{18–21} This case-definition for the FAS facial phenotype was derived analytically by Astley and Clarren,¹⁸ matches the original 1979 definition by Smith,³⁷ and has been

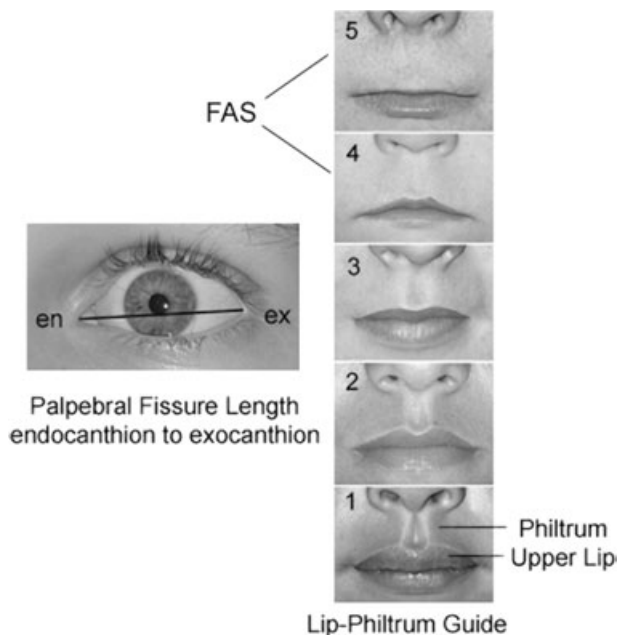


Figure 1. The three facial features of FAS include: (a) palpebral fissure lengths (the distance between the endocanthion and exocanthion landmarks) two or more S.D.s below the mean; (b) philtrum smoothness = Rank 4 or 5 on Lip-Philtrum Guide; and (c) upper lip thinness = Rank 4 or 5 on Lip-Philtrum Guide.

found to be highly sensitive and specific to FAS.¹⁸ Image analysis software²⁰ developed by the FAS DPN was used to measure the magnitude of expression of the FAS facial phenotype from the digital images. This procedure is described in detail by Astley and Clarren¹⁹ and demonstrated in a CD-ROM.²²

All children who screened positive for the FAS facial phenotype were scheduled for a diagnostic evaluation at the FAS DPN clinic where they received a comprehensive diagnostic evaluation and treatment plan by the interdisciplinary team using the 4-Digit Diagnostic Code.^{2,15,21} The 4-Digit Diagnostic Code is an objective, case-defined method for diagnosing the full spectrum of outcomes associated with prenatal alcohol exposure. It was developed to increase diagnostic accuracy and reproducibility. The screening activity was approved by the Human Research Review Boards of Washington State and the University of Washington. This ongoing screening activity has demonstrated that: (1) the prevalence of FAS in this foster care population is 10–15 per 1000, (2) the screening tool performs with 100% sensitivity and 99% specificity in this population-based sample, and (3) screening can be accomplished on a near complete (98%) sample of this population.⁵ The population screened ranged in age from 3 months to 15 years, was 48% female and had a racial distribution of 48% Caucasian, 32% African American and 12% Native American. The subset of five children diagnosed with FAS ranged in age from 1 to 11 years, three were female, two were Caucasian and three were African American.

Since Washington State PRAMS data documented a significant decline in maternal use of alcohol during pregnancy from 1993 to 1998, one might expect to see a decrease in the prevalence of FAS among children born in those years (1993–98). The Foster Care FAS Screening/Diagnostic Program provided a unique opportunity to assess change in prevalence of FAS over time in a high-risk population-based sample. The first step was to identify all children born from 1993 to 1998 ($n = 264$) among the first 600 children who participated in the screening programme. This was necessary because maternal use of alcohol during pregnancy from 1993 to 1998 could only affect children born in those years.

The proportion of children who were diagnosed with FAS within each of these six birth cohorts was computed. The prevalence of FAS was computed by birth cohort rather than by year of entry into foster care because the former would be far more sensitive in

documenting change. While a reduction in maternal drinking over time should result in a decrease in the prevalence of FAS among children entering foster care in each successive year, the decrease from year to year would be quite small because children entering foster care in any single year will range in age from birth up to 18 years old. For example, children entering foster care through the Foster Care Passport Program in the year 2000 will have been born across 12 different birth cohorts from 1988 to 2000. The prevalence of FAS among children entering foster care in 2000 would be influenced by the drinking patterns of their mothers from 1988 to 2000. Any reduction in drinking in the late 90s could be masked by the higher prevalence of drinking in the early 90s. Thus, to document the impact of maternal drinking on risk of FAS from year to year, the prevalence of FAS needs to be based on the year the child was born, not the year the child entered foster care.

Analysis

The chi-square test for linear trends was used to assess the change in prevalence of maternal drinking and change in prevalence of FAS from 1993 to 1998. Pearson and Spearman ρ correlation coefficients were computed to assess the correlation between prevalence of maternal drinking in early and late pregnancy and prevalence of FAS. Pearson and Spearman ρ were used when data were normally and not normally distributed respectively.

Results

Change in prevalence of alcohol use over time

Washington State PRAMS data show that the prevalence of maternal alcohol use 3 months prior to pregnancy and during the third trimester declined significantly (chi-square = 66.9, $P < 0.001$ and chi-square = 101.3, $P < 0.001$, respectively) from 1993 to 1998, exceeding the Healthy People 2010 objective of 6%³⁸ (Table 1, Fig. 2). This decline is most striking among the women reporting the highest levels of use (>14 drinks/week) in early pregnancy (chi-square = 64.5, $P < 0.0001$).

Change in prevalence of FAS over time

Of the first 600 children who participated in the foster care FAS Screening Program,⁵ 264 were born from 1993

Table 1. Decline in the prevalence of women reporting alcohol use 3 months prior to pregnancy and in the third trimester of pregnancy in Washington State from 1993 to 1998²³⁻²⁵

| Year | N | Average drinks per week | | | | | |
|---|------|-------------------------|------|------|------|------------------|-------------------|
| | | <1 | 1-3 | 4-6 | 7-13 | >14 | >0 |
| Three months before pregnancy ($n = 12\ 063$) | | | | | | | |
| 1993 | 1254 | 50.1 | 29.0 | 11.9 | 4.2 | 4.9 ^a | 51.5 ^a |
| 1994 | 2430 | 51.3 | 29.0 | 10.7 | 5.3 | 3.7 | 57.3 |
| 1995 | 1985 | 55.5 | 26.6 | 12.4 | 3.5 | 2.0 | 49.8 |
| 1996 | 2069 | 51.8 | 29.9 | 12.1 | 3.4 | 2.8 | 49.6 |
| 1997 | 2098 | 51.7 | 28.4 | 11.7 | 6.3 | 2.0 | 46.5 |
| 1998 | 2227 | 55.4 | 27.9 | 12.4 | 3.6 | 0.7 | 44.3 |
| Third trimester ($n = 12\ 198$) | | | | | | | |
| 1993 ^c | 1293 | 72.8 | 21.3 | 0.2 | 3.2 | 2.5 | 14.6 ^b |
| 1994 | 2430 | 75.0 | 20.1 | 2.0 | 2.8 | 0.1 | 7.8 |
| 1995 | 2017 | 79.6 | 17.5 | 2.5 | 0.1 | 0.4 | 8.2 |
| 1996 | 2105 | 83.7 | 12.8 | 3.4 | 0.0 | 0.1 | 8.3 |
| 1997 | 2116 | 88.2 | 8.3 | 3.2 | 0.0 | 0.3 | 6.2 |
| 1998 | 2237 | 80.6 | 18.3 | 0.9 | 0.1 | 0.2 | 3.9 |

^aThree months before pregnancy: test for linear trend from 1993 to 1998. Drinking >0 drinks/week: chi-square = 66.9, $P < 0.0001$. Drinking >14 drinks/week: chi-square = 64.5, $P < 0.0001$.

^bThird trimester of pregnancy: test for linear trend from 1993 to 1998. Drinking >0 drinks/week: chi-square = 101.3, $P \leq 0.001$. The Healthy People 2010 objective is to reduce the prevalence of drinking during pregnancy to 6%.³⁸

^cData were collected over 9 months in this first year with a response rate of 61%. For all other years, data were collected over 12 months with an average response rate of 70%.

to 1998. Five of these 264 children screened positive and were diagnosed with FAS.⁵ A diagnosis of FAS required all of the following to be present: height and weight at or below the 10th percentile; all three FAS facial features as described in Fig. 1; evidence of significant structural, neurological or functional CNS damage and a confirmed history of prenatal alcohol exposure.² The prevalence of FAS among these 264 foster children declined significantly (chi-square = 4.7, $P = 0.03$) across each birth cohort from 1993 to 1998. (Table 2, Fig. 2).

Correlation between maternal drinking and FAS prevalence

The prevalence of alcohol use during pregnancy from 1993 to 1998 in this statewide sample of women was compared with the prevalence of FAS among children born between 1993 and 1998 in this King County foster care population. Despite the cross-sectional nature

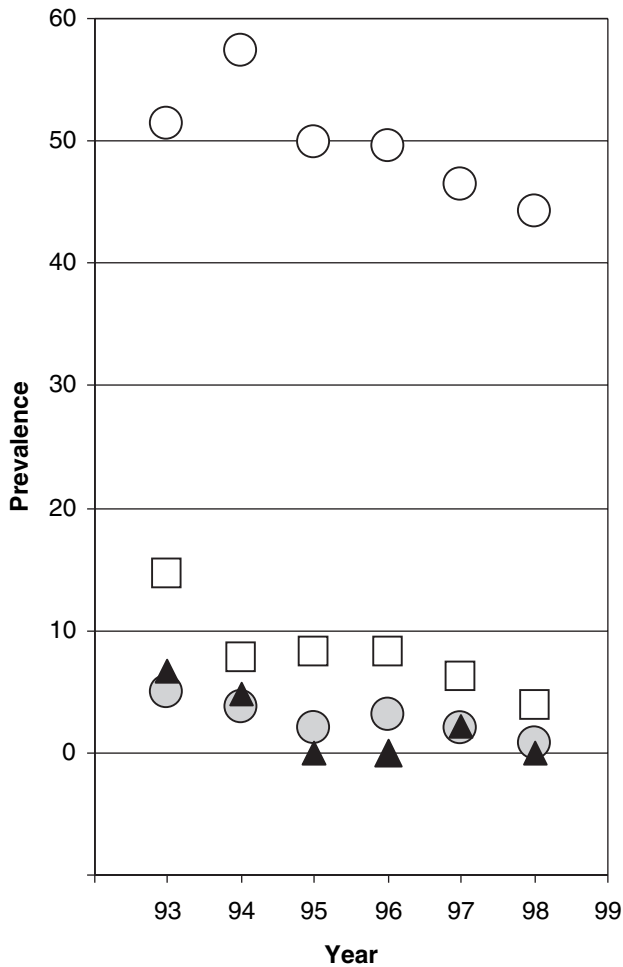


Figure 2. Decline in the prevalence of alcohol use by women in Washington State. (○) Any level of alcohol use 3 months prior to pregnancy; (□) Any level of alcohol use in the third trimester of pregnancy; (●) Heavy alcohol use (>14 drinks/week) 3 months prior to pregnancy from 1993 to 1998;²³⁻²⁵ (▲) Prevalence of FAS among children in the King County Foster Care Passport Program born from 1993 to 1998.⁵

of these data, remarkably strong correlations were observed that increased in strength as the risk in timing and level of maternal alcohol use increased. When the analysis focused on the timing of alcohol use, the decline in FAS was more strongly correlated with the decline in maternal alcohol use in early pregnancy (Pearson Correlation Coefficient = 0.61, $P = 0.200$) than in late pregnancy (Spearman's $\rho = 0.33$, $P = 0.52$). This stronger correlation between FAS and early pregnancy exposure would be expected since (1) the FAS facial features are caused by alcohol exposure in the first 8 weeks of pregnancy and (2) the PRAMS question 'How much were you drinking in the third trimester?' would fail to identify a subset of women at high risk

for bearing children with FAS (namely, women who did not drink in the third trimester, but did drink heavily in early pregnancy before they knew they were pregnant). When the analysis focused on both timing and level of alcohol use, the decline in FAS was most strongly and significantly correlated with the decline in prevalence of women reporting the highest-risk drinking pattern (>14 drinks/week in early pregnancy). The Pearson Correlation Coefficient was 0.86 ($P = 0.028$). The power to detect statistically significant correlations was limited by the number of years data were available ($n = 6$). This study had 80% power to detect correlation coefficients ≥ 0.85 , at a two-tailed alpha level of 0.05.

Discussion

PRAMS data document a significant decline in the prevalence of maternal alcohol use during pregnancy from 1993 to 1998. The FAS DPN Screening/Diagnostic Program documents a significant decline in the prevalence of FAS among foster children born between 1993 and 1998. These observations support the likelihood that FAS prevention efforts in Washington State are working successfully. While this study was not designed to determine which prevention efforts were most effective, the prevention literature strongly supports that a comprehensive approach that utilises the entire spectrum of effort from public health education to targeted intervention has the greatest impact.^{10,11}

Although the declines in maternal drinking and FAS observed in this study were statistically significant, these declines are based on very subtle annual reductions (each declining by just a few percentage points per year). Detection of subtle changes in FAS prevalence requires use of diagnostic and screening methods that are specifically case-defined so that they can be

Table 2. Decline in the prevalence of FAS among 264 children in the King County Foster Care Passport Program born from 1993 to 1998

| Birth cohort | Number screened | FAS prevalence ^a | FAS <i>n</i> |
|--------------|-----------------|-----------------------------|--------------|
| 1993 | 30 | 6.67% | 2 |
| 1994 | 42 | 4.76% | 2 |
| 1995 | 39 | 0.00% | 0 |
| 1996 | 45 | 0.00% | 0 |
| 1997 | 46 | 2.17% | 1 |
| 1998 | 62 | 0.00% | 0 |

^aChi-square test for linear trend from 1993 to 1998 = 4.7, $P = 0.03$.

accurately and reproducibly administered from year to year on a complete population-based sample. Typically, FAS surveillance activities to date have been population-based and rely on records-based data abstraction to generate prevalence estimates.³⁹⁻⁴² The FAS DPN screening, diagnostic and surveillance programme is unique because it integrates all three of these activities, capitalising on the inherent strengths of each. For example, the FAS DPN starts by screening all children in a foster care population with a FAS screening tool that performs with 100% sensitivity and 99.8% specificity. The FAS screening tool performs with the accuracy of a diagnostic tool because it is the diagnostic tool used to measure the FAS facial phenotype. The screening activity doubles as a highly effective surveillance activity because near-complete case ascertainment (98%) is achieved. High ascertainment is accomplished because the photographic screening tool can be practically administered and is targeted to a population that is (1) specifically defined, (2) readily tracked over time, and (3) motivated to participate because of the direct benefits. All children who screen positive receive a comprehensive diagnostic evaluation by an interdisciplinary team using the *same* diagnostic method (the 4-Digit Diagnostic Code).

At the time of diagnosis, the team reviews all medical, social service and educational records available on the child in addition to obtaining measures of growth, face, brain function and alcohol exposure through caregiver interview and direct clinical evaluation of the child. In contrast, a typical surveillance programme will rely on the passive chance that: (1) a child utilises the health care system; (2) a FAS diagnostic evaluation is conducted; (3) the surveillance programme obtains a copy of the relevant medical record(s); and (4) the record contains both accurate and sufficient information to confirm the child does or does not have FAS.

In a records-based approach to surveillance, all children will not have an equal chance of being correctly identified because the diagnostic methods used will vary from child to child and the accuracy of the data in the medical record cannot be confirmed as the child is never seen directly by the surveillance programme. This will limit the ability of the programme to track subtle changes in prevalence over time. In addition, surveillance programmes that rely on record abstraction often have to rely on proxy case definitions of FAS rather than the true clinical definition because the data needed to confirm a diagnosis of FAS is often not present in the medical or psychological records.³⁹ The

inadequacy and variability of medical record documentation of FAS is well documented in the literature⁴⁰ and has been our experience in the FAS DPN clinic for the past 12 years.

Of the first 1390 patients evaluated in the FAS DPN clinic, 91 received a diagnosis of FAS using the 4-Digit Diagnostic Code. Although 60% of these 91 patients were over the age of 5 years at the time of diagnosis, only 8 had records documenting a previous evaluation and diagnosis of FAS. These 8 patients ranged in age from 1 to 24 years old. Of the 1299 patients that did not receive a diagnosis of FAS in the FAS DPN clinic, 24 had previously been diagnosed with FAS, but neither their medical records nor direct evaluations could substantiate the diagnosis. Similar observations were made among the children enrolled in the Foster Care FAS Screening/Diagnostic Program. All 10 children who screened positive for FAS among the first 600 children enrolled had no documentation of risk of FAS in their medical, school or psychological records even though the oldest was 11 years old and most had several inches of records. All 10 children would have been missed by a records-based approach to surveillance.

Another important and unique strength of the FAS DPN approach to surveillance is the potential for direct benefit to the child and their family. Typically, surveillance programmes have no direct contact with subjects and thus, provide no direct benefit to them. In contrast, the FAS DPN program, through its initial screening and diagnostic phases, provides children who screen positive with the benefits of an accurate diagnosis and intervention plan. This can lead to improved access to services, more appropriate foster/adoptive placements and prevention of secondary disabilities.⁵ The potential for direct benefit also serves as a powerful motivator for participation as demonstrated by the 98% participation rate. Maintaining a high participation rate depends in large part on the families' experiences with the screening programme. It is imperative that false-positive screening outcomes are minimised. This is achieved with the FAS Facial Photographic Analysis Software screening tool because the FAS facial phenotype is highly sensitive and specific to FAS.^{5,18} Growth deficiency and cognitive/behavioural dysfunction are not specific to FAS. Screening children positive for FAS, who do not have FAS, not only costs time and money to conduct unnecessary diagnostic evaluations, but more importantly, can come at a high emotional cost to families, especially to the birth mothers.

Although the FAS DPN approach to tracking the prevalence of FAS over time has many strengths, it is important to consider what factors may affect the validity of the results presented here. Although the observed decline in FAS was statistically significant, it is important to note that the prevalence estimates within each birth cohort are limited by the small sample sizes. Two factors, however, support the validity and stability of these interim results. First, the prevalence estimates are based on near complete (98%) ascertainment of the eligible population and thus it is highly unlikely that any FAS cases were missed. Secondly, when we look forward in time to all 908 children screened to date in this ongoing screening programme, a significant decline in the prevalence of children screening positive for FAS continues to be observed across the subset of 383 children born between 1993 and 1998 (chi-square = 4.03, $P = 0.04$). For comparison, the CDC also recognised the impact of small sample sizes on the precision of prevalence estimates, thus they required a minimum of 30 respondents and a response rate of >70% before prevalence estimates could be reported from the PRAMS data.²³ These criteria were met or exceeded in this FAS prevalence study. An obvious solution to small sample sizes is to expand the screening statewide and discussions are underway to do that.

One of the key goals of surveillance is to assess the effectiveness of primary prevention. This approach to tracking the prevalence of FAS over time in a high-risk foster care population offers an interesting alternative to tracking the prevalence of FAS across an entire state population. If statewide prevention efforts and statewide reduction in maternal alcohol use are effectively reducing the prevalence of FAS in this foster care population, it would be difficult to argue that similar reductions are not also being realised across the entire general population. The same can be said for the impact of prevention efforts on the full spectrum of disorders caused by prenatal alcohol exposure. If maternal drinking during pregnancy is reduced, the full spectrum of disorders caused by that drinking will be reduced, not just the disorder called FAS.

Although there is tremendous merit and value in tracking the prevalence of individuals damaged by prenatal alcohol exposure who do not meet the diagnostic criteria for FAS, an accurate and valid screening tool to achieve this does not currently exist. Until such time, reduction in maternal drinking during pregnancy and reduction in the prevalence of FAS in foster care can serve as valid proxy measures for reduction

of the full spectrum of disorders associated with prenatal alcohol exposure across the general population.

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References

- 1 Jones KL, Smith DW, Ulleland CN, Streissguth AP. Pattern of malformation in offspring of chronic alcoholic mothers. *Lancet* 1973; **1**:1267–1271.
- 2 Astley SJ, Clarren SK. Diagnosing the full spectrum of fetal alcohol exposed individuals: introducing the 4-Digit Diagnostic Code. *Alcohol and Alcoholism* 2000; **35**:400–410.
- 3 Abel EL, Sokol RJ. Incidence of fetal alcohol syndrome and economic impact of FAS-related anomalies. *Drug and Alcohol Dependence* 1987; **19**:51–70.
- 4 National Institute on Alcohol Abuse and Alcoholism. *Seventh Special Report to the U.S. Congress on Alcohol and Health*. DHHS publication No. ADM (90-1656). Washington, DC: US Department of Health and Human Services, 1990.
- 5 Astley SJ, Stachowiak J, Clarren SK, Clausen C. Application of the FAS facial photographic screening tool in a foster care population. *Journal of Pediatrics* 2002; **141**:712–717.
- 6 *Surgeon General's Advisory on Alcohol and Pregnancy*. FDA Drug Bulletin, 11(2). Rockville, Maryland: Department of Health and Human Services, 1981.
- 7 *Alcohol Beverage Labeling Act of 1988*. Public Law 100-690, 100th Congress, 2nd session, November 18, 1988.
- 8 National Institute on Alcohol Abuse and Alcoholism. *Program Strategies for Preventing Fetal Alcohol Syndrome and Alcohol-Related Birth Defects*. DHHS Publication No. (ADM) 87-1482. Washington, DC: Superintendent of Documents, US Government Printing Office, 1987.
- 9 Stratton K, Howe C, Battaglia F. *Fetal Alcohol Syndrome: Diagnosis, Epidemiology, Prevention, and Treatment*. Washington, DC: National Academy Press, 1996.
- 10 Hankin JR. Fetal alcohol syndrome prevention research. *Alcohol Research and Health* 2002; **26**:58–65.
- 11 Hankin JR. FAS prevention strategies: passive and active measures. *Alcohol Health and Research World* 1994; **18**:62–66.
- 12 Greenfield TK, Kaskutas LA. Five years exposure to the alcohol warning label messages and their impacts: evidence from diffusion analysis. *Applied Behavioral Science Review* 1998; **6**:39–68.
- 13 Prugh T. Point-of-purchase health warning notices. *Alcohol Health and Research World* 1986; **10**:36.
- 14 Clarren SK, Astley SJ. Development of the FAS Diagnostic and Prevention Network in Washington State. In: *The Challenge of Fetal Alcohol Syndrome: Overcoming Secondary*

- Disabilities*. Editors: Streissguth A, Kanter J. Seattle, WA: University of Washington Press, 1997; pp. 40–51.
- 15 Clarren SK, Carmichael-Olson H, Clarren SGB, Astley SJ. A child with fetal alcohol syndrome: the interdisciplinary team diagnostic process. In: *Handbook of Clinical Assessment for Young Children with Developmental Disabilities*. Editor: Guralnick MJ. Baltimore, MD: Paul H. Brookes, 2000; pp. 307–326.
 - 16 Astley SJ, Bailey D, Talbot C, Clarren SK. FAS primary prevention through FAS diagnosis: Part I. Identification of high-risk birth mothers through diagnosis of their children. *Alcohol and Alcoholism* 2000; **35**:499–508.
 - 17 Astley SJ, Bailey D, Talbot C, Clarren SK. FAS primary prevention through FAS diagnosis: Part II. A comprehensive profile of 80 birth mothers of children with FAS. *Alcohol and Alcoholism* 2000; **35**:509–519.
 - 18 Astley S, Clarren SK. A case definition and photographic screening tool for the facial phenotype of fetal alcohol syndrome. *Journal of Pediatrics* 1996; **129**:33–41.
 - 19 Astley SJ, Clarren SK. Measuring the facial phenotype of individuals with prenatal alcohol exposure: correlations with brain dysfunction. *Alcohol and Alcoholism* 2001; **36**:147–159.
 - 20 Astley SJ. *Fetal Alcohol Syndrome Facial Photographic Analysis Software*, Release 1.0.0. Seattle, WA: University of Washington, 2002.
 - 21 Astley SJ. *Diagnostic Guide for Fetal Alcohol Spectrum Disorders: the 4-Digit Diagnostic Code*, 3rd edn. Seattle, WA: University of Washington Publication Services, 2004.
 - 22 Astley SJ, Clarren SK, Gratzner M, Orkand A, Astion M. *Fetal Alcohol Syndrome Tutor™ Medical Training Software CD-ROM*. Wilkes Barre, PA: March of Dimes Fulfillment Center, 1999.
 - 23 Lipscomb LE, Johnson CH, Morrow B, Gilbert BC, Ahluwalia IB, Beck LF, et al. *PRAMS 1998 Surveillance Report*. Atlanta: Division of Reproductive Health, National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention, 2000.
 - 24 *Pregnancy Risk Assessment Monitoring System (PRAMS) Surveillance Report: 1993–1994*. Olympia, WA: Maternal and Child Health Assessment Section, Community and Family Health, Washington State Department of Health, 1996.
 - 25 Eaglin ME, Robbins JM, Zaichkin J, Pilkey D. *1996–1998 Washington State Pregnancy Risk Assessment Monitoring System (PRAMS) Surveillance Report, Volume II*. Olympia, WA: Maternal and Child Health Assessment Section, Community and Family Health, Washington State Department of Health, 2002.
 - 26 Streissguth AP, Martin DC, Martin JC, Barr HM. The Seattle longitudinal prospective study on alcohol and pregnancy. *Neurobehavioral Toxicology and Teratology* 1981; **3**:223–233.
 - 27 LaDue R, Hartness C. *Journey Through the Healing Circle*. Olympia, WA, USA: Washington State Department of Social & Health Services, 2000.
 - 28 Little RE, Streissguth AP, Guzinski GM. Prevention of fetal alcohol syndrome: a model program. *Alcoholism, Clinical and Experimental Research* 1980; **4**:185–198.
 - 29 Grant TM, Ernst CC, Streissguth AP. An intervention with high-risk mothers who abuse alcohol and drugs: the Seattle advocacy model. *American Journal of Public Health* 1996; **86**:1816–1817.
 - 30 Albert DH. *Tobacco, Alcohol, and Other Drug Abuse Trends in Washington State, 2002 Report*. Olympia, WA, USA: Division of Alcohol and Substance Abuse, Department of Social and Health Services, September 2002.
 - 31 Astley SJ, Clarren SK, Quinby R, Lair C. Academic/public health collaboration leads to establishment of FAS Clinical Network, diagnostic guide, screening tool, and screening programs. Association of Schools of Public Health, 14th Annual National Preventive Medicine Meeting, Atlanta, GA, March 20–23, 1997.
 - 32 Olson HC, Gendler B, Kraegel P, Rosengren D, Clarren S, Astley S. A targeted approach to FAS prevention: the FAS DPN first bridges program. *Alcoholism, Clinical and Experimental Research*, 2002; **26**(Suppl):176A.
 - 33 *Fetal Alcohol Syndrome Act of 1995*. Substitute Senate Bill 5688, State of Washington, 54th Legislature, 1995.
 - 34 DeVries J, Waller A. The parental role in changing public policy. In: *The Challenge of Fetal Alcohol Syndrome: Overcoming Secondary Disabilities*. Editors: Streissguth A, Kanter J. Seattle, WA: University of Washington Press, 1998.
 - 35 Floyd RL, Decoufle P, Hungerford DW. Alcohol use prior to pregnancy recognition. *American Journal of Preventive Medicine* 1999; **17**:101–107.
 - 36 Day NL, Cottreau CM, Richardson GA. The epidemiology of alcohol, marijuana, and cocaine use among women of child-bearing age and pregnant women. *Clinical Obstetrics and Gynecology* 1993; **36**:232–245.
 - 37 Smith DW. The fetal alcohol syndrome. *Hospital Practice* 1979; **14**:121–128.
 - 38 US Department of Health and Human Services. *Healthy People 2010*. Washington, DC: US Department of Health, 2000.
 - 39 Hymbaugh K, Miller LA, Druschel CM, Podvin DW, Meaney FJ, Boyle CA, the FASSNet Team. A multiple source methodology for the surveillance of fetal alcohol syndrome – The Fetal Alcohol Syndrome Surveillance Network (FASSNet). *Teratology* 2002; **66**:S41–S49.
 - 40 Centers for Disease Control and Prevention. Linking multiple data sources in fetal alcohol syndrome surveillance-Alaska. *Morbidity and Mortality Weekly Report* 1993; **42**:312–314.
 - 41 Egeland GM, Perham-Hester KA, Gessner BD, Ingle D, Berner JE, Middaugh JP. Fetal alcohol syndrome in Alaska, 1977 through 1992: an administrative prevalence derived from multiple data sources. *American Journal of Public Health* 1988; **88**:781–786.
 - 42 Miller LA, Shaikh T, Stanton C, Montgomery A, Rickard R, Keefer S, et al. Surveillance for fetal alcohol syndrome in Colorado. *Public Health Reports* 1995; **110**:690–696.