2004-11-30

State of the evidence: Fetal Alcohol Spectrum Disorder (FASD) prevention: Final report

Basford, Lynn

Submitted to the Alberta Centre for Child, Family and Community Research (ACCFCR)

University of Lethbridge

ALBERTA CENTRE FOR CHILD, FAMILY & COMMUNITY RESEARCH

State of the Evidence: Fetal Alcohol Spectrum Disorder (FASD) Prevention

FINAL REPORT

November 30, 2004

Principal Investigator: Dean Lynn Basford

Co-Principal Investigators: Dr. Karran Thorpe and Dr. Robert William

Research Associate – Kelly Cardwell

THE TEAM

CORE TEAM MEMBERS

Lynn Basford, BA (Hons), MA, RGN, NDN, PWT, RNT, Nurse, Social Science
Karran Thorpe, RN, PhD, Nurse, Methodology
Rob Williams, PhD, C.Psych, Clinical Psychology
Judy Droessler, PhD, MLIS, Professional Librarian, Researcher
Sameer Deshpande, PhD, Social Marketing, Researcher
Alexandre Bureau, PhD, Epidemiology & Genetics, Researcher
Noella Piquette-Tomei, MEd, PhD, Education, Researcher
Kelly Cardwell, BA, Sociology, Research Associate

EXTENDED CORE TEAM MEMBERS

Sharlene Campbell, AAN, MN, FASD Program Project Co-ordinator, CHR
Vedna McGill, RSW, Crisis Intervention Specialist, CHR Family Health Home Visitation Programs
Cathy OHama, BEd, InfantPreschool Development Educator, CHR Family Health Home Visitation Programs
Bev West, BHSc, Research Assistant
Rebecca Many Grey Horses, BA, Coordinator, First Steps for Healthy Babies
Mike Basil, PhD, Social Marketing

STEERING COMMITTEE MEMBERS

Vern Jubber, MD, Vice President, CHR
Gary Roberts, PhD, Gary Roberts and Associates, Guidance and Advice
Robert Sutherland, PhD, Psychology and Neuroscience
Charlie Weaselhead, Director of Public Health, Blood Tribe Health

ACKNOWLEDGMENTS

We would like to acknowledge Ms. Alyssa Reed who served as Research Associate in the initial stages of this project. Further we would like to thank all members of the team who have contributed to the writing of the report and to our consultants for their advice.

Table of Contents

Chapter One

Introduction

Contextual Background

The medical condition known as Fetal Alcohol Spectrum Disorder (FASD), which encompasses the more commonly know conditions of Fetal Alcohol Syndrome (FAS) and Fetal Alcohol Effects (FAE), is the term given to children who shared a constellation of physical abnormalities, failure to thrive, and emerging neurobehavioural effects. In all these circumstances the common denominator was that their mothers were users of alcohol during pregnancy (Jones & Smith, 1973; Jones & Smith, 1976**;** Ullenland & Streissguth 1973; Smith, 1980; Sowell et al., 1996; Stratton et al., 1996).

The Frenchman, Lemoine (Lemoine et al., 1968) is the first known person in the modern era, to describe the pattern of physical deformities in eight children of alcoholic mothers. Five years later in the United States, Jones and Smith (1973) classified and codified these characteristic features as Fetal Alcohol Syndrome (FAS). FAS quickly became a universally accepted (i.e., medical) terminology. The common features of FAS first identified by Jones and Smith (1973) were:

- Developmental delay - social and motor performance related to mental, not chronological age.
- Microcephaly - with no significant catch up through early childhood.
- Pre-natal growth deficiency - length reduced proportionately more than weight.
- Post-natal growth deficiency - lack of catch up growth in spite of adequate nutrition.
- Short palpebral fissures.
- Maxillary hypoplasia with relative prognathism.
- Epicanthal folds.
- Joint anomalies.
- Cardiac anomaly.

While these features suggest a common aetiology, there was an absence of empirical knowledge and understanding relating to: (i) aetiology and epidemiology; (ii) the definitive diagnostic criteria; (iii) short- and long-term effects of FAS/FAE; (iv) how to engage in effective preventative models; and, (v) what were the best intervention strategies to use.

What is alarming is that three decades later these questions are an enigma to present day scientists. McAlhany and colleagues (2000) contend that, “the mechanisms of ethanol-induced brain damage in the developing nervous system are not well understood” (p. 209). Further, Wilson-Jones and Bass (2003) suggest that the diagnostic tools identified in 1979 remain relatively unchanged today. The latter point could indicate one of two things: that the diagnostic battery of tools are effective and warrant no change, or, that little attention has been given to designing instruments that can more accurately enable practitioners to make early diagnosis. Williams, Odaibo and McGee (1999) also indicate that the diagnostic ability of health professionals is found wanting, and, in some instances, there is a reluctance to make the diagnosis due to stereotyping and blaming women. This point is to some degree

understandable given the degree of adverse effect from maternal alcohol consumption across a continuum. On the one hand, there is a full expression of FASD, while on the other hand, there is the more benign and subtle behaviour of neurobehavioral effects (Loock, 1990).

The situation is indeed complex and has served to baffle scientists and health professionals in that the real incidence and prevalence is relatively unknown (Sampson et al., 1997). This situation is compounded further due to the absence of sophisticated databases that are standardized across the globe. Often, public policy and marketing messages are very different from government to government, and as a consequence, serve to confuse individuals and professional bodies alike with regard to the safety of alcohol consumption during conception, or, pregnancy, or, throughout the period of lactation. Nonetheless, it is generally acknowledged that there is a significant growth in the rate of FASD children being born on a global scale with some communities more at risk than others (Stratton et al., 1996). In this respect it is well recognized that the growing phenomena of children being born with either FASD or FAE is of major importance to public health and answers to the following questions are urgently required:

- What is the true aetiology and epidemiology of FASD/FAE?
- Is there a point in the gestational period that is more dangerous for maternal alcohol consumption?
- Does maternal alcohol consumption prior to conception have an effect?
- Is there a safe threshold level of alcohol consumption during pregnancy?
- Is there a co-morbid relationship between the consumption of alcohol, and, either smoking, or other drug combinations?
- Is there a genetic predisposition with alcohol intolerance?
- Do we need to consider intergenerational factors?
- Is there a co-relationship with paternal alcohol consumption at the point of conception?
- Is there a paternal affect due to the sperm quality affected by alcohol consumption?
- Is there a co-relationship with poverty, poor environment, living conditions, and nutritional intake?
- Is there any linkage with the non-involvement in pre conceptual, and/or ante-natal care?
- Is regular alcohol consumption and/or binge drinking a heightened risk factor?
- Can FAE be reversed or the effects minimized throughout the life span?
- Are there any known modifying effects?

These questions illustrate the complexity of the situation. Nonetheless, the scientific community, epidemiologists, policy makers, and health professionals require a strategic framework through which they can significantly make a difference to the incidence and prevalence of FASD/FAE. While statistics do vary, there is general consensus that FASD is one of the largest forms of mental disability and is nearly double the rate of Downs Syndrome and nearly five times that of Spina Bifida (Morrissette, 2001; Streissguth, 1997), and it is a wholly preventable tragedy. Guidance is, therefore, clearly needed with regard to the current state of the evidence and the directions for: (i) future research, (ii) best practice, (iii) education, and (iv) policy. The rigorous methodology discussed below was followed to assess the current state of knowledge surrounding FASD and provide this needed guidance.

Chapter Two

Project Methodology

The review of the literature on Fetal Alcohol Spectrum Disorder (FASD) Prevention was carried out by a multidisciplinary team using a Cochrane-style systematic review methodology. A key component of the review was the double-blind reading by two independent reviewers of all documents identified in a broad scope literature search. The objective of this methodological approach was to create a comprehensive context through which policy and practice can be informed and developed from a sound evidence base.

Data Sources

Scope of Literature Search

A comprehensive approach, encompassing a variety of study types, publication formats, and disciplines, was taken to identify current best evidence relevant to the primary, secondary, and tertiary prevention of FASD. International research published from 1970 forward was examined across a wide range of disciplines including the health sciences, addictions, epidemiology, psychology, social marketing, sociology, education, Native American studies, and social work. Reports of research were retrieved in a number of different publication formats that included journal articles, monographs, and "grey literature"[1] such as government publications, reports, theses and dissertations, and WWW documents. A variety of study types were retrieved, representing both quantitative and qualitative research related to prevention models, strategies, outcomes, and evaluation.

Emphasis was placed on randomized controlled trials where a comparison is made between groups or populations exposed to a prevention strategy and groups that were not. In addition, studies were sought that examined before and after drinking patterns of pregnant women who were exposed to a prevention strategy or intervention. Individual case reports and anecdotal accounts were excluded.

Literature Search Strategy

The literature search methodology generally followed that set forth by the Cochrane Collaboration, as outlined in the *Cochrane Reviewer's Handbook* (Alderson, Green, & Higgins, 2003). Relevant research was identified through searches of online databases, library catalogues, reference lists, key journals, personal contacts, and the World Wide Web.

Online Databases

The majority of the literature reviewed consisted of peer-reviewed journal articles retrieved through comprehensive searches of 26 electronic databases, both bibliographic and full-text. All searches were carried out by a Professional Librarian in the spring of 2004.

[1] *Grey literature* can be defined as "that which is produced on all levels of government, academics, business and industry in print and electronic formats, but which is not controlled by commercial publishers" (Fourth International Conference on Grey Literature, 1999).

Table 1 lists the specific databases searched and the search terms used. Search strategies necessarily varied with each database, taking into account variations in indexing and retrieval factors unique to each database. The overall strategy was to achieve a balance between recall (i.e., number of citations found or comprehensiveness) and precision (i.e., relevance of citations found), while aiming for as complete as possible retrieval of items related to FASD prevention.

Wherever possible, controlled vocabulary terms (i.e., standardized subject terms assigned by indexers) were used as search terms. Use of controlled vocabulary terms allows the use of a single word or phrase to retrieve items relating to a concept that can be described by several different words or phrases. At the same time, controlled vocabulary searches limit the number of "false drops," where search terms are used in other than the intended context. Whenever possible, terms were exploded to obtain narrower terms. Following controlled vocabulary searches, various combinations of keyword searches were used as a check to identify any relevant citations not retrieved by the controlled vocabulary search terms.

In databases lacking controlled vocabularies, key words and key word phrases were searched as words in titles, subjects, and abstracts. Where appropriate, Boolean search techniques and truncation were used (e.g., [alcohol* OR ethanol] AND [maternal or paternal or prenatal or mother or father or fetus or fetal]). In cases where database indexing was insufficiently detailed or reliable to narrow a search to prevention-related topics, a broad search on maternal drinking behavior and effects of alcohol consumption on the fetus was conducted. All searches were scanned initially by the searcher and items that were clearly irrelevant (false drops) were deleted. Where possible, database "alerts" were set up to ensure awareness of citations added to databases after initial searches were conducted.

Table 1
Databases Searched

DATABASE	PRODUCER	SEARCH TERMS[2]
Academic Search Premier	EBSCO Information Services	FETAL ALCOHOL SYNDROME, CHILDREN OF PRENATAL ALCOHOL ABUSE, alcohol related birth defects, alcohol related neurodevelopmental disorder*, arbd, arnd
ACP Journal Club	American College of Physicians, British Medical Journal Group	Fetal alcohol syndrome, fetal alcohol effects, fetal alcohol spectrum disorder, alcohol related birth defects, alcohol related neurodevelopmental disorder*, fas, fae, fasd, arbd, arnd, maternal, prenatal, pregnan*, fetus, fetal, alcohol*, ethanol, drinking, prevent*, women
Alcohol Studies Database	Rutgers University Center of Alcohol Studies	FETUS AND ALCOHOL, fetal alcohol syndrome, fetal alcohol effects, fetal alcohol spectrum disorder, alcohol related birth defects, alcohol related neurodevelopmental disorder*, fas, fasd, fae, prevent* arbd, arnd

[2] Controlled vocabulary terms are given in uppercase, keywords in lower case. Truncation is indicated by an asterisk (*).

DATABASE	PRODUCER	SEARCH TERMS[2]
Canadian Research Index		Fetal alcohol syndrome, fetal alcohol effects, fetal alcohol spectrum disorder, alcohol related birth defects, alcohol related neurodevelopmental disorder*, fas, fae, fasd, arbd, arnd
CINAHL: Cumulative Index to Nursing and Allied Health Literature	CINAHL Information Systems	FETAL ALCOHOL SYNDROME; ALCOHOL DRINKING; ALCOHOLISM; ETHANOL; PRENATAL EXPOSURE DELAYED EFFECTS; PREGNANCY; PREGNANCY TRIMESTER, FIRST; PREGNANCY TRIMESTER, SECOND; PREGNANCY TRIMESTER, THIRD; PREGNANCY OUTCOME; PREGNANCY, HIGH-RISK; PREGNANCY IN ADOLESCENCE; PREGNANCY COMPLICATIONS; FETUS; PREVENTION & CONTROL; alcohol related birth defect*, alcohol related neurodevelopmental disorder*, arbd, arnd, best practice*
Cochrane Central Register of Controlled Trials	Cochrane Collaboration	Fetal alcohol syndrome, fetal alcohol effects, fetal alcohol spectrum disorder, alcohol related birth defects, alcohol related neurodevelopmental disorder*, fas, fae, fasd, arbd, arnd, maternal, prenatal, pregnan*, fetus, fetal, alcohol*, ethanol, drinking, prevent*, women
Cochrane Database of Systematic Reviews	Cochrane Collaboration	Fetal alcohol syndrome, fetal alcohol effects, fetal alcohol spectrum disorder, alcohol related birth defects, alcohol related neurodevelopmental disorder*, fas, fae, fasd, arbd, arnd, maternal, prenatal, pregnan*, fetus, fetal, alcohol*, ethanol, drinking, prevent*, women
CRISP: Computer Retrieval of Information on Scientific Projects	Office of Extramural Research at the National Institutes of Health (US)	FETAL ALCOHOL SYNDROME; ALCOHOLISM/ALCOHOL ABUSE; ALCOHOL ABUSE PREVENTION; PREGNANCY, fetal alcohol spectrum disorder, fetal alcohol effects, alcohol related birth defects, alcohol related neurodevelopmental disorder*, fas, fasd, fae, arbd, arnd
DARE: Database of Abstracts of Reviews of Effects	National Health Services' Centre for Reviews and Dissemination (NHS CRD) at the University of York, England	Fetal alcohol syndrome, fetal alcohol effects, fetal alcohol spectrum disorder, alcohol related birth defects, alcohol related neurodevelopmental disorder*, fas, fae, fasd, arbd, arnd, maternal, prenatal, pregnan*, fetus, fetal, alcohol*, ethanol, drinking, prevent*, women
Dissertation Abstracts		Fetal alcohol syndrome, fetal alcohol effects, fetal alcohol spectrum disorder, alcohol related birth defects, alcohol related neurodevelopmental disorder*, fas, fae, fasd, arbd, arnd

DATABASE	PRODUCER	SEARCH TERMS[2]
EMBASE	Elsevier Science B. V.	FETAL ALCOHOL SYNDROME, PRENATAL EXPOSURE, ETHANOL, ALCOHOL, ALCOHOL ABUSE, DRINKING BEHAVIOR, ALCOHOL CONSUMPTION, ALCOHOL INTOXICATION, ALCOHOL TOLERANCE, PREVENTION, PREVENTION & CONTROL, PRIMARY PREVENTION, SECONDARY PREVENTION, PREGNANCY, ADOLESCENT PREGNANCY, FIRST TRIMESTER PREGNANCY, SECOND TRIMESTER PREGNANCY, THIRD TRIMESTER PREGNANCY, PRENATL EXPOSURE DELAYED EFFECTS, NEUROPROTECTION, PROTECTION, RISK FACTOR, fetal alcohol spectrum disorder, alcohol related birth defects, alcohol related neurodevelopmental disorder*, best practice*
ERIC	Educational Resources Information Center, U.S. Dept. of Education	FETAL ALCOHOL SYNDROME, PRENATAL DRUG EXPOSURE, fetal alcohol effects, fetal alcohol spectrum disorder, alcohol related birth defects, alcohol related neurodevelopmental disorder*, fas, fae, fasd, arbd, arnd
ETOH: Alcohol & Alcohol Problems Science Database	National Institute on Alcohol Abuse and Alcoholism	FETAL ALCOHOL SYNDROME; FAS; FETAL ALCOHOL EFFECTS; PRENATAL ALCOHOL EXPOSURE; PRENATAL AOD EXPOSURE; PREVENTION, TREATMENT, AND MAINTENANCE. HEALTH CARE; RISK ASSESSMENT; RISK AND PROTECTIVE FACTORS; RISK FACTORS; PROTECTIVE FACTORS; RISK AND RESILIENCY CHARACTERISTICS; RISK FACTOR MODEL; fetal alcohol spectrum disorder*, alcohol related birth defects, alcohol related neurodevelopmental disorder*, fasd, arbd, arnd
First Nations Periodical Index	Saskatchewan Indian Federated College, Saskatoon; Saskatchewan Indian Cultural Centre; and Library Services for Saskatchewan Aboriginal Peoples Committee	Fetal alcohol syndrome, fetal alcohol spectrum disorder, fetal alcohol effects, alcohol related birth defects, alcohol related neurodevelopmental disorder*, fas, fae, fasd, arbd, arnd
HSRProj	National Information Center on Health Services Research & Health Care Technology, National Library of Medicine	FETAL ALCOHOL SYNDROME; ALCOHOL DRINKING; ALCOHOLISM; PREGNANCY; PREVENTION; PREVENTION & CONTROL
Latin American and Caribbean Literature on the Health Sciences (LILACS)	Latin American and Caribbean Center on Health Sciences Information (BIREME)	FETAL ALCOHOL SYNDROME

DATABASE	PRODUCER	SEARCH TERMS[2]
Medline	National Library of Medicine	FETAL ALCOHOL SYNDROME; ALCOHOL DRINKING; ALCOHOLISM; ETHANOL, PRENATAL EXPOSURE DELAYED EFFECTS, PREGNANCY; PREGNANCY TRIMESTER, FIRST; PREGNANCY TRIMESTER, SECOND; PREGNANCY TRIMESTER, THIRD; PREGNANCY OUTCOME; PREGNANCY, HIGH-RISK; PREGNANCY RATE; PREGNANCY MAINTENANCE; PREGNANCY IN ADOLESCENCE; PREGNANCY COMPLICATIONS; FETUS; PREVENTION & CONTROL; fetal alcohol spectrum disorder, alcohol related birth defects, alcohol related neurodevelopmental disorder*, best practice*
National Database of FASD and Substance Use During Pregnancy Resources	Canadian Centre on Substance Abuse	ALCOHOL CONSUMPTION-RISK DRINKING, COMMUNITY MOBILIZATION, EARLY INTERVENTION, HEALTH PROMOTION, INTERVENTION, PRENATAL EXPOSURE, PREVENTION
Pre-Medline	National Library of Medicine	Fetal alcohol syndrome, fas, fetal alcohol spectrum disorder, fasd, fetal alcohol effects, fae, fetus, pregnanc*, alcohol*, drinking behavior, maternal, alcohol related birth defects, alcohol related neurodevelopmental disorder*, arbd, arnd
Project CORK	Project Cork Institute, Dartmouth Medical School	FETAL ALCOHOL SYNDROME, PATERNAL DRINKING, MATERNAL DRINKING
PsycEXTRA	American Psychological Association	FETAL ALCOHOL SYNDROME, PRENATAL EXPOSURE, PREVENTION, RISK FACTORS, AT RISK POPULATIONS, SUSCEPTIBILITY-DISORDERS, ALCOHOLISM, ALCOHOL ABUSE, ALCOHOL DRINKING PATTERNS, ALCOHOL DRINKING ATTITUDES, DRUG EDUCATION, ALCOHOL INTOXIATION, ALCOHOL REHABILITATION, CHRONIC ALCOHOLIC INTOXICATION, fetal alcohol spectrum disorder*, alcohol related birth defects, alcohol related neurodevelopmental disorder*
PsycINFO	American Psychological Association	FETAL ALCOHOL SYNDROME, PRENATAL EXPOSURE, PREVENTION, RISK FACTORS, AT RISK POPULATIONS, SUSCEPTIBILITY-DISORDERS, ALCOHOLISM, ALCOHOL ABUSE, ALCOHOL DRINKING PATTERNS, ALCOHOL DRINKING ATTITUDES, DRUG EDUCATION, ALCOHOL INTOXIATION, ALCOHOL REHABILITATION, CHRONIC ALCOHOLIC INTOXICATION, protect*, best practice*, fetal alcohol spectrum disorder*, alcohol related birth defects, alcohol related neurodevelopmental disorder*

DATABASE	PRODUCER	SEARCH TERMS[2]
ScienceDirect	Elsevier B.V.	Fetal alcohol syndrome, fetal alcohol effects, fetal alcohol spectrum disorder, alcohol related birth defects, alcohol related neurodevelopmental disorder*, fas, ae, fasd, arbd, arnd, prevent*, risk*, protect*, best practice*
Social Services Abstracts	Cambridge Scientific Abstracts	FETUS, PREGNANCY, ALCOHOL ABUSE, ALCOHOLISM, ALCOHOL DEPENDENCY, PREVENTION, RISK FACTORS, Fetal alcohol syndrome, fetal alcohol effects, fetal alcohol spectrum disorder, alcohol related birth defects, alcohol related neurodevelopmental disorder*, fas ,fasd, fae, arbd, arnd, maternal, prenatal
Social Work Abstracts Plus	National Association of Social Workers	Fetal alcohol syndrome, Fetal alcohol spectrum disorder, alcohol related birth defects, alcohol related neurodevelopmental disorder*, fetal alcohol effects, fasd, fas, fae, arbd, arnd, fetus, pregnancy, alcohol abuse, alcoholism, drinking behavior, prenatal exposure
Sociological Abstracts	Sociological Abstracts Inc.	Fetal alcohol syndrome, fetal alcohol spectrum disorder, fetal alcohol effects, alcohol related birth defects, alcohol related neurodevelopmental disorder*, fasd, fas, fae, arbd, arnd, fetus, pregnancy, alcohol abuse, alcoholism, drinking behavior
Web of Science	Institute for Scientific Information	Fetal alcohol syndrome, fetal alcohol effects, fetal alcohol spectrum disorder, alcohol related birth defects, alcohol related neurodevelopmental disorder*, fas, fae, fasd, arbd, arnd, prevention, risk, protect*, best practice*

Library Catalogues

Online catalogues of several libraries likely to have relevant materials related to FASD were searched to identify significant non-journal literature related to the problem area. Items thus identified included monographs, government publications, theses, dissertations, reports, conference proceedings, and other types of grey literature. Standard search terms such as Library of Congress Subject Headings and NLM Classification terms were used where possible. Catalogues searched included those of the National Library of Medicine (LocatorPlus), The Canadian Centre on Substance Abuse (Library Collection Database), the Centre for Addictions and Mental Health, Library and Archives Canada (AMICUS), the National Library of Australia, and the University of Lethbridge Library.

Checking Reference Lists

The reference lists of all publications obtained were checked for references relevant to the review that had not yet been identified, and copies of these items were obtained.

Review articles were particularly helpful in this regard. Reference lists were especially useful for identifying earlier publications and grey literature, which is less likely than recent periodical literature to be retrieved from electronic databases.

Handsearching of Key Journals

In addition to searching electronic databases, the *Journal of FAS International*, a key journal in this area, was searched by hand to ensure that all relevant articles were included in the review. Time and resource limitations did not permit handsearching of any additional journals.

WWW Searching

World Wide Web searches were also used to identify grey literature related to FASD. Web searches were especially useful for locating research and policy documents of various levels of government agencies, as well as the reports of non-government agencies and organizations working in the area of FASD prevention.

The primary WWW search strategy involved the exploration of over 50 sites maintained by institutions, governments, and organizations known to be engaged in, or supporting, FASD-related research and prevention. Most of the relevant web documents retrieved were identified in this manner. Search engines such as Google and Yahoo were used as a check to identify documents missed through the initial strategy of exploring sites focused on subjects related to FASD.

Contact with Experts

Finally, 15 key investigators in the area of Fetal Alcohol Spectrum Disorder were contacted in order to identify important studies that may have been overlooked, research that was completed but not published, and studies currently in process.

Review Process

In order to minimize bias and permit a broad, multidisciplinary approach to evaluating the literature on FASD prevention, the 14-member review team was drawn from a variety of disciplines representing both research and clinical perspectives. The team included a Core Group of eight researchers from disciplines including Nursing, Psychology, Addictions, General Social Science, Epidemiology and Genetics, Social Marketing, Communications, Social Psychology, Education, Sociology, and Health Sciences Librarianship. In addition, the team included an Extended Core Group of six health care practitioners from clinical settings that deal with FASD in the Lethbridge and nearby First Nations communities. Finally, ongoing expert advice and guidance was provided to the review team by a Steering Committee of four individuals with recognized expertise in the areas of Medicine, FASD, and First Nations Public Health.

The selection, critical review, and synthesis of FASD prevention literature took place during a period of approximately four months during the summer and early fall of 2004. During this time the review team met frequently in order to ensure consistency in the review process and to build a common base of knowledge and ideas as the review progressed.

Members of the Core and Extended Core Groups met approximately every two weeks from the end of April through June to develop review procedures and to ensure consistency in the application of these procedures. The team met weekly throughout the summer and approximately biweekly in early fall to share knowledge and insights gained from reading the literature, to develop a common understanding of the current status of knowledge related to FASD, and to identify gaps in the existing evidence on FASD prevention.

In addition to the Core Group meetings, two meetings were scheduled with the Steering Committee in July and September of 2004. These meeting times were selected in order to coincide with critical points in the review process where advice and direction from the Steering Committee would be most useful

Study Selection and Critical Appraisal

Execution of the search strategy described above resulted in the identification of 1400 studies. Copies of 1081 of these publications relevant to FASD prevention were obtained and bibliographic information, abstracts, and keywords for each study were added to an EndNote database. Copies of each study were then distributed to two independent reviewers for critical appraisal. Team members were selected to review particular studies based on their subject expertise relative to the study subjects.

Reviewers evaluated each paper in terms of its scope, objectives, samples, research methods, and application to models of best practice. A range of outcome measures, both quantitative and qualitative, were considered. The success of prevention strategies or models can be measured by a reduction in incidence and prevalence of FASD, a reduction in number of women drinking while pregnant, or a reduction in amount of alcohol consumed while pregnant. Although the latter outcome is based on a harm reduction model, which acknowledges that the goal of prevention should be abstinence, some studies identified reduced consumption as a positive outcome and were, therefore, included in the review. The use of drug and nutritional intervention in animal models to ameliorate the effects of maternal alcohol consumption on the fetus was also considered as an outcome measure in this review.

Following the critical review of each study, reviewers made independent recommendations as to whether a particular study should be included in or excluded from the review. Studies were selected for inclusion if they were judged to be relevant to FASD prevention and of highest quality. Specific criteria for inclusion included: appropriate prevention strategy/model; appropriate participants; experimental study design; time frame; emphasis; language; and peer review. Document quality was ranked according to a three-point categorical scale: 1 (poorly done); 2 (average); and 3 (exceptional, that is, ground breaking or seminal research, rigorous methods applied). Studies were included if they had been categorized as a "3" quality level by both independent reviewers. Selected studies, coded "2" quality, were also included, mainly for illustrative purposes. All studies coded "1" were excluded. Of the 1081 documents reviewed, approximately 400 met project standards of quality and relevance, and were included in the systematic review process.

Following the completion of the independent reviews, reviewers met to compare and discuss their findings and to resolve any differences of opinion as to the quality of particular

studies and whether or not they should be included in the review. Differences of opinion not resolved in this way were brought forward for discussion to the rest of the project team.

Data Extraction and Synthesis

The review process was facilitated by the development of an electronic data extraction form (see Appendix A) to assist reviewers with independently conducting systematic and comprehensive reviews. Together with an accompanying codebook containing definitions, categories and rating scales, the data extraction form served also to standardize review criteria across reviewers. The form allowed for the recording of both quantitative and qualitative data describing key aspects of the study under review including its objectives, methodology, findings, conclusions, and significance. Before being used, the data extraction form was subjected to a trial whereby each reviewer used an initial version of the form to assess several articles representing a variety of study types. The review team then met as a group to discuss the review experience, evaluate the form, clarify procedures for its use, and modify the form to eliminate problems encountered in the trial review process.

Following its completion by the reviewer, each data extraction form was submitted to a central database of data extraction forms. Review team members did not have access to this database until after the blind-review phase of the project was completed. At this point, access was opened up to all team members, allowing them to easily retrieve and share knowledge and ideas gained from critical reading of the literature. The database of data extraction forms also facilitated the writing of the final report.

Regular review team meetings were used to bring the knowledge gained from reading and the subject expertise of various team members together to bear on the state of the evidence related to FASD prevention. Through these regular discussions, a common knowledge base was synthesized and consensus emerged regarding the state of the evidence, including gaps and problem areas. These discussions thus provided a foundation for the writing of the final report that follows, which was shared among team members according to their areas of expertise and the subject focus of their reading.

Chapter Three

Historical Perspective

Ancient Period

The notion that FASD or FAE is a recently identified phenomena is questionable given that reviews undertaken by historians and scholars of FAS often cite numerous sources from antiquity to the post-modern world, that highlight the dangers of alcohol consumption and its effect on the unborn child (Abel, 1984; Danis et al., 1981; O'Leary, 2002; Overholser, 1990; Peak & Del Papa, 1993; Streissguth et al., 1980). The Great Greek philosopher, Aristotle, identified the relationship between abnormal fetal development and maternal alcohol consumption and in early biblical times the following quote from Judges (13:3-4) is often referenced:

> "Behold now, thou art barren, and bearest not: But thou shall conceive, and bear a son. Now therefore beware, I pray thee, and drink not wine, nor strong drink, and eat not any unclean thing."

Peak & Del Papa (1993) cites Soranus (Rome first or second century AD):

> The seed when attached must be nourished. But in drunkenness al vapor is spoilt and the pneuma is rendered turbid. Therefore, danger arises; the seed changes for the worse and the satiety due to heavy drinking hinders the attachment to the uterus. (p. 245)

This quote suggests there is difficulty with the fertilized ova from firmly attaching to the walls of the uterus and may be the reason why alcohol procures spontaneous abortion.

Abel (1984) cites the following quote from Rabbathi and Nadarim (20b),

> "Children begotten during a state of inebriety are mentally deficient" (p. 2).

The point in the above sentence suggests that ancient people did have some knowledge and understanding of alcohol related teratogenicity. However, quotations taken out of context can sometimes lose their true meaning and there is a danger that interpretations are made using personal bias. For instance, interpretations can serve the needs of the historian or researcher as opposed to being an objective observation. Abel (1984) is an advocate that caution should be heeded given that verification is not available to ratify one's interpretations. He illuminates this fact by further examining quotes that are often used by historians and providing alternative viewpoints. This caveat being said, not all writers heed this caution and often portray commonly held views or interpretations as fact. Danis et al. (1981), for example, points out the Roman physician, Soranus of Ephesus, second century, AD, was indeed well informed of the dangers of the harmful effects of alcohol on the fetus, and of the nursing child. Further to this observation, Danis makes reference to the ancient Carthaginian ritual that forbade alcohol consumption by the groom and his bride to prevent birth defects of children. This ritual is of particular note given that there is a recommended

abstinence of alcohol consumption by the bride and groom, which may indicate that there was knowledge that FASD can be associated with maternal and paternal alcohol consumption. In support of this concept, Danis et al. (1981) makes reference to Burton (1621) who wrote, "if a drunken man get a child, it will never likely have a good braine" (p. 7). While this view may not be supported by empirical knowledge, it does reflect some element of understanding and generally goes further than most contemporary literature that heavily focuses on maternal alcohol consumption with FAE.

Early Modern Period

The legislation of alcoholic beverages in England circa 1720-1750 brought about the overproduction of cheap Gin that flooded the market and raised concern among certain legislators about the effects of excessive alcohol consumption among the poor. In 1751, an Act of Parliament limited the sale and production of distilled spirit in response to a rising social consciousness that alcohol consumption was having a major effect on social order and public health. Throughout these intervening years numerous references by physicians were made to FAE. Warner and Rosett (1975), in their historical review of the period, have captured some of these expressed and publicly documented concerns. For example, (i) the College of Physicians (1726) reported to Parliament that maternal alcohol consumption was the cause of weak and ill-tempered children; (ii) in 1736, a committee of the Middlesex Sessions reported similar sentiments, further adding that children born of alcoholic mothers were shriveled and had an old appearance; (iii) in 1751, there were observatory remarks made that a drop in birth rate and non-viable infants was directly associated with parental alcohol consumption during the Gin epidemic; (iv) women drinkers were more than likely to suffer from spontaneous abortions; and, (v) those children who lived usually had some form of mental retardation.

This period in history was influential in formulating the moralistic thinking behind the English and American Temperance movements whose proponents condemned the partaking of alcohol in the quest to support abstinence and temperance. These moralistic judgments, while based on real-life observation, ignited defensive responses towards the benefits of alcohol consumption from both journalistic and medical writings. Nonetheless, during this period, there were the beginnings of scientific reasoning that at least supported the moralistic claims that alcohol consumption did affect the fetus.

Benjamine Rush (cited in Danis et al., 1981) was an early American Statesman who had the insight to warn against the practice of prescribing alcohol to women to relieve morning sickness. Rush also warned of the consequences on the fetus of parental inebriety during conception, and the continued pattern of women drinking alcohol throughout the duration of pregnancy. Other moralistic scholars of the same period issued similar warnings. Indeed, Charles Darwin also contributed to the debate suggesting that the consumption of liquors was more than likely a contributing factor to alcoholic-related diseases that could be passed on to second and third generations. In England, Thomas Trotter (cited in Abel, 1984; Danis et al., 1981; Streissguth et al., 1980) addressed alcoholism as a disease entity and predicted that alcoholic women would indeed produce children who had low intelligence due to brain damage. Trotter also claimed that the cause was due to deficient germinal cells of the

parents resulting from heavy consumption of alcohol. While Trotter was attempting to break new ground, his work was never really taken seriously.

Early Epidemiological Studies

By the middle of the 1800s increasing attention was been given to FAE. In 1834, a Select Committee reporting on the evidence of drunkenness highlighted the fact that alcoholic mothers often produced children who had deformed features and looked emaciated. In this same time period, Howe (cited in Abel, 1984; Streissguth et al., 1980), an early epidemiological pioneer, investigated 300 inmates who were classified as mentally retarded, and found that nearly half of their parents were reported as being alcohol dependent. Indeed, one couple, who were heavily alcohol dependent, produced seven idiot children. Sulliven, in 1899 (cited in Abel, 1984; Danis et al., 1981; Streissguth et al., 1980;) examined 120 alcoholic women from a Liverpool prison. The outcome of his study determined that these women had between them 600 children of whom only 44% survived beyond the age of two. In addition, he found that when women were incarcerated and unable to gain access to alcohol, the survival rates of children were higher than their children born in freedom, when assumingly, they were drinking alcohol. In Switzerland, Bezzola (cited in Abel, 1984) began to correlate evidence arising from the conception of children and annual festivals where binge drinking was part of the celebrations. From his calculations, Bezzola was able to determine that "between 1880-1890, 8196 'imbeciles and idiots' were born out of approximately 1 million births" (Abel, 1984, p. 17). Conception occurred during the festival period for most of these births, thus indicating that alcohol consumption and, in particular, binge drinking was a significant factor in birth anomalies. Classical epidemiological studies by Laitenen (cited in Abel, 1984) concluded that spontaneous abortion affects moderate (four times) and heavy (almost seven times) drinkers greater than abstainers. Further, Madden (1899, cited in Danis, 1981) proclaimed that alcoholism and degeneracy were passed on to the child from the father through alcohol-damaged sperm and via the mother through her lifestyle throughout pregnancy. This view was supported by Stockard (1913, cited in Danis, 1981), who re-eamined the evidence pertaining to paternal influence on the developing fetus. He quotes the results of Rosch (1837) who found testicular degeneration in male alcoholics and Simmonds (1898) who observed there was a high incidence of dysmorphology in male alcoholics. In 1907, Horsley and Sturge (cited in Danis et al., 1981) identified that alcohol was the cause of disability and malformed children, or they were stillborn.

In 1910, the debate relating to FAS shifted direction. Elderton and Pearson (1910) suggested the focus of attention was too narrow and that there were other factors that should be considered, for example, parents' health; social influences, and environmental factors. This perspective was a seminal piece of investigatory inquiry that considered both protective and mediating factors.

Early Animal Studies

At the beginning of the 20th century, in the quest to explain the phenomena of FAE, scientists embarked on animal studies to best explain the physiological effect of ethanol and how alcohol affected pregnancy under controlled conditions. While their experimental design may have been rudimentary (Abel, 1984), the results often confirmed epidemiological and

clinical studies that had been reported. Between 1894 and 1903, Fere (cited in Abel, 1984; Danis et al., 1981) experimented with exposing hens' eggs to alcohol vapours. His results confirmed that ethanol influenced large numbers of stillbirths and dysmorphology that was consistent. Nonetheless, it is interesting to note that not all hens were affected even though they were subjected to the same ethanol exposure. This findings creates a conundrum that continues today. Some hypotheses suggest that alcohol can have a positive and negative affect and can be a selective agent attacking the weak and leaving the strong (Pearl, cited in Abel, 1984). Other animal studies of this period portrayed a similar picture in that alcohol did have some teratological effect on the developing fetus. In 1940, Jellineck and Jolliffe, minimized the teratogenic ability of alcohol in humans. They concluded that there was no valid evidence and the production of idiots through maternal or paternal alcohol intoxication was in the realm of fantasy. These assumptions were the root cause of confusion for the next 30 years, with arguments polarizing from the position that ethanol was teratogenic towards the fetus or that there were other confounding environmental and social issues that add to FAE. For example, Haggard and Jellinek (cited in Abel, 1984) proclaimed that:

> There is a need to separate the impact on the germ from its impact on the home environment. . . . The child of the inebriate may suffer great handicaps, but these handicaps are inherent not in the germ but in the unfavourable environment which the inebriety of the parent creates. (p. 23)

Following this form of logic, preventative strategies appeared unwarranted. Such ideological musings became entrenched in the political and scientific rhetoric. In 1955, Keller, in a pamphlet, said, "old notions about children and drunken parents can be cast aside" (Abel, 1984, p. 25). This concept was supported ten years later by Montague,

> It can now be stated categorically, after hundreds of studies covering many years, that no matter how great the amount of alcohol taken by the mother or father--neither the germ cells nor the development of the child will be affected. (Abel, 1984, p. 35)

Summary

We have incorporated a contextual and historical background so as to illustrate the key developments and milestones in the recognition that there is a universal understanding that alcohol does indeed affect the development of the fetus in a plethora of ways. While historical records have, through intuitive knowing, acknowledged the dangers associated with maternal alcohol consumption on the fetus, it is only in the modern period that real progress has been made with regard to understanding the aetiology, epidemiology, and prevalence. Nonetheless, it is well acknowledged by contemporary scientists that there still remain some unanswered questions so that we can truly inform the public and professional community on the basis of sound empirical evidence. Given this situation, we can hypothesize that our ancestors were not that far behind contemporary research in their collective understanding since numerous historical sources do acknowledge the fact that FASD/FAE is a preventable human tragedy and caused by alcohol consumption.

Chapter Four

Diagnosis

The original description of the constellation of characteristics identified by Jones and Smith in 1973 and, thus labeled FAS, has been re-examined over the intervening years. The Fetal Alcohol Study Group of the Research Society on Alcoholism has been instrumental in providing a list of minimal criteria that must be met before a diagnosis can be made for FASD. These criteria include prenatal and post-natal growth retardation, and at least, two of the following characteristic facial features:

- Microcephaly,
- Microopthalmia, and/or, short palprebal fissures,
- Midfacial hyperplasia (defined as absent or rudimentary philtrum, thin vermilion, border of upper lip, hypoplastic maxilla).

If these criteria are not met in the absolute form, but damage from ethanol is still suspected, then FAE can be attributed as the diagnosis. As Table 2 illustrates, the range of symptoms that is associated with FAE/FASD is extensive, and can be classified from physical, cognitive, and affective domains.

Table 2
Associated Symptoms

Physical	Cognitive	Affective
Short palpebra fissures (eye slits) / microopthalmia	Mental retardation	Tremulousness (infants cry easily, are awakened easily, react with a startle to mild stimuli – irritable and jittery)
Low nasal bridge and short nose	Abnormal EEG	Hyper responsiveness
Indistinct philtrum (ridges between nose and mouth)	Learning disability	Infants – attachment insecurity (high maternal support can offset)
Narrow vermilion of upper lip	Low IQ – delay in reaching developmental milestones	Expression of negative emotions
Flat midface	Intellectual disability (Oleary)	Young children: impulsive
Prenatal and postnatal growth retardation (Danis, O'Leary)	Poor concentration	Uninhibited
Poor coordination	Memory problems	Overly friendly
Hyperactivty	Perceptual problems	Inquisitive
Abnormal state regulation (sleep-wake cycle) in infants	Widely varying abilities	Demanding for affection and physical contact

Physical	Cognitive	Affective
Microcephaly or other structural brain anomalies with no catchup	Difficulties with math, memory, fluctuating capacity, spatial orientation, self-awareness , reflection	Intrusive
Hypoplastic maxillary (underdeveloped upper jaw)	Inflexibility of thinking	Insensitive to social cues
Low nasal bridge	Difficulties with executive functioning: planning, judgment, delayed gratification, impulse control, future orientation, organization, focus, concentration	Lacking in social judgment
Short nose	Danis abnormal neuronal and glial migration	Aggressive behavior
Low birth weight, length, head circumference	Incomplete cortical development (agyria and thinning)	Inattention
Epicanthal folds (corner of upper eyelid)	Small, malformed cerebellum	Problems with social interaction and attention
Lack of catch up growth – even with adequate nutrition	Abnormal cerebrospinal fluid hemodynamics (hydrocephaly, syringomyelia, enlarged ventricles	Intensity, urgency
Low weight to height ratio	Brainstem malfomations	Little ability to recognized feelings
Delayed development – fine and gross motor difficulties, poor motor skills (walking, balance, weak sucking reflex, weak grasping, poor coordination)	Abnormal cavities in the brain substance (porencephaly)	Little ability to articulate feelings
Hyper acusis (extreme hearing)	Lack of olfactory cortex (arhinencephalia)	Mood disorders
Hypotonia (no muscle tone)	Agenesis of the corpus callosum	Rage disorders
Reduced adipose tissue	Reduced synaptic binding of Ca	Vulnerability to mental illness
Speech and language difficulties	Increased norepinephrine uptake	Inability to read social cues
Hearing disorders: delay in auditory maturation, sensorineural hearing loss, intermittent conductive hearing loss due to serous otitis media (o'leary, 04)	Elevated cAMP and cGMP neuron levels	Lack of empathy, bonding
Spontaneous abortion & still birth	Increased acetylcholine release at motor endplates	Inability to distinguish truth from fiction
Pathologic organ conditions	Decreased Ach release in the CNS	Externalization of blame

Physical	Cognitive	Affective
Heart and kidney problems		
Visual disorders	Depressed postsynaptic potentials	Excessive demand for attention
Allergies, asthma, ear infections	Accelerated postsynaptic decay	Superficial fluency
Ptosis (drooping eylid)	Altered REM sleep	Talkativeness
Strabismus (crossed eyes)	Decreased sarcolemmal membrane function	Parroting of others; speech patterns
Cardiac defects (primarily septal defects)	Inhibited amino acid uptake through blood-brain barrier	Expressive language better than receptive
Renal anomalies (primarily hypoplasias)	Impaired sodium current	General delay in communication
Abnormal palmar creases	Increased choline transport	
Pectus excavatum	Inhibited Na – K pump	
Weak sucking reflex in infants	Increased brain GABA and DOPA levels	
Low-set posteriorly rotated ears	Decreased brain Ach and glutamate levels	
Presacral dimple	Altered cell transport mechanisms	
Micrognathia in childhood – receding chin	Membrane Na-K ATPase inhibition	
Prognathia in adolescence – jaw protrusion - underbite	Increased fluidity of cell membranes	
Low APGAR scores (indication of respiratory distress)	Decreased adenyl cyclase activity	
Repetitive, self-stimulatory behavior	Altered NAD redox potential	
Congenital hip dislocation	Inhibited aminoacyl tRNA synthetase	
Inability to fully extend elbows	Inhibited mRNA transport	
Camptodactyly and clinodactyly (abnormal bending of fingers)	Altered mitochondrial morphology	
Short metacarpals and metatarsals	Expanded cellular membrane	
Radioulnar stenosis	Inhibited ribosome function	
clubfeet	Depressed fatty acid oxidation	
Cleft palate	Stimulated ketogenesis	

Physical	Cognitive	Affective
Hypoplasia of toenails	Increased body fluid/electrolyte retention	
Septate vagina	Respiratory and metabolic acidosis	
Hypoplasia of labia majora	Inhibited antidiuretic hormone release	
Cryptorchidism (undescended testicles)	Serum and tissue hypocalcemia	
Neonatal seizures	Decreased sex steroid production	
Hydrocephaly	Increased glucocorticoid release	
Capillary hemangiomas	Reduced absorption of vitamins B folate	
Spinal stenosis	Increased plasma triglycerides and very low density lipoproteins	
Pectus carinatum	Reduced serum amino acids	
Hepatic abnormalities	Reduced vitamin and hormone transport	
Abnormal hair patterns	Reduced conversion of vitamins to active forms	
Hirsutism in infancy	Reduced vitamin storage	
	Inhibited uptake of bloodborne nutrients	
	Inhibited absorption of all nutrients from G1 tract	
	Inhibited folate metabolism	
	Increased excretion of Zn, Mg, Ca	
	Hypokalemia due to diarrhea, vomiting	
	Inhibited pancreatic digestive secretions	
	Tissue iron retention	
	Inhibited thyroid function	
	Inhibited glucagon effect	
	Increased glycolysis	
	Decreased glycogen synthesis, gluconeogenisis	
Black = almost always present, Blue = frequent more than 20% of patients, Green = not often reported (Danis, 1981)		

Danis et al. (1981) suggests that this list, as it appeared in 1981, was too exhaustive and negated a true reflection of the characteristics of FASD/FAE, although it must be noted that it has grown since that time. In reality, it has more to do with the over zealous enthusiasm of investigators attributing any fetal/infant developmental abnormality that can not be readily explained, as being somehow associated with maternal alcohol consumption. His rationale, for this observation, is due to the absence of any "clinical observations [that have] been controlled by determining the incidence of the supposed anomaly in a population of infants without FASD" (p. 12).

While the physical facial deformities are a most striking feature of FASD, the cognitive damage to children can be severely debilitating and has tragic consequences for the duration of their lives. The mental acuity of such children is far below normal ranges and is often measured on Baley scales of infant development, followed by the Stanford-Binet form L-M or the WISC-IQ test when older. Most FASD children do not reach normal developmental milestones, cry easily, and quickly react to any mild stimuli. FASD children are often described as hyperactive and troublesome children who have poor or minimal concentration levels. Notwithstanding, attributing all of these symptoms with the direct consequences of maternal alcohol consumption is problematic, in that, other environmental factors may be the cause of some of these behavioral and cognitive disabilities.

Children with FASD/FAE are susceptible in developing secondary disabilities such as: mental health problems, school disruptive experiences, negative experiences with the legal system, inappropriate sexual behaviour, and alcohol- or drug-related health issues (Burd et al., 2003).

What is known is that ethanol exposure does affect the children's cognitive abilities to some degree or other; therefore, it is not surprising that the small number of autopsies undertaken (Clarren & Smith, 1978; Jones & Smith, 1973, 1976; Pfeiffer et al., 1979) have demonstrated that there is neuro-pathological damage. All the deceased children under study were microcephalic, with the exception of one, and, they all had small brains. Further, EEG tracings of children whose mothers were classified as heavy drinkers showed some degree of abnormality, indicating that there is a potentially elliptogenic state. Nonetheless, such evidence needs further enquiry given that the spectrum of anomalies associated with FASD/FAE can have various effects and degrees on different individual. In addition, there are questions raised regards any mediating effect associated with the children's external environment throughout the nurturing period.

It is clear that diagnosis of newborns, and adults, is extremely difficult coupled by the fact that reliable cumulative data on ethanol exposure during pregnancy does not exist (Stratton et al., 1996). Other diagnostic difficulties occur due to the fact that: (i) a large number of FASD children are adopted or in foster care at the time of diagnosis, (ii) information regarding the birth mother's alcohol exposure during pregnancy is inadequate, and, (iii) there are no known biomarkers. These obstacles are challenging given the notion that early diagnosis provides the infrastructure needed to facilitate a meaningful deficit-based treatment program.

It is essential that health professionals have full knowledge and understanding of FASD/FAE and not confuse the diagnosis with other syndromes such as: (i) Noonan's

Syndrome, which is characterized by a broad face, ptosis, short stature, heart disease with a normal kayotype and usually an undetermined inheritance (Hall & Orenstein, 1975; Noonan, 1976); (ii) Cornelia De Lange's syndrome, which is characterized by physical retardation, cognitive dysfunction, heart disease, and a characteristic face; (iii) Dubowitze syndrome, a rare disease, which is characterized by physical retardation, cognitive dysfunction, hyperactivity, microcephaly, and midfacial hyperplasia (Majewski et al., 1975); (iv) Turner's syndrome; (v) Bloom's syndrome; and (vi) fragile X syndrome (Burd et al., 2003).

Provision of information related to FASD is a necessary step in prevention and health promotion. As a means to provide information about FASD, consequences of alcohol consumption during pregnancy and the profound effect that FASD has on society, initial diagnosis strategies are required. It is apparent that the early diagnosis of FASD plays a role in long-term interventions (Stoler & Holmes, 2004; Streissguth, Kogan, Bookstein, & Barr, 1996). This information includes the protective factors encompassing stable living environments and eligibility for special services. Diagnosis is an advantage when developing appropriate and probable plans for the management of future behaviour expressions, health problems, school placement, and general life functioning (Appelbaum, 1995). Early diagnosis of FASD can lead to long-term benefits on familial, medical, educational, and societal levels (Appelbaum, 1995*)*. Unfortunately, diagnosis at the time of birth is very difficult as the presenting characteristics may be subtle (Abel, 1998).

Beyond diagnosing as the means toward FASD prevention, accurate evaluations will also demarcate similar appearing developmental disabilities. Diagnosis is important in order to delineate between FASD and other diagnoses that may appear to be alike but the resultant interventions and remediation issues may be vastly different (Olson, Streissguth, Sampson, Barr, & Bookstein, 1997). Cole (2001) examined effects of prenatal alcohol exposure on attention factors and the relationship between these factors and attention deficit hyperactivity disorder (ADHD) diagnosis through a comparison of ADHD children with FASD children (N = 149). Results found that these two groups of children had unique attentional profiles, calling into question assumptions regarding FASD children as having similar neurocognitive deficits as ADHD children. Children with FASD may be misdiagnosed at the school level with Learning Disabilities as they experience persistent academic failure with a reduction in reading, mathematics, and spelling skills (Jacobson & Jacobson, 1999).

There is no single diagnostic test for FASD and there is considerable overlap with other syndromes. Diagnosis is typically made on infants and young children in clinical settings through healthcare workers. The diagnostic approaches utilized are typically either through characteristic facial features, growth delays, and central nervous system anomalies (see Table 3). The use of facial features can be achieved by using a cutoff of four out of the six facial characteristics associated with FASD; researchers hope that physicians will be able to identify FASD at an early age (Stoler & Holmes, 2004). Using children under the age of ten years, Astley and Clarren (1995) found that a trained dysmorphologist was able to distinguish between children with FASD and those without FASD using only facial phenotypes. Features characteristic of FASD individuals include short palebral fissures, epicanthal folds, midface hypoplasis, depressed wide nasal bridge, anteverted nares, long hypoplastic philtrum, and a thin upper vermilion border (Abel, 1998; Appelbaum, 1995; Stoler & Holmes, 2004). Limitations to this type of identification includes physician training

regarding the identification of these facial features, a clear definition of these characteristics must be created – and one in which racial or ethnic features are taken into account, and facial characteristics may not always be indicative of FASD effects in later childhood.

A refinement of utilizing the facial features has been achieved through the 4 Digit Diagnostic Code (4DDC), encompassing quantitative and qualitative measurement scales and definitions in an attempt to overcome the use of a gestalt approach of diagnosis (Astley & Clarren, 2001). Practitioner feedback and resulting studies confirm the 4DDC to be a viable and precise diagnostic tool in clinical settings for FASD diagnosis. Adding other mitigating factors present in FASD has created a further refinement of the facial features evaluation. The Fetal Alcohol Behaviour Scale describes the behavioural factors of FASD regardless of age, gender, cultural background or intelligence level (Streissguth, Bookstein, Barr, Press, & Sampson, 1998). This scale incorporates the Personal Behaviors Checklist (PBC), first developed by the first author in the 1970s, as a 68-item questionnaire. A shorter, 36-item version of the PBC was used in this study, along with a Life History Interview. Research results find this diagnostic tool to have adequate test-retest reliability although the utility as a screening tool has not been verified. It appears to hold potential to capture the long-term consequences and behavioural effects of FASD.

Table 3
Identification Tools for FASD: Diagnostic

Tool	Description	N	Research findings	Researchers
Facial anomalies	Clinical examination for identification through: Short palpebral fissures Epicanthal folds Midface hypoplasis Depressed wide nasal bridge Long hypoplastic philtrum Thin upper vermilion border	293	Correlated with screening tools. Low sensitivity to facial score but overall accuracy was high	Stoler & Holmes (2004)
Facial phenotypes	Clinical examination for identification through: Eyes and eyebrows (i.e., palpebral fissures, canthal distance) Midface (i.e., flat nasal bridge, hypoplastic) Mouth (i.e., philtrum, palate) Palmar creases (i.e., hockey stick crease) Body measures (i.e, height)	194	Effective in distinguishing between FASD and nonFASD individuals by dysmorphologist	Astley & Clarren, (1995)
4 Digit Diagnostic Code	Clinical examination for diagnosis using combination of growth deficiency, facial phenotypes, brain damage/dysfunction, and gestational alcohol exposure	445	Advantageous over gestalt methods of FASD identification. High correlation between face and brain.	Astley & Clarren (2001)

Diagnostic procedures, designed to determine an individual's actual condition, are often time consuming, invasive, and costly. In contrast, screening methods are relatively inexpensive and require little time to administer. Burd et al. (2000) offer a comprehensive

argument in favor of screening for FASD, stating that this practice will improve identification and outcomes, decrease costs, allow for more reliable prevalence estimation, reduce secondary disabilities and facilitate interventions for mothers. Screening devices are focused on maternal behaviour rather than the presenting characteristics of the child. Currently, brief questionnaires represent the most efficient method of screening for risk drinking during pregnancy (see Table 4). Identifying women who drink during pregnancy poses challenges, particularly because the definition of risk drinking has been altered and refined over time (Chang, 2001).

Two diagnostic tools that appear to be promising during prenatal intake appointments in health units are the TWEAK and T-ACE. The TWEAK was found to have best predictive ability for a lifetime alcohol diagnosis and for at-risk drinking through a clinical study of 1165 women in the United States (Chang, Wilkins, Berman, & Goetz, 1999). These women were predominantly Caucasian, nonsmokers, average age of 30 years, and mean of 16.25 weeks gestation. Information was collected at a hospital setting with intake questions and telephone interview follow-up. There is consensus that self-report measures provide the most accurate information regarding prenatal alcohol use and identification of risk drinking (Russell, Martier, Sokol, Mudar, Jacobson, & Jacobson, 1996). Until there is one "gold standard" of diagnosis, self-report measures in conjunction with counselling appears to be the most effective means of screening and intervention. Although the MAST, CAGE and AUDIT screening tools are also readily available, consensus is that they offer no advantage to the TWEAK and T-ACE (Chang, 2001; Chang, Wilkins-Haug, Berman, & Goetz, 1999; Russell, Martier & Sokol, 1994). Chang (2001) advocates that screening instruments be included in all intake encounters as routine use of screening instruments in clinical practices may reduce the stigmatization of asking patients about their alcohol use and result in accurate and consistent evaluation.

Table 4
Identification Tools for FASD: Screening

Tool	Description	N	Research findings	Researchers
TWEAK	Alcoholism screening questionnaire Self-report measure Designed to detect alcoholism or heavy drinking 5 items Acronym based on: Tolerance, Worry, Eyeopener, Amnesia, Cut down on drinking	1165	Promising screening instrument for identifying risk drinking during pregnancy.	Chang et al., . (1999).
		4743	High sensitivity. Limited ability to generalize findings. May not have significant advantages to T-ACE.	Russell, Martier, & Sokol (1994)
T-ACE:	Alcoholism screening questionnaire Self-report measure First validated screen for risk drinking 4 items Acronym based on: Tolerance, Annoyed, Cut down, Eyeopener	971	Superior to MAST and CAGE. Efficient to use; takes one minute per client.	Sokol, Martier, & Ager (1989)
		1420	High efficiency rating. Sensitivity was adequate with low positive predictive value	Russell, et al., (1996)

Tool	Description	N	Research findings	Researchers
		350	Sensitive to lifetime alcohol diagnosis, risk drinking, and current alcohol consumption. Efficient screen for potential risk drinking.	Chang et al., (1998)
MAST	Alcoholism screening questionnaire Self-report measure 25 items Acronym based on: Michigan Alcoholism Screening Test	238	Better indicator than self-reports of drinking although underreporting of drinking is problematic	Morrow-Tlucak et al, (1989).
CAGE: Cut down on drinking, Annoyed, Guilty, Eyeopener	Alcoholism screening questionnaire Self-report measure Assesses lifetime rather than current alcohol related problems 4 items Acronym based on: Cut down on drinking, Annoyed, Guilt, Eyeopener	196	Less sensitive in psychiatric settings Women may feel guilty identifying alcohol consumption, thus lowering specificity of test	Watterson & Murray-Lyon (1988)
TWEAK, T-ACE, MAST & CAGE	Comparison of the four alcoholism screening questionnaires	2717	TWEAK more sensitive than T-ACE in every item TWEAK and T-ACE more sensitive to risk drinking CAGE not sensitive to risk drinking MAST is cumbersome to use due to length	Russell, et al., (1996)
AUDIT: Alcohol Use Disorders Identification Test	Screening questionnaire Self-report measure 10 items Identifies hazardous and harmful drinking	350	Superior predictive ability, best overall accuracy in determining lifetime alcohol diagnosis.	Chang et al., (1998)

A serious limitation with screening devices and face-to-face interviews is the purposeful veiling of alcohol consumption information. When interviewed prenatally, women with serious alcohol related problems are increasingly likely to underreport their alcohol consumption, and this view was found to be consistent when reporting during pregnancy (Morrow-Tlucak, Ernhart, Sokol, Martier, & Ager, 1989). A means of mediating this secrecy would be to create a nonjudgmental atmosphere with trained personnel who could provide relevant information for alcohol abstinence approaches and treatment strategies. A multidisciplinary setting in which numerous treatment, diagnosis and intervention, and support services could be housed is one manner in which an individual could elicit needed assistance.

Multidisciplinary diagnostic clinics may facilitate the proper diagnosis of conditions in patients who have been appropriately identified in other clinical settings (Clarren & Astley, 1998). This multidisciplinary approach may be particularly useful in identifying individuals with FASD so their conditions can be diagnosed and their birth mothers can be

identified and referred to prevention services. Focusing prevention efforts on at-risk mothers, namely those who have previously given birth to an alcohol exposed child, could reduce the incidence of FASD births dramatically with little impact on the present healthcare and alcohol treatment systems (Astley, Bailey, Talbot, & Clarren, 2000; Clarren & Astley, 1998). Inadequate criteria and untrained healthcare workers for diagnosis are thought to be reasons for the failure of recognition and diagnosis of children who have experienced prenatal alcohol exposure (Stoler & Holmes, 2004). Multidisciplinary settings could also pool their personnel expertise for diagnosis and screening.

A barrier in FASD screening is linked to the training of healthcare personnel who engage in the evaluation techniques. In a study reviewing the practices and training of nurses in the USA, nurses (N = 827) reported that they screen more pregnant patients in comparison to those of childbearing age, and screen more for tobacco use in comparison to alcohol consumption or use (Betzner, Luxenberg, Evered, & Rainey, 2001). Fewer than 30% of the nurses indicated that they screen all women of childbearing age at all clinic visits for alcohol use, although 49% of the nurses reported that they do not screen during acute care visits. Conclusions drawn from this study indicate that 75% of the nurses believe they require additional training for alcohol screening and protocol for post-screening interventions or education.

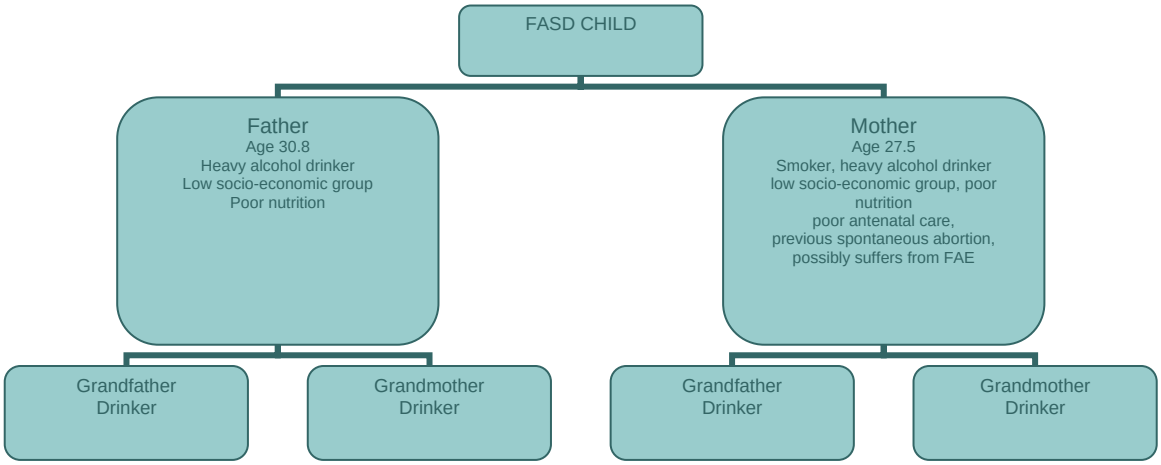
Biochemical Markers

In view of the difficulties with current self-report alcohol screens, the growing evidence of the efficacy of biochemical markers is becoming more attractive. Existing biomarkers for ethanol use during pregnancy all have some degree of utility, but further research is needed. Among these potential markers are ethanol concentration; metabolites of ethanol; enzymes involved in ethanol metabolism; products of the interaction of ethanol metabolites and cellular components; alteration of target protein (e.g., CDT); and early indication of target organ damage. The CDT test provides a reliable estimate of long-term alcohol intake (Golka, 2004). However, this type of testing may not detect women who may be occasional binge drinkers, a practice that clearly puts the fetus at risk.

Ethyl oleate concentration in meconium assayed by GC/MS/MS provides a highly sensitive and specific indicator of maternal ethanol use during pregnancy (Bearer et al., 2003). Whole blood-associated acetaldehyde; carbohydrate-deficient transferrin, glutamyl transpeptidase, mean red blood cell volume were more predictive of infant outcome than self-report measures of ethanol consumption (Stoler et al., 1998). The use of GGT in screening for ethanol abuse in pregnant women has poor sensitivity, and, because of the low prevalence, many false positives (Whitfield, 2001).

In making an accurate diagnosis, it is important to recognize that there are often some familial tendencies that are generational and other causal links such as co-morbidity with other drugs and the environment in which they live. In making a diagnosis, a family tree can be useful to determining familial lifestyle behaviors and living conditions.

Figure 1
Typical Familial Profiles



Summary

Diagnosis of FASD/FAE remains a contentious issue in that there is an extensive range of symptomology, which can be classified from the physical, cognitive, and affective domains. Table 2 illustrates this point in an explicit fashion. Questions have been raised if all of these symptoms are truly associated with FASD/FAE, or, is it that the professional community labels clusters of symptoms that can not be associated with another disease category to FASD/FAE (Danis et al., 1981). Such a cluster of symptoms may indeed be associated with other environmental factors that are rarely considered during the diagnostic assessments. Notwithstanding, the evidence suggests that, whilst essential, diagnosing the newly born infant is difficult to undertake in all instances. It is only when the child fails to thrive, or, demonstrates learning difficulties, or, antisocial behaviors that questions arise relating to maternal alcohol consumption during pregnancy. Unfortunately, any delays in making an accurate diagnosis compound the ability of the professional workforce to assist in any meaningful long-term interventions that support positive health outcomes for the child, the mother and immediate family members.

The question is often asked with regard to the efficacy of the tools and instruments used in helping make the diagnosis. Some of these tests were adopted from other domains and have been relatively unchanged for the last three or four decades. The reliability and validity of these tests have been called into question by the scientific community, and, there is now a move to validate these instruments and tools in an attempt to standardize a universal diagnostic approach. Adopting a universal model would enable the battery of tests used to be truly evaluated for their efficiency and effectiveness in helping the professional community make accurate diagnoses.

The evidence from our review also points to the need for the professional community to be educated and trained to have the relevant knowledge and skill that would enable them to be truly competent in this field. In addition, there is a need that professional workers must

be able to work together in a multidisciplinary team framework as no one person has all the answers. Clarren and Astley (1998) have illustrated the need for multi-professional diagnostic clinics as a specialist service for the identification of FASD/FAE. This suggestion appears to be a model of good practice.

Currently, science does not offer specific biochemical markers, which help in the diagnostic assessment. Potential markers have been identified such as: ethanol concentrations, metabolites of ethanol, enzymes involved in ethanol metabolism and alteration of target proteins.

In making an accurate diagnosis there are other factors that come into play such as the environment in which women live, their nutritional status, and other familial and generational links.

Chapter Five:

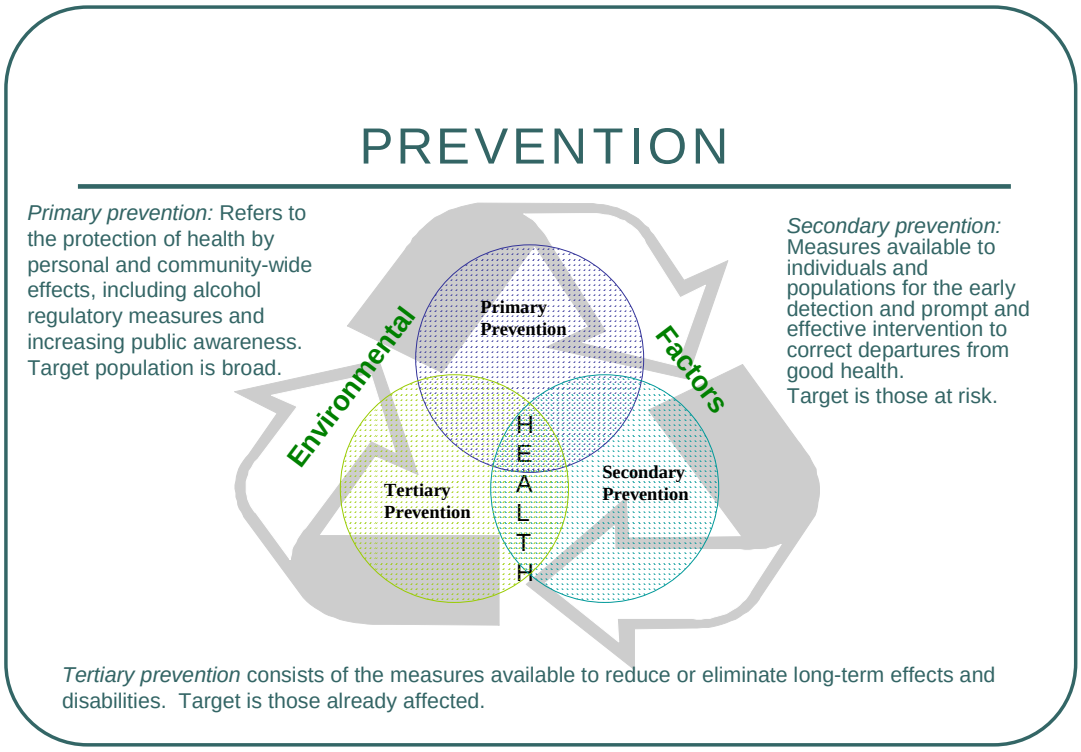
Prevention: Theoretical F rameworks

Preventing ill health is considered to be the panacea to eradicate health-related problems associated with our lifestyle and living conditions. Indeed, the public health improvements to our living conditions over the last century (e.g., clean water supplies, effective and managed sewage disposal systems, unpolluted air supplies, better housing, good nutritious food supplies, and effective transportation) have been instrumental in improving health and longevity for individuals, communities, and populations. Nonetheless, while this public health incentive was acknowledged by all to have reduced the incidence of some diseases that were endemic to societies, the preventative models that are seemingly effective in changing lifestyle behaviours has been significantly under resourced and little understood by the health community. In part, this situation was due to the political direction and focus on developing a health service infrastructure that supported the advancement of medical services that offered cure and treatment regimes as a priority. Preventative medicine, while acclaimed to be useful in eradicating disease, has been a *Cinderella* service with respect to securing the necessary financial and human resources from successive governments. Nonetheless, with the growth in non-communicable diseases that has seemingly accelerated an early death or contributed to years of morbidity and a poor quality of life, governments across the globe are now rethinking their health service infrastructures to embrace the concept of preventative medicine and preventative health care in the acknowledgement that prevention is clearly the better option in: (i) sustaining health, and (ii) in the promotion of healthy longevity. This decision is not to say contemporary medicine does not play a significant role relating to the promotion of health and on saving lives when illness has already occurred. But, what must be emphasized is that modern health care must adopt a balanced approach through the incorporation of traditional medicine and preventative models of care. For this change to happen, effective government policy must seek ways to support preventative models that support individuals and communities in improving their health and wellness and in addition, meet health targets that have been determined by the World Health Organization (1998).

What is Preventative Medicine/Preventative Health Care?

Preventive medicine or, in a broader sense, preventative health care is framed around three dimensions, that of primary, secondary, and tertiary preventative models. The challenges relating to the primary concepts are fundamentally directed to alter lifestyle behaviors that will work towards the procurement of health throughout the lifespan.

Figure 2
Prevention Model



To be able to develop and advance primary public health models that indeed are effective in enabling people to make lifestyle changes and to sustain them, it is recognized that we must first understand the nature of lifestyles and people's health belief models. Failure to do so means lifestyle changes are neither achieved, nor sustained, over a significant period of time.

Lifestyle as a Generic Construct

Lifestyle has been defined and discussed in the context of sociological and psychological discourses that acknowledge lifestyle in a holistic template and explores an individual's practice and behaviours across a diverse set of domains and contexts. These behaviors, and ways in which people carry on their everyday activities, reflect personal, group and social identities (Giddens, 1991). Thus, an understanding of the delicate and intricate nature of lifestyles is crucial in enabling individuals to undertake lifestyle changes. Working in the field of FASD clearly illustrates this point in that women who are with spouses, friends and families who commonly drink alcoholic beverages are unlikely to engage in total abstinence. Leading on from the latter concept, Simmel (1950) illustrates that the development of lifestyle is intrinsically linked to the development of societies and cultural norms. Simmel further postulates the influence of modernity and how the growth of the metropolis came at the expense of individual autonomy and freedom. What followed, in Simmel's opinion, was that the loss of personal freedom meant individuals were required to buy into socially-constructed and expected patterns of behaviour; he goes on to say:

> that the deepest problems of modern life derive from the claim of the individual to preserve the autonomy and individuality of his (or her) existence in the face of overwhelming social forces, of historical heritage, or external culture, and of the technique of life. *(*p. 409)

This kind of logic may explain how cultural expectations on individuals are very strong and how there is a cognitive tension between individual direction and the prescribed lifestyle that is dominant and historically perpetuated in the societies within which they live. From this perspective, it is evidently clear that preventative public health models need to be multi-generational and engage in models that have multi-levels of influence if success is to be achieved.

Max Weber (1864-1920; cited in Wrong, 2003) embraces the notion that the status (i.e., caste or ethnicity) of an individual or group of people is of more relevance than their standing in a class system. Weber elaborates on this point by suggesting that a status group denotes members of similar status, shared cultural background and political persuasion, and yet, of significant importance is the fact that they all share similar lifestyles. We can see from this explanation that in cultures or communities that are closely intertwined how important it would be to initiate preventative models of practice that target the individual, but also the higher standing members of the group or clan. Engaging and influencing senior members of the group is, therefore, important to support and sustain lifestyle health promoting behaviours in the recognition that these group leaders will promote and support lifestyle changes to individuals whose health is at risk.

The focus so far has been on explaining lifestyle as an independent construct, however, as Cockerham et al. (1997) elaborate on Max Weber's theory of a duel exchange between life chances and life choices. Weber determined that there are two distinct characteristics of lifestyle: (i) life conduct, or life choices and (ii) life chances. Life choices can only be truly realized if there are choices to make; for example, we can only choose to adopt health-seeking behaviours if there are opportunities and resources to do so (i.e., nutritious food can only be chosen if individuals have the options and the resources to do so, or, if the alternative to consuming alcoholic beverages is contaminated water then there is no real choice to be had). Therefore, when advocating lifestyle changes, health professionals must ensure there are choices to be had that would sustain the changes recommended. Supporting this position, Dahrendorf (1979) postulated that life chances are not only concerned with economics or material assets, but extend to the constraints – actual or perceived - individuals assume. Moving on Weber's discourse and analysis of lifestyle, Cockerham et al. (1997) have pointed out that there are other factors that come into play such as gender, age, race, and ethnicity. Further, Bourdieu (1984) provides an explanation that emphasizes the dialectical interaction between agency and structure. He suggests that there is a dynamic interplay in that the individual behaviors can also inform and continue to shape the environment. Bourdieu notes this dynamic interplay with one's environment as 'Habitus,' a model that relates to:

> Systems of durable, transportable dispositions, structural structures predisposed to operate as structuring structures, that is, as principles which generate and organize practices and representations that can be objectively adapted to their outcomes without presupposing a conscious aiming at ends or

> an express mastery of the operational necessary in order to attain them. (p. 53).

Habitus, thus described, relates to a cognitive map that individuals can call upon to make socially acceptable healthy lifestyle decisions. The cognitive map (figuratively speaking) is the computerized data, which are stored and developed or reconstructed from prior experience. From birth, our external and internal experiences continually inform this cognitive map. If, for instance, their early social experience with community celebrations is that alcoholic beverages are a central component of the feast that results in drunkenness and associated risks, then their cognitive map would have computed this activity as an acceptable part of lifestyle behaviours.

The cognitive map is not a linear framework but is likened to a schematic framework. Rummelhart and Norman (1981) suggest that there are three distinct processes: accretion, tuning, and restructuring.

- **Accretion:** the individual absorbs and records the existence of a new form of schema and records this within the existing cognitive map.
- **Tuning:** describes how the individual capitalizes on experiences to elaborate and refine attributes of the schema.
- **Restructuring**: describes how the individual creates new schema by applying existing schema to a new situation.

The implications here are that by adulthood, individuals have a worldview that has developed a broad range of schemas related to various contexts. This worldview would address the ways in which they live their lives that includes healthy (or, alternatively, non-healthy) lifestyle behaviors. Their worldview is in no way static, as we know from experience the human form has the capacity to adapt to new life situations. Cockerham et al. (1995) concur that when an individual encounters a situation and a habitus is called upon, the individual is able to make independent judgments as to whether that particular lifestyle or route is chosen. Bourdieu (1984) makes the claim that the selection process is bound by societal or cultural rules and that habitus can be an unconscious or conscious response rather than a reflective interpretation. He continues to claim that there is a choice associated with lifestyle but no such thing as free will because of the restraining nature of habitus developed through adhering to societal norms and expectations.

Lifestyle as a Health Construct: Behaviors

Steptoe and Wardle (1996) have produced an overview of definitions, which distinguishes between healthy lifestyle behaviours and those that are deemed risky and damaging to health. Risky behaviour is a term that relates to lifestyle activities that have an increased risk of incurring injury and/or the development of a disease state. The increase in risk may be associated with their external environment. Risky behaviours can include smoking, alcohol consumption, and other drug cocktails, or it could be associated with living in a toxic environment. By contrast, healthy behaviours are more difficult to define (Steptoe & Wardle, 1996), in that a universal definition as to what constitutes healthy lifestyles is subjected to an explosion of varying scientific information over the proceeding decades. A low-fat diet, once postulated as a “healthy alternative” is now questioned relating to its

validity. Nonetheless, Mildred Blaxter (1990) suggests there is a danger in considering health behaviours from a purely individual perspective. She states that the majority of people participate in behavioural patterns that have both a positive and negative effect on health profiles and it is usually social characteristics of the individual that determine which behaviors are likely to be performed. In essence, Blaxter, is suggesting that while health behaviours may be relevant to health, they cannot be addressed in isolation of the social environment in which people live. Cockerham et al. (1997) further postulate that healthy lifestyles are also value laden and require a certain attitude towards health. These characteristics are strongly associated with the individual's surrounding cultural and economic backdrop. This message is repeated by other scholars and should be of central consideration when addressing the issue of alcohol consumption during gestation.

The Complex Phenomenon of Health Lifestyles

O'Brian (1995), speaking of contemporary societies, suggests that the concept of lifestyle has indeed taken on a different perspective in that lifestyle as a phenomenon has infiltrated the frameworks of health care practice. He postulates there are two dimensions to this notion; (i) the influences relating to individual's experiences of health care provision, and (ii) the recognition that lifestyle will alter the ways in which health care is provided. As mentioned in earlier paragraphs, the focus on health care, over this last century, has been on a 'cure and treat' model. Political persuasion and the recognition that the increases in chronic illness are due directly to lifestyle and living conditions have forced a challenge to traditional ways of working and philosophical thought. Modern medicine has, therefore, begun to explore the concept of health through a lifestyle perspective rather than examining the human body in separate parts. Arguably, this paradigm shift regarding lifestyle and health should drive the research agenda to advance knowledge relating to the intricate relationships between the two concepts. Unfortunately, there remains a degree of confusion and contradiction due to the diverse political, economic, social, and cultural forces that underpin health promotion and lifestyles. Instead of simplifying research, there is a complexity that to some researchers may be viewed as a muti-faceted kaleidoscope that requires unraveling to a simple form that is understandable. Lifestyle in itself, is not a simple entity and, therefore, is not a tangible independent variable but is a phenomenon that embraces both cognitive and behavioral elements as part of the whole. In addition, lifestyle choices must be viewed within an individual's social location. For example, what may be seen as acceptable health behaviour in one culture may be viewed as detrimental to health in another.

Locus of Control as a Model of Health Behaviour

When considering health beliefs it is important to understand the concept of empowerment through the process of control. Control models used in the locus of control context attempt to elucidate whether the person sees that health is based on: (i) individual power and control, or (ii) the responsibility of powerful others, or, (iii) is related to fate or chance. For example, for those who view the world from an internal perspective they would state that, "If I get sick, it is my own behaviour that determines how soon I get better," or, "When I get sick I am to blame," or, "If I take care of myself, I can avoid illness." Those who hold the belief of powerful others might make the following statements: "My family has a lot to do with my becoming sick or staying healthy," or, "Regarding my health, I can only do

what the doctor tells me to do." Finally, from the perspective of chance or fate, statements like the following are appropriate: "Most things that affect my health happen to me by accident," or, "If it's meant to be, I will stay healthy."

Jenkins and Bursih (1995) contend that there are differing suggestions made in the literature that relate to the importance of health locus of control varying from the fact that internal beliefs are associated with health prevention activities to concepts that allude to the fact that individuals who display external attribution display greater benefits to health. Neither of these comments considers the adaptive element, and others have identified that both the internal and external attribution is more akin to adaptation (Rotter, 1996).

Through highlighting the differing theoretical discourses that relate specifically to lifestyle and health beliefs, it becomes evident that to implement effective preventative models there needs to be a comprehensive understanding regarding the ways in which people can change their lifestyle behaviours to better improve their own, and others' health and well-being. The following discussion provides an understanding about how the various prevention domains are applied in practice.

FASD Prevention

All scientists throughout the ages concur that FASD/FAE is a wholly preventable condition (Abel, 1984). Ideally, the utopian preventative model would be aimed at complete abstinence of alcohol use prior to conception, during pregnancy and throughout the period of lactation by the woman, her partner, and support group (Burd et al., 2003). Universal prevention, thus described, would be supported by sustained and high profile public health policy and effective social marketing strategies to better inform the public and health professionals. In essence, primary prevention is directed at the healthy population to maintain or enhance their physical, mental, social, and spiritual health, and well-being. Primary prevention activities are focused on people's lifestyles and living conditions, and if necessary, requires behavioural changes, improved systems, and/or attention to their environment (Robertson & Nanson, 2000).

At one level, primary prevention considers a population health approach that is informed by empirical evidence from research findings and directed through public policy. All aspects that affect health are considered, that is, (i) socioeconomic factors (e.g., education, employment, income levels, and social support); (ii) environmental factors, such as air pollution, water quality, sanitation, housing, leisure activities and transportation, and (iii) biological (e.g., bacteria and viruses that may cause epidemic disease manifestations).

Universal Prevention: A Public Health Model Defined

Public health is a universal model that attempts to prevent ill health and promote the health and well-being of all individuals, communities, and populations. Therefore, models that apply to a universal model are targeted to the general public or an entire population group that is identified based on risk analysis. Examples of such models would be water fluoridation and childhood immunizations. Other sub-categories of the universal model are referred to as (i) Selective, and, (ii) Indicated Intervention.

Interventions designed specifically for women during pregnancy or the pre-conceptual period are related to a universal prevention model. It could include the encouragement of abstinence as a responsible approach to motherhood, which if applied universally, would have the effect of eradicating FASD/FAE. From a general principle, the population would be informed, have knowledge of, and fully understand the relationship between the consumption of alcohol and its teratogenic effect. The purpose of this approach would ensure pregnant mothers, and possibly fathers, do not engage in risky behaviours that are likely to harm their unborn child. Public education either through the television, radio or other media approaches serves to fulfill the universal model, but it can also include changes in the social environment (Single, 2002). For example, social environments can be altered through changes in the law and regulation, or changing social and cultural norms that consider alcohol consumption during pregnancy to be unacceptable.

In principle, the universal model certainly has an appeal, and yet, universal preventative practices have not produced a satisfactory reduction in FASD/FAE. This situation begs the question relating to the efficacy of the universal model as it is applied to date. Is it because the target audience is the higher socioeconomic group in society that are usually more educated and widely read? Is the message conveyed supported by all of the literature? Are there various messages delivered that serve to confuse? Or, in our consumer driven societies, do we no longer internalize what we read as fact? Warning labels on cigarette packages and alcohol products do not appear to have an impact on the reduction in risky behaviors.

Health care providers are ideally situated to use universal prevention models given that at some stage in the pregnancy the mother, and possibly the father, will come in contact with a health professional. During these visits, opportunities are presented to convey messages of the dangers of alcohol consumption during pregnancy (American College of Obstetricians and Gynecologists, 1994). It has been shown that just learning of a pregnancy has been stimulus enough for women to decrease their alcohol consumption. However, to effect long-term change, follow-up programs are considered to be necessary given that targeted information regarding alcohol consumption and FASD within prenatal clinics has shown some positive changes in the behaviour of pregnant women both during and post pregnancy. Such an approach is often referred to as selective prevention and incorporates both counselling and information giving.

Selective prevention interventions are targeted to individuals or, a subgroup of the population, who are severely disadvantaged and/or at greater risk. For example, a woman whose female family members may have died through breast cancer would be considered at risk and invited to have yearly mammograms. World travelers who go to countries with identified water-born infections (e.g., typhoid paratyphoid would seek immunization). From an FASD perspective, a risk reduction model would be encouraged. This model would include providing the mother and father with greater scientific information relating to the social and personal risk factors associated with FASD. The epidemiological risk factors identified to date demonstrate the complexity surrounding FASD and are confounded by the history of alcohol consumption (i.e., frequency and amount), marital status, consumption of other addictive substances such as smoking or cocaine, age, and nutritional status of the mother, her social environment, and level of education.

Selective prevention strategies are primarily focused on women but the influence of their spouse, and significant others, have been noted. Therefore, selective prevention should also include fathers. It may well be that there are some alcoholic effects on the sperm that are significant in FASD, but to date, no empirical evidence exists to support such a notion. However, it is well known that a heavy drinking spouse continues to support alcoholic consumption in the women throughout her pregnancy (Abel, 1993; Russell, 1991). Consequently, it is imperative that the spouse is equally as informed as the women relating to the dangers of maternal alcohol consumption.

The success of selective prevention programmes is still under scientific scrutiny but what is evident is that approximately 40% of women continue to drink during pregnancy even when they know about the risks. Eighty percent of women decrease their consumption upon learning of a pregnancy, but only 60% quit drinking alcoholic beverages completely. Those women who continue to drink do so because they see no other socialization alternatives available and have less motivation to quit drinking (Hankin et al., 1993; Kaskutas, 1995).

Indicated prevention intervention strategies are used to target high-risk individuals who either have biological markers as an indication of disease, or, who are showing signs of early symptoms. Such targeted interventions have been modified to be appropriate for pregnant alcoholic women to be targeted for treatment for their alcoholic dependency as an appropriate prevention intervention for the developing fetus (Stratton et al., 1996).

Primary Preventive Strategies

Social marketing is generally a primary prevention tool. A prime example is the policy calling for warning labels placed on alcoholic beverages in the United States. Such programs can be both educative and legal, but follow-up studies suggest these strategies are only of limited efficacy. In spite of this relative lack of success, social marketing theory espouses that this approach is worthwhile and well-designed primary prevention programs can impact in a positive manner.

Social marketing or social change strategies have been employed to promote abstinence among pregnant women. Three broad-based approaches have been utilized: education only (media campaigns), legal approach (warning labels in US), and community based programs. Media campaigns and warning labels have had some success in effecting behaviour change through awareness; however, community-based programs have only been effective with addicts.

Educational programs have had some success in preventing FASD. Targeted education to assess current knowledge awareness of fallacies and increasing awareness of alcohol effects has increased women's knowledge of the timing and nature of possible effects of alcohol consumption. In follow-up studies, both female and male students were able to identify the links between alcohol and FASD. Information provided to junior-high and high-school populations regarding alcohol consumption and the direct links to FASD has increased knowledge of alcohol effects in general. Again, follow-up with female students indicate that they will not consume alcohol during pregnancy. These results indicate that curriculum inclusion of education on the effects of alcohol consumption from elementary to

postsecondary settings to educate females prior to, and during, childbirth years is effective in reducing FASD. Further, educating women on the risks of drinking through counselling and providing guidance to improve nutrition and reduce environmental risk factors have had some success. Therefore, it is recommended, in addition to the education of health care professionals to better assess alcohol consumption and diagnose FASD as a primary prevention tool, that those working in educational settings (e.g., counsellors, health teachers) must become more knowledgeable in order to disseminate alcohol and FASD information through adolescence.

Culturally, primary prevention has followed the practice of exploring the kinds of information possessed by, for example, Native Indian women as well as their drinking practices when they learn about their pregnancy. It is recognized that prevention programs can be effective in increasing knowledge among Native women. In one study, O'Connor and Whaley (2003) noted that the best predictor of post-conception alcohol consumption was the woman's high-risk drinking score as measured by the TWEAK. It is also acknowledged that more FASD programs are available in rural areas and on reservations than in urban areas for Native people. This finding causes concern for the large proportion of women in urban settings who do not have access to culturally specific prevention programs. Unfortunately, researchers, who pursue culturally diverse populations, also inform us that Native Indian women are often predisposed to drinking behaviour because of their upbringing within familial tendencies to excessive drinking by their parents and spouses or partners. Further, they tend to use alcohol as a means of escaping abuse. Within such environments, when educational opportunities are limited, researchers in the southeast US observe that young women of African-American, Hispanic, and White descent, are likely to follow this behaviour, too. Researchers in Southern California note that Native Indian women often lose their children as a direct result of their addiction to alcohol. In their work with Northern Plain Indians, Kvigne and colleagues (2003) conclude that intervention is critical for women who have an FASD child, because successive children with FASD demonstrate even more dramatic effects. Again, the need to prevent this tragedy is paramount.

Secondary Preventive Strategies

It has been shown that just learning of a pregnancy has been stimulus enough for women to decrease their drinking, suggesting there is an optimum time window to reach women at risk. Nevertheless, to effect long-term change, follow-up programs will be necessary. Targeted information regarding alcohol consumption and FASD within prenatal clinics has shown some positive changes in the behavior of pregnant women both during and post pregnancy. This change includes abstention and decreased alcohol use. Counselling sessions coupled with information to reinforce educational efforts is deemed to be the most effective strategy. Success in programs from Washington State and Toronto, Canada indicate education of women at risk in the areas of the risks of alcohol consumption and the value of improved nutrition and other protective factors can improve birth outcomes.

Tertiary Preventive Strategies

Tertiary prevention is designed to ameliorate the symptoms and problems of those already affected by FASD. These programs include treatment for substance abuse, training to

provide enriched environments that may improve the life quality expectancies of those impacted. Coping with FASD (discussed later) is primarily a tertiary preventive strategy.

The key to tertiary prevention policy is leadership provided through meaningful policy emanating from government departments (e.g., vehicle, social, and health departments).

Summary

All scientists throughout the ages concur that FASD/FAE is a wholly preventable condition (Abel, 1984). Ideally, the utopian preventative model would be aimed at complete abstinence of alcohol use, prior to conception, during pregnancy and throughout the period of lactation by the woman, her partner, and support group (Burd et al., 2003). Universal prevention, thus described, would be supported by sustained and high profile public health policy and effective social marketing strategies to better inform the public and health professionals. In essence, prevention is directed at the healthy population to maintain or enhance their physical, mental, social, and spiritual health, and well-being. Universal prevention activities are focused on people's lifestyles and living conditions, and if necessary, requires behavioral changes, improved systems, and/or attention to their environment (Robertson & Nanson, 2000).

At one level, universal prevention considers a population health approach that is informed by empirical evidence from research findings and directed through public policy. All aspects that affect health are considered, that is, (i) socioeconomic factors (e.g., education, employment, income levels and social support), (ii) environmental factors, such as air pollution, water quality, sanitation, housing, leisure activities and transportation, (iii) biological (e.g., bacteria and viruses that may cause epidemic disease manifestations).

For women who have admitted they are consuming alcohol throughout their pregnancy a selective prevention strategy is used that provides the women with more detailed information and counseling. Women who are deemed as high risk and/or may have symptoms of alcoholic abuse will be appropriately targeted for treatment regarding their alcohol dependency.

Prevention is seen as a utopian intervention for many of today's ills, including FASD. However, working in the preventative field is often viewed as a lesser skill than those high technical interventions used in secondary care. Notwithstanding, to execute prevention models in its various guises requires a great deal of skill and mastery. Assessing and diagnosing the current health status and the levels of alcohol consumption is just one dimension, and the knowledge of people's lifestyles, living conditions and cultural norms are of equal importance if a sustained abstinence of alcohol is to be achieved throughout pregnancy, and the period of lactation. In addition, knowledge of the ways in which people cope with adversity and health problems is also necessary.

Chapter Six:

Coping with FASD

When a family has to face the fact that they have a child with FASD or FAE, there are untold emotions that have to be addressed so that they can best cope with the situation. It is not a short-term situation that is quickly dispelled, it continues over the women's lives and that of their FASD children. Such a situation is bad enough, and yet women are often ostracized by the community in which they live, and by professional bodies who they and their child encounter along the way. There is indeed stigmatization and blaming that is directed at these women, the suggestion being that it is totally the women's fault, and yet, is it? If science, professional understanding, and political guidance is found wanting are women and their families really to blame? Is there not a social and professional responsibility and a public conscience?

Our review illustrates that there are confused messages within the literature that deny a universal understanding relating to FASD. We have also heard the anecdotal tales of women who, for the most part, do not understand the detailed nature of their circumstances, and their child's situation. This situation is compounded by the fact that the diagnosis is often delayed beyond the realm of reasonableness, and throughout this conundrum, mothers of FASD children are often imbued by guilt, shame, anger, denial, blaming others, and withdrawal from family and community life.

Throughout all of the emotional backdrop and protracted grieving, mothers are faced with the added burden of coming to terms with not only helping their affected child who may present with challenging behaviors and other health deficits, but in developing coping strategies that facilitate their own health and well being, and those of other family members. Coping strategies used may have a positive or negative health outcome; sadly, the latter is more commonly the norm and the negative coping strategies used are often undiagnosed or assessed by the professional community.

Notwithstanding the above, it is important to remember that women and their immediate families develop positive coping strategies so as to prevent other compounding social and environmental problems within the family unit. For example, negative coping strategies can facilitate further emotional damage to the child that can impede their development. Additionally, negative coping strategies can act as a catalyst to family violence. In this sense, violence begets violence, which, in turn, can lead to depression of the women. Depression can increase the need for alcohol to block out feelings. Alcohol facilitates violent and disturbing behaviors, and so the cycle continues (see Figure 3).

Figure 3
Negative Coping Strategy Cycle

One of the problems here is that health professionals, in the main, fail to have an in-depth knowledge and understanding of positive and negative coping strategies that are used in the context of FASD. Further, health professionals commonly fail to draw on other scientific literature that addresses the issue of healthy coping strategies so that a sound knowledge base can be gained to prevent secondary and tertiary FASD complications. Notwithstanding, the professional community cannot take full responsibility given that the literature we examined was devoid of such explicit understanding. Indeed, only one article was reviewed that considered coping strategies as a direct focus (Lazarus, 1993). It was, therefore, felt necessary that we highlight foundational theories to provide insights into the nature of healthy coping behaviors that can be used to prevent FASD/FAE.

Health Coping Strategies

Reference in the proceeding paragraph has identified that coping strategies can have either a negative, or positive, health outcome. Verbrugge (1990) has stressed the importance of effective accommodation to particular stressful events, acting as a buffer to prevent the loss of functional disability, or, a diminished sense of well-being. In addition, effective coping strategies are considered essential in enabling the achievement of personal goals and feelings of self-efficacy and personal control. Nevertheless, to be able to adopt positive coping strategies is not a simple task, and it involves several processes. Lazarus (1981), in his seminal piece of work, has identified three distinct phases relating to the coping process. The first involves the appraisal of the threat or harm posed by the stressor, second, the individual's ability to confront the stressful situation and, lastly, the individual's assessment of the effectiveness of coping strategies employed by the expectations of success. Lazarus cautions readers in assuming the three stages are linear as one stage may re-invoke a preceding phase. Knowing, for instance, that a coping response is effective and readily

available may result in reappraising the stressor as less threatening. Alternatively reappraising the level of threat or coping strategy used may result in a negative coping event. Manne and Zautra (1990) have reported on several mediating factors that may influence the coping effort. Primarily, they (i) examine the success or failure of previous coping strategies used, which may influence the individual's ability to cope more effectively, and (ii) examine the perceived coping effectiveness that results from their self-evaluation, which may have either a positive or negative outcome.

Coping definitions

Within the health domain there are several definitions of coping as a paradigm (Craig & Edwards, 1983; Folkman & Lazarus, 1985; Thornbury, 1982). Scott et al. (1980) defined coping as:

> "a process characterized by continuous use of goal directed strategies that are initiated and maintained over time and across encounters by means of cognitive appraisal and the regulation of emotional and physiological responses" (p. 177).

Lazerus and Folkman (1984) defined coping as a series of interventions with which individuals engage to avoid being harmed by life stressors. Coping is a complex phenomenon that requires changes in the cognitive and behavioral efforts through which external and internal demands are appraised as demanding to the point whereby the individual's coping resources are exhausted. Weisman (1979) proclaims that, "coping combines perception, performance, appraisal, correction, followed by further activity and directed motivated behaviour" (p. 27). From this perception, the aim of coping is to gain mastery, control and resolution (Weisman & Worden, 1977).

Gaining a universal understanding is further complicated through the relationship and similarities with the term *coping style.* From a psychological perspective, coping style refers to a relativly enduring mode of functioning (Kirby et al., 1997). Thus, an individual's coping style relates to characteristic perceptions, thoughts and actions. How an individual copes with erroneous situations is a result of their coping style (e.g., denial, withdrawal, fate, etc.) and current situational variables. Notwithstanding, *coping strategies* refer to intra-psychic activities, in addition to communications and actions used to alleviate the suffering caused by the distress. Coping strategies are more varied, diverse, and changeable than coping styles and account for the person's perception of meaning and attitude towards the adverse situation (Lipowski, 1970). The problem of definition arises because the two terms are used interchangeably within the literature. For the purpose of this review we have focused on coping strategies.

Coping Theories

Individuals perceive their situations in differing ways, for example, the glass is either half full, or half empty. For some women having a child with FASD is a situation that is easily overcome, while for others it is a lifetime tragedy. Such differences of opinion influence the ways in which they cope. On reviewing the literature it is evident that scientific authors describe various ways through which coping is examined. There is little consensus

concerning the most effective way to conceptualize coping responses. Some authors have attempted to categorize coping strategies into a higher order framework. Pearlin and Schooler (1978) have described four functions of coping behaviour: (i) prevention of stress from events or situations, (ii) alteration of the situation or problem, (iii) change in the meaning of the situation, and (iv) management of the symptoms or reactions to the stress. Lazarus and Folkman's (1984) narrative on coping suggests that cognitive behavioral theory of stress describes coping as a process that involves two aspects, appraisal and coping. Appraisal requires cognitive functioning that evaluates the degree of threat that is generated by the situation. The appraisal process can ascertain if the situation is positive and one to be embraced, or negative, in that the threat will harm the physical or psychological self. After the initial primary appraisal has been concluded, secondary appraisal may come into play. The secondary appraisal involves the assessment of what might be done about the situation and whether a given coping strategy will be effective. From this perspective, two general higher order constructs can be formulated: (i) problem-focused coping or behavioral strategies, which aim at managing the external environment aspects of the stressor; and, (ii) emotion-focused coping or cognitive strategies that regulate the individual's internal state or emotional reactions to the stressor. Problem-focused coping strategies and cognitive appraisals are considered to have a positive health outcome, as opposed to emotion-focused, or avoidance strategies that do not deal with the situation at hand. Research has consistently reported on the relationship with emotion-focused coping strategy with negative health outcomes (Folkman & Lazarus, 1985; Roth & Cohen, 1986). Mullen and Sulls (1982) offers a different perspective suggesting that flexibility is the key and the different use of coping strategies throughout the different stages of the threat/stressor optimizes people's ability to effectively cope. Cohen and Wills (1985) have identified two constructs through which social support may influence health: (i) the buffering hypothesis, and (ii) the main effect hypothesis.

The Buffering Hypothesis

The buffering hypothesis considers the fact that social support has significant benefits for individuals who are exposed to stressful life events (Sherbourne et al., 1992). Its existence is verified when the perceived availability of social support is calculated (De Ridder et al., 1997). Cohen and Wills (1985) expand this concept by asserting that measurements illustrate that social support in the forms of emotion, esteem, or information have buffering effects regardless of the stressors encountered. While other aspects of social support such as instrumental support and companionship, have a buffering effect only when there is a direct relationship to the stressful event. Further, the buffering effect does not have a sustained effect and is an issue to consider when long-term support is required.

There are differing opinions relating to the stress-buffering model with health outcomes. Arling (1987) contends that there is limited evidence to support the buffering effect on positive health outcomes, activities of daily living impairment and economic deprivation on psychosomatic symptoms and emotional distress. Revenson et al. (1983) concluded that social support did not impact on the social well-being of individuals.

The Main Effect Hypothesis

The main effect hypothesis is conceptualized through the concept of social integration that embraces feelings of stability, predictability, and self-worth. Further, it recognizes the negative impact social isolation and social exclusion have on health. The problem with this concept is that there are no guarantees that social integration is always an indicator of effective coping strategies. Nonetheless, it does reflect the functional outcome of social integration.

Individual Health - Coping Strategies

As previously mentioned, individuals determine and internalize life events in differing ways. There are those who will take active steps to remedy the situation while others will resign themselves to their fate. The coping strategy used is also influenced by external factors such as demographic elements such as age or sex; environment in which they live; individual's locus of control; and, the nature and extent of the situation. The effectiveness of an individual's ability to cope is dependent on previous coping strategies, hardiness characteristics, cognitive appraisal, personal control, attitude towards health, perceived competency and sensitivity to health professionals, and compliance with treatment regimes.

The literature does identify that women use different coping strategies than men. For example, men use more self-control (e.g., keeping their feelings to themselves) than women, while women use more positive appraisal than men. Men rely on spouses emotionally more than women, and the majority of informal social support is provided by women thus increasing women's social networks.

Folkman and Lazarus (1985) identified the different sources of stress between the genders that explains the differing ways in which men and women cope. McCrae (1999) reported a greater use of neurotic coping mechanisms by women (e.g., hostile reaction, expression of feelings, distraction, passivity, wishful thinking and sedation) when coping with a stressful situation. Although women reported more interpersonal coping problems than men, there was no evidence to suggest that the types of coping problems faced by women required less behavioral coping strategies than those experienced by men (Basford et al., 2001).

The influence of cultural norms and expectations should not be understated with respect to coping strategies deployed. The influences of religion, education, belief systems, and socialization all have an impact on the ways in which people cope. However, when one's ability to cope effectively with health is undermined, or limited, our social networks, or professional help are called upon to provide assistance and support. The nature of social support is varied and can in itself have a positive or negative health outcome depending on the type of support offered, the knowledge and skill of the provider and the context in which it is given and received.

Social Factors Affecting Coping

So far the discussion has centered on the individual's ability to engage with positive coping strategies. John Donne's (1624) famous quote "no man is an island" illustrates how man is intertwined within complex social networks and organizational structures (Lopez & Mermelstein, 1987). In maintaining our health we often need to interact with others in a reciprocal manner, often from a moral obligation and a sense of duty (Holahan et al., 1997). Supporting each other in times of crisis is a fundamental necessity that forms the basis of our intricate lives. (There are exceptions to this view as in the case of hermits.) It is a process that has responded and adapted to changes in the external environments, human behaviours, cultural norms, and values (Johnson, 1998; Lazarus & Folkman, 1984; Miller, 1983; Sugisawa et al., 1994). Social support, thus defined, has been fully recognized by the scientific community (Felton, 1990; Lanza & Revenson, 1993; Nissen & Newman, 1992) and investigations have focused on the context and constructs of social support and its associated relationship to health. Notwithstanding, others have found that not all social support has a positive health outcome (Antonucci, 1985; Coyne & Delongis, 1986; and Holahan et al., 1997). Such polarization of views serves to confuse, but it should be remembered the concept is complex and defies a universal explanation. Hutchison (1999) suggests the term is a *meta concept* and often misunderstood between what is meant by social support and social networks.

Social Support Versus Social Networks

Social support is associated with social networks and they are often intertwined for purposes of definition. However, Cohen and Wills (1985) clearly illustrated the fact that social support is more concerned with quality indicators or functional context of relationships, and social networks relate more specifically to number and various dimensions associated with these relationships. Laireiter and Baumann (1992) have fully recognized that social support is a complex matrix system and should be examined at each level and dimension within a given taxonomy. In this way, they assert, one might best identify and differentiate between the following categories:

- Social integration,
- Network resource,
- Supportive climate and environment,
- Received and enacted support, and
- Perception of being supported.

Cobb (1976) adds specificity to the definition in asserting that social support transmits information to the recipient that they are loved and valued in the context of a caring environment. The internalization of the experience has intricate and delicate manifestations in that perceptions of social support can only be an individualistic phenomenon (Lazarus & Folkman, 1984). Other features to be included in this debate are the intentions or behaviors of support given. Shumaker and Brownell (1984) illustrate this concept as being: "an exchange of resources between two individuals perceived by the provider or the recipient to be intended to enhance the well being of the recipient" (p. 13).

The very nature of the relationship between those who receive functional support and those who provide functional support frequently provides a focus for scientific enquiry.

Paradigmatically, a process of formal psychological analysis draws explanations from: (i) social exchange theory, (ii) social comparison theories, (iii) social learning theories and self-esteem theories (Sugisawa et al., 1994). These theories can offer insights into the nature of coping and attribution (Liebermann, 1986). For example, Bandura's (1986) Social Cognition theory suggests that there is a "reciprocal relationship between cognition and other personal factors, environment and behaviour" (p. 380). The underlying proposition behind these theories is that all these elements are inter-related and inter-dependent with individuals having the capacity to "use symbols, think about things before they happen, learn from personal and communal experiences and make decisions based on personal and communal standards and reflect on their past" (Bandura, 1986, p. 380). In other words, it is a problem-solving approach towards their unique situation. An individual's uniqueness is of utmost importance and must be considered in the context of the receiver and provider of social support.

Recipient Characteristics

Each individual has characteristics that are personal to them and will have an impact on the size of their social network, their willingness to receive support, and their ability to gain and sustain support through the duration of need. Personality traits and cultural and social roles influence these characteristics (Antonucci, 1985; Cohen & Syme, 1985; Penninx et al., 1997). For example, people who are well liked by others, and are less dogmatically independent, may well receive and accept the required support. On the one hand, independence can be seen as a positive coping behaviour, but, on the other hand, independency denies reciprocity and the health benefits received from supporting activities. For instance, people who are independent may be so distressed that they are unable to ask for support, or their actions may appear threatening to others so that support is not offered in the first place. Providing support for such individuals becomes problematic in the sense of assessing the benefits to health, and maintaining the respect of people's privacy and independence.

Measuring Social Support

Measuring social support from a health coping perspective is not an easy task; however, it has been commonly divided into categories such as: emotional, instrumental, informational, or sources such as family or friends (House & Khan, 1985; Veiel & Baumann, 1992). Others have included social integration that reflects both the structural and functional elements of support networks (Cutrona et al., 1986). Emotional support is a conceptual framework through which support is given that affords an exchange of information with an individual in a confidential manner. The provider of emotional support is someone who is trustworthy and sympathetic to one's situation. Emotional support can be either on an individual basis or within a group context (Solomon & Peterson, 1994). Informational support initiates the activity of others to gather information so that individuals can be enabled and empowered to cope with their dysfunctional capacity in a meaningful and informative way. Functional and instrumental support is thought to be an intrinsic aspect of social support through which individuals can better cope with everyday life activities (Cohen & Wills, 1985).

Empowerment as a Health Coping Strategy

A lot is said about empowerment as a conceptual framework. It is considered to be a process and a product (Basford et al., 2001). The philosophy of empowerment infers that individuals care about themselves, taking responsibility for their own health while making informed decisions (Byrne, 1998). Health education and health support systems are the main approaches used to empower people through increasing patient/client autonomy, expanding freedom of choice, knowledge, skills, and providing a heightened awareness of values and needs (McWilliam et al., 1997). Such ideologies can be achieved through the process of empowering others to gain information, solve problems, redefine needs and priorities, control extraneous stress, develop positive coping strategies, engage with social support, increase motivation and enhance self–awareness, enable individuals to increase their control over their health situation, and help to achieve and optimize their goals and aspirations. Empowerment through the educational process also assists patients and clients to examine the emotional, social cognitive and spiritual dimensions of their lives. Failing to empower people occurs when individuals assume a passive role as an *object* acted upon in their environment as opposed to *subject* acting in and on the situation, which ultimately leads to alienation (Friere, 1973). Maintaining health equilibrium is subjected to the fact that individuals must be able to bring about life changes, including their social and organizational context within which they live (Anderson et al., 1995).

Feste and Anderson (1995) recommended that an empowerment educational program should include strategies that enhance the well-being, self-image, motivation, adaptability, stress reduction, problem-solving abilities, and social support. Utilizing an empowerment model fosters improved assertiveness, positive coping behaviors and the ability to acknowledge denial (Arnold et al., 1995). Through the development of positive coping strategies individuals develop mastery with their situation. In essence, they come to terms with their health issues, which, in turn, contribute to their sense of wholeness and well-being. (McWilliam et al., 1997) suggests that the empowerment process promotes this holistic sense of self and is a positive coping strategy to which all should aspire.

Summary

Being presented with an FASD/FAE child is a traumatic experience that has far reaching effects on the mother, the child, and other family members. The child with FASD/FAE can be affected emotionally, socially, cognitively, and physically. Dealing with these health variables requires a holistic framework that so often orthodox medicine is able to meet. By better understanding health coping strategies that have an effect positively and negatively on health outcomes, health professionals can assist the mother, the affected child, and their family, to adopt positive coping strategies that help them prevent secondary and tertiary complications from occurring. Minimizing the effect of FASD/FAE will enable these children to reach their full development potential in a safe and loving environment, and this approach will maintain the health and well being of all members of the family unit.

In the broad sense, positive coping strategies are those that utilize the empowerment concept, education, social support, self-management, problem-solving abilities, self-worth, modifying behaviour, information seeking, accepting responsibility, positive reappraisal,

faith, compliance, mastery and self management. Negative coping strategies are associated with avoidance, catastrophising, blaming, denial, acceptance, resignation, social support, helplessness and withdrawing from other people.

These coping activities have been drawn from other scientific literature. It would be useful to seek out empirical knowledge in the ways in which mothers, families, and affected children cope with their stressful situation. The purpose would be to determine if there are positive coping strategies that can be usefully adopted in supporting others specific to the problem of dealing with FASD.

The preceding sections have provided an overview of the historical knowledge of the impacts of alcohol consumption during pregnancy on the fetus. We have also discussed different potential coping strategies within the theoretical frameworks in which such strategies are best understood. In addition, the diagnosis of FASD has been examined as have diagnostic tools utilized for assessing alcohol consumption and FASD screening. Difficulties in diagnosis identified in this discussion also have consequences for measurement of the true prevalence rate of FASD.

Chapter Seven:

Incidence, Birth Prevalence and Prevalence of FASD

Establishing the prevalence and incidence (or birth prevalence) of FAS is fraught with methodological and practical difficulties. The terms prevalence and incidence are themselves inconsistently applied in the literature. Difficulties are compounded for the less well defined fetal effects of alcohol consumption during pregnancy such as ARND. It is, therefore, exceedingly difficult to establish a clear picture of the situation. In the following, we review some of the issues and summarize the available results.

Definitions of Incidence, Birth Prevalence and Prevalence

The terms *incidence* and *prevalence* have not been used consistently in the FASD literature, and the two terms have sometimes been applied by different authors to designate the same quantity. The usual epidemiologic definition of incidence is the number of new cases in a specified population over a defined time period, and the definition of prevalence is the number of existing cases in a defined population at a specified point in time (Jewell, 2004). It is usually agreed that new cases of FAS or other fetal alcohol effects occur at birth, since the damage to the child is already done when he or she is born even though the symptoms may not be readily apparent. In the past, most authors have, therefore, designated the proportion of liveborn infants with FAS as the incidence of FAS (Habbick, 1996; Maillard et al., 1999; May et al., 1983; Sampson et al., 1997; Williams et al., 1999). May and Gossage (2001), however, argued that alcohol damage occurs before birth so that a fetus could be afflicted with FAS up to seven months prior to birth, and incidence could theoretically be measured before birth. They prefer the term “birth prevalence” to designate the proportion of cases among live born infants. In recent years, many authors have simply used the term *prevalence* to designate the number of cases among a birth cohort when diagnosis was performed at a point in time long after birth (CDC, 1997b; Fox & Druschel, 2003; Egeland et al., 1998; Miller et al., 2002). However, others have calculated prevalence in a population of children at a given point in time, which is the most common definition of prevalence. In this report we distinguish birth prevalence from prevalence in its usual sense. For the purpose of this review, we classify rate estimates based on the denominator that was used to compute them:

Birth prevalence: number of cases over the number of live births in a given time period. This is what some authors define as incidence.

Prevalence: number of cases over the current population examined, possibly restricted to a certain age range.

This perspective corresponds to the current use of these terms in the literature and allows a non-overlapping grouping of rate estimates. If one stratifies the population by birth year, then the difference between birth prevalence and age-specific prevalence will be small provided that nearly all children born in a given year are still alive today.

Difficulties in Estimating Current Prevalence and Birth Prevalence

Sampson et al. (1997) summarize the difficulties in estimating current prevalence and birth prevalence of FAS and ARND. First, while diagnostic criteria for FAS have been established, as discussed earlier, there is no objective laboratory test or validated checklist, and diagnosis remains based on the judgment of clinicians. Inconsistencies are, therefore, likely between clinicians with various levels of training and experience. Second, the timing of case ascertainment will affect the estimates. If diagnosis is made at birth, FAS facial features may not be apparent and neurobehavioral testing cannot be performed. Sampson et al. (1997) claim that the age range when diagnosis is easiest is between eight months and eight years. When ascertainment is performed several years after birth, the most severe cases of FAS may have died. Some cases will have moved outside the study area. Also, information on maternal alcohol consumption during pregnancy may not be available for children placed in foster care, or mothers may recall less accurately their alcohol use. Third, the method of ascertainment will affect the estimates.

The advantages and disadvantages of various methods of ascertainment have been most extensively reviewed by May and Gossage (2001). Passive surveillance methods take advantage of existing health care systems, programs and records such as birth certificates, birth defects registries and medical records to identify cases. They, therefore, offer a relatively inexpensive and easy approach but "lack the rigor and consistency of diagnosis of other systems" (May & Gossage, 2001, p.160). They are also vulnerable to low data quality. For example, Miller et al. (1995) did not even attempt to estimate FAS prevalence from their surveillance data because they did not feel their data were reliable. Clinic-based studies have the following advantages: they gather maternal history data, study a large number of pregnancies with various levels of alcohol exposure, the health services provided are an incentive for participants and the prospective nature of the design provides control and rigour in measuring the important variables. Diagnosis can be made blind to the alcohol exposure. Disadvantages are: the subjects are self-selected and may exclude women of highest risk, studies conducted in publicly funded hospitals and clinics over-represent disadvantaged populations, and children are examined at birth when FAS is hard to diagnose and cases can be missed. Active case ascertainment methods focus on "finding children with FAS at appropriate ages for accurate diagnosis . . . active, effective and comprehensive outreach in a population is most likely to uncover children with FAS and alcohol-abusing mothers at the highest risk", and "by studying entire communities or populations, this method can eliminate much selectivity" (May & Gossage, 2001, p.161). However, "such research is very labor intensive, time consuming and costly" (May & Gossage, 2001, p.161). It also requires the cooperation from many non-researchers in the study population, and failure to secure cooperation of some constituencies results in under estimation of prevalence at birth or in the current population. The studies exhibiting many of these drawbacks have mostly been conducted in high-risk populations and their findings do not generalize to more general populations. Passive surveillance and active case ascertainment have sometimes been combined: passive surveillance is used to screen for children potentially affected by alcohol use in pregnancy, and the screened children are then contacted and examined (e.g., Duimstra et al., 1993).

Clinic-based studies usually involve a preliminary screen of infants for characteristics associated with FAS such as growth retardation or a record of maternal alcohol abuse in order to limit the number of infants submitted to a full examination. Initial screening using surveillance records aims at the same goal. Some proportion of the screened infants will, however, not be examined for a number of reason, such as failure to locate them, lack of resources, or refusal of the parents. Researchers then know that the rate computed based on the cases they identified among the examined infants will be an underestimate. Nevertheless, they can project what the rate would be if they had examined all the screened infants by assuming the proportion of cases would be the same in the infants not seen as in the infants they examined (Sampson et al., 1997). An unadjusted, conservative estimate based only on the actual observed cases and a projected rate are then usually reported. The projected rate may itself be an underestimate if there is reason to believe that FAS cases are more likely not to be examined.

Estimates of Prevalence and Birth Prevalence of FAS

Estimates of FAS birth prevalence reported in studies included in this review are presented in Table 5. When applicable, both the unadjusted rate and the projected rate are reported. Most studies have been conducted in North America and many were conducted either in Aboriginal populations or in populations where a large proportion of FAS cases are Aboriginal. We, therefore, divided the Table in three sections, one for studies in Aboriginal populations, one for studies with large proportions of Aboriginal FAS cases, and one for studies with few Aboriginal FAS cases or conducted outside North America. Within these subdivisions, studies are grouped by method of ascertainment. Estimates of FAS prevalence are presented in Table 6. Studies have been conducted in North American Aboriginal populations and in South Africa and all used an active case ascertainment strategy.

Table 5
Birth Prevalence

Reference	Population	Type of study	Study period	Estimate (per 1,000 live births)	Unadjusted rate (per 1,000)	Denominator	Comment	Aboriginal cases
May, P.A. et al. (1983)	Navajo in Southwest US	Active case ascertainment in 1980-82	1968-1980	1.4	NA	unspecified	Number of births reconstructed from demographic data	All
May, P.A. et al. (1983)	Pueblo in Southwest US	Active case ascertainment in 1980-82	1968-1980	2.0	NA	unspecified	Number of births reconstructed from demographic data	All

Reference	Population	Type of study	Study period	Estimate (per 1,000 live births)	Unadjusted rate (per 1,000)	Denominator	Comment	Aboriginal cases
May, P.A. et al. (1983)	Plains Indians in Southwest US	Active case ascertainment in 1980-82	1968-1980	10.3	NA	unspecified	Number of births reconstructed from demographic data	All
Duimstra, C. et al. (1993)	Plains Indians in 4 South Dakota reserves	Surveillance + case ascertainment	1988-1989	8.5	3.9	1022	Only 46% of cases meeting the screening criteria were examined	All
Egeland, G.M. et al. (1998)	Alaska Natives	Surveillance from multiple sources	1977-1980	1.4	NA	7160		All
Egeland, G.M. et al. (1998)	Alaska Natives	Surveillance from multiple sources	1981-1984	3.8	NA	8971		All
Egeland, G.M. et al. (1998)	Alaska Natives	Surveillance from multiple sources	1985-1988	4.1	NA	10150		All
Egeland, G.M. et al. (1998)	Alaska Natives	Surveillance from multiple sources	1989-1992	2.5	NA	11065		All
Miller, L. et al. (2002)	Alaska Natives	Surveillance from multiple sources	1995-1997	5.6	NA	7117	FASSNet program	All
Miller, L. et al. (2002)	Arizona Native Americans	Surveillance from multiple sources	1995-1997	2.5	NA	15685	FASSNet program	All
Williams, R.J. et al. (1999)	Northeast Manitoba	Clinic-based, examination at 2 years of age	1994	14.8	7.2	745	Only 41 of the 90 cases meeting the screening criteria were examined	Large proportion
Habbick, B.F. et al. (1996)	Saskatch-ewan	Surveillance + case examination in 1992-94	1973-1977	0.52	NA	77670		Large proportion
Habbick, B.F. et al. (1996)	Saskatch-ewan	Surveillance + case examination in 1992-94	1978-1982	0.62	NA	85480		Large proportion
Habbick, B.F. et al. (1996)	Saskatch-ewan	Surveillance + case examination in 1992-94	1983-1987	0.61	NA	88520		Large proportion

Reference	Population	Type of study	Study period	Estimate (per 1,000 live births)	Unadjusted rate (per 1,000)	Denominator	Comment	Aboriginal cases
Habbick, B.F. et al. (1996)	Saskatch-ewan	Surveillance + case examination in 1992-94	1988-1992	0.59	NA	79800		Large proportion
Sampson, P.D. et al. (1997)	Cleveland, OH	Clinic-based, examination 72h after birth	1979-1981	4.6	3.0	8331	Not all screened infants seen	Unknown
Maillard, T. et al. (1999)	Reunion Island	Clinic-based, examination at birth	1996	1.8	NA	2778	More stringent FAS criteria than North American criteria	NA
Sampson, P.D. et al. (1997)	Seattle, WA	Clinic-based, examination shortly after birth	1974-1975	2.8	1.4	1439	Only 80 of the 204 cases meeting the screening criteria were examined	One out of two
Sampson, P.D. et al. (1997)	Roubaix, France	Clinic-based, examination shortly after birth	1977-1979, 1986-1990	1.3	NA	21402	More stringent FAS criteria than North American criteria	NA
CDC (1997b)	Atlanta, Georgia	Surveillance from multiple sources	1981-1989	0.10	NA	285538		Unknown
Mathis, M.P et al. (1995)	Atlanta, Georgia	Surveillance from multiple sources	1989-1992	0.23	NA	152000		Unknown
Fox, D.J. & Druschel, C.M. (2003)	Western New York State	Surveillance from multiple sources	1995-1998	0.37	NA	117860	FASSNet program	Small proportion
Miller, L. et al. (2002)	Western New York State Blacks	Surveillance from multiple sources	1995-1997	1.6	NA	13465	FASSNet program	None
Miller, L. et al. (2002)	Western New York State Whites non-Hispanic	Surveillance from multiple sources	1995-1997	0.3	NA	68932	FASSNet program	None
Egeland, G.M. et al. (1998)	Alaska Non-natives	Surveillance from multiple sources	1977-1980	0.1	NA	28092		None

Reference	Population	Type of study	Study period	Estimate (per 1,000 live births)	Unadjusted rate (per 1,000)	Denominator	Comment	Aboriginal cases
Egeland, G.M. et al. (1998)	Alaska Non-natives	Surveillance from multiple sources	1981-1984	0.1	NA	37301		None
Egeland, G.M. et al. (1998)	Alaska Non-natives	Surveillance from multiple sources	1985-1988	0.2	NA	38010		None
Egeland, G.M. et al. (1998)	Alaska Non-natives	Surveillance from multiple sources	1989-1992	0.3	NA	36016		None
Miller, L. et al. (2002)	Alaska Whites non-Hispanic	Surveillance from multiple sources	1995-1997	0.3	NA	19007	FASSNet program	None
Miller, L. et al. (2002)	Arizona Whites non-Hispanic	Surveillance from multiple sources	1995-1997	0.1	NA	114851	FASSNet program	None
Miller, L. et al. (2002)	Arizona Hispanic	Surveillance from multiple sources	1995-1997	0.3	NA	80628	FASSNet program	None
Miller, L. et al. (2002)	Denver Whites non-Hispanic	Surveillance from multiple sources	1995-1997	0.2	NA	63653	FASSNet program	None
Miller, L. et al. (2002)	Denver Hispanic	Surveillance from multiple sources	1995-1997	0.4	NA	21579	FASSNet program	None

Table 6
Prevalence

Reference	Population	Type of study	Time	Age	Estimate (per 1,000)	Denominator	Comment
May, P.A. et al. (1983)	Navajo in Southwestern US	Active case ascertainment in 1980-82	1979	0-14 years	1.6	unspecified	
May, P.A. et al. (1983)	Pueblo in Southwestern US	Active case ascertainment in 1980-82	1979	0-14 years	2.2	unspecified	
May, P.A. et al. (1983)	Plains Indians in Southwestern US	Active case ascertainment in 1980-82	1979	0-14 years	10.7	unspecified	
Robinson et al. (1987)	Aboriginal community in British Columbia	Active case ascertainment in 1984-85	1985	0-18 years	190	123	Includes FAS and FAE.

Reference	Population	Type of study	Time	Age	Estimate (per 1,000)	Denominator	Comment
May, P.A. et al. (2000)	Western Cape Province of South Africa	Active case ascertainment in first grade children	1997?	5-9 years	42.9	1,072	First graders
May, P.A. et al. (2000)	Western Cape Province of South Africa	Active case ascertainment in first grade children	1997?	6-7 years	40.5	< 1,072	First graders
May, P.A. et al. (2000)	Western Cape Province of South Africa	Active case ascertainment in first grade children	1997?	6-7 years	39.2	???	Community wide
Viljoen, D. et al. (2003)	Gauteng Province of South Africa	Active case ascertainment in first grade children	2000?	5-9 years	19	830	First graders

The Tables illustrate the difficulty of drawing a clear picture of the birth prevalence and current prevalence of FAS, because estimates for different populations have been obtained by different methods. Two surveillance-based studies (e.g., Egeland et al., 1998; Habbick et al., 1996) reported birth prevalence rates over multiple time periods. Based on their reported figures, Habbick et al. (1996) argued that the birth prevalence of FAS in Saskatchewan remained unchanged over a 20-year period. However, for both studies, data collection was performed in the last few years of the period covered instead of being collected consistently over the entire period, which casts a doubt on the comparability of the figures for different time periods. Cases born in the earlier years are more likely to have died or moved away before being recognized and cases born in the later years may not have been diagnosed yet.

The results of surveillance in Alaska reported by Egeland et al. (1998) and Miller et al. (2002) offer a direct comparison between Alaska Native and Non-native populations using a consistent FAS case definition and reveal rates 5 to 30 times higher in Alaska Natives. Miller et al. (2002) also report results for Arizona, where prevalence in Native Americans is about 20 times higher than in Whites. Habbick et al. (1996) did not have access to numbers of births broken down by ethnicity but noted that 86% of the FAS patients in Saskatchewan were Aboriginal while Aboriginal births account for approximately 15-20% of births in Saskatchewan, suggesting a much higher birth prevalence among the Aboriginal communities than in other groups. May et al. (1983) studied Native Americans and also reconstructed birth prevalence retrospectively, but contrary to Habbick et al. (1996) and Egeland et al. (1998) they did not track FAS cases that had died prior to the study. This fact may explain why their incidence (birth prevalence) rates are lower than the prevalence rates that they also computed. The merit of the study by May et al. (1983) is to highlight that the birth prevalence differs between Native American cultures, with the Plains Indians exhibiting much higher rates than Navajo and Pueblo communities.

The FASSNet surveillance program in four US states also provides FAS birth prevalence estimates broken down by White non-Hispanic, Black and Hispanic ethnicity, allowing comparisons (Miller et al., 2002). In western New York State, the FAS birth

prevalence in Blacks is about 5 times higher than the rate in Whites non-Hispanic, while in Arizona and in the Denver area, the FAS birth prevalence in Hispanics is 2-3 times higher than in Whites non-Hispanic.

Clinic-based studies have produced a range of birth prevalence rates depending on the setting of the study. Williams et al. (1999) reported a particularly high rate of 14.8 per 1,000 live births in a mostly Aboriginal population in Northeastern Manitoba. Birth prevalence rates estimated using the FAS definition developed in France are not directly comparable to birth prevalence estimated using North American FAS definitions, the French definition being more stringent.

Recent prevalence studies conducted in first grade students in South Africa revealed prevalence rates much higher than those generally observed in North America. The prevalence was particularly high in the wine growing region of Western Cape, but was still considerable outside the wine growing regions (May et al., 2000).

An even higher prevalence of 190 per 1,000 for FAS and FAE combined was reported by Robinson et al. (1987) in an Aboriginal community in British Columbia with high prevalence of alcohol abuse. It is important to note, however, that such high-risk communities do not represent the entire North-American Aboriginal population. Further discussion of cultural-specific difficulties in FASD research will follow in a later section addressing culture specifically.

Estimates of the Prevalence of Other Alcohol Related Effects

The difficulties in establishing consistent diagnoses are even greater when it comes to adverse effects of alcohol use in pregnancy less severe than full FAS. Neurodevelopmental deficits cannot be diagnosed at birth and may only become measurable when the child is a few years old. Birth defects and neurodevelopmental deficits are not necessarily specific to alcohol exposure. An additional difficulty is to link the occurrence of these deficits to alcohol exposure in pregnancy. The only rigorous effort that we have found in the literature is with ARND in a prospective study in Seattle (Sampson et al., 1997). These researchers conducted five waves of psychometric observations from birth to age seven years on 581 children exposed to alcohol in pregnancy out of a cohort of 1,439. This assessment gave them a total of 474 neurobehavioural outcome measurements on each child. These measurements were divided into 15 blocks, and for each block the researchers extracted a combination of measurements that correlated best with a series of measures of alcohol consumption during pregnancy, to define composite neurobehavioural variables that were most likely to be related to alcohol exposure. Next, a score was computed for each of the children on each of the composite outcomes and on an alcohol exposure composite variable. The researchers then identified 11 children who were below average for all composite neurobehavioural variables and also in the top 7.5 percentiles of the alcohol score. Classifying these children as “true ARND,” adding the FAS cases and adjusting for the under ascertainment of alcohol exposed children, they estimated a combined FAS and ARND prevalence of 9.1 per 1,000.

Drinking Behavior Among Women: Prevalence, Individual Characteristics, and Causal Factors

According to Centers for Disease Control and Prevention's (1997b) Behavioral Risk Factor Surveillance System, 51% of childbearing age women (18-44 years) in the U.S. reported consuming alcohol in the month prior to the survey. According to another U.S. study (Kaskutas, Greenfield, Mija, & Cote, 1998), while 61% women went alcohol free once they realized they were pregnant (80% had decreased their drinking), 39% of pregnant women continued to drink during pregnancy from their pre-pregnancy days. Although from separate studies, if these findings were taken together, these results translate into roughly 20% (39% of 51) of all childbearing age women drinking during pregnancy. The drinking prevalence among 20% women has also been reported by Bowden and Rust (2000), and Handmaker, Miller, and Manicke (1999).

Demographic and Behavioral Characteristics

Women of childbearing age who drink: According to one U.S. study, women who are likely to drink frequently (six drinks or more per week) during the periconceptional period, are those women who are unmarried, smoke, White non-Hispanic, 25 years of age or older, or college educated (Floyd, Decoufle, & Hungerford, 1999).

Women who continued to drink during pregnancy: There seems to be two distinct profiles of at-risk women. Waterson and Murray-Lyon (1990) found most at-risk mothers for prenatal alcohol consumption to be older, multiparous, poorly educated, African American and smokers with a high incidence of divorce and separation. While in another study by Mills and Graubard (1987), similar behaviour was displayed by a very different group of women: mothers who are of European descent, young, and highly educated. Similar descriptions have been reported by Serdula, Williamson, Kendrick, Anda, and Byers (1991), Oklahoma State Department of Health (1991), and Floyd et al. (1999). This two-cluster profile of at-risk women is also revealed by a Canadian report as discussed later in the report.

Women who stopped drinking during pregnancy: Ockene, Ma, Zapka, Pbert, Goins, and Stoddard (2002) concluded that women who were among an ethnically diverse, largely unmarried pregnant women, Hispanic ethnicity, younger age, and who had more social support to quit smoking were related to spontaneous alcohol abstinence.

Adolescents who continue to drink during pregnancy: According to a study conducted by Wiemann and Berenson (1998) among pregnant adolescents (below 17 years old), those who continued to drink during pregnancy were more likely to be Mexican-Americans, have quit school, report recent tobacco use, have a partner who drinks, and will have used alcohol during sexual activities. Adolescents who stopped using alcohol during pregnancy were significantly more likely to have witnessed or been a victim, or known a victim, of violence.

Prevalence of Drinking Behaviors and Characteristics of Drinkers in Canada:

Findings reported in this section were extracted from Alberta Alcohol and Drug Abuse Commission (2004) report. According to recent Canada census information, there were around 622,000 women in Alberta aged 18 to 44 in 2001 (Statistics Canada, 2002a). According to the Canadian Community Health Survey (Statistics Canada, 2002b), 69.3% of

Alberta women who were pregnant at the time of the survey reported drinking alcohol within the last 12 months, versus 80.4% of who were not pregnant. At the national level, these numbers were 72.8% 82.3% respectively. Similarly, fewer women pregnant at the time of the survey drank at least once per week (11.1%), indulged in binge drinking once or more per month (10.3%), or smoked daily or occasionally (15.7%) than women who were not pregnant (17.6%, 18.4%, and 30.8% respectively). Similar trends were true at the national level.

Around 9% Alberta women (13.7% in Canada) stated that they drank during their last pregnancy. Women with higher incomes ($60,000 or more) were much more likely to report using alcohol during their last pregnancy than women in the lower income brackets (Statistics Canada, 2002b). Alcohol use during pregnancy was reported by 7.5% of mothers with higher percentage of mothers likely to be younger (12 to 20 years old) rather than older, 40 years and older (Alberta Health and Wellness & Alberta Medical Association, 2002).

Surprisingly, heavy drinking (i.e., regular drinking of more than 12 drinks per week) was more likely to be reported by pregnant women in Alberta (12.1%, 6.9% for all Canada) than those who were not pregnant (10.4%, 8.6% for all Canada). No clear age or income differences were found in heavy drinking habits (Statistics Canada, 2002b).

Women in the youngest (18 to 20 years) and the oldest (31 to 44 years) age groups drank with higher frequency that those in between. The youngest group also reported to binge drink most frequently. When income levels are considered, the poorest women (i.e., those with less than $10,000 per year) drank less frequently, but they were more likely to binge when they did drink. These findings on frequency and higher binge drinking among select groups are consistent with other research on drinking patterns, including US studies (Statistics Canada, 2002b).

Few Alberta women reported extreme life, health or work stress. Further, pregnant women in Alberta (also true across all Canada) seemed to receive strong emotional and instrumental social support and health care when needed (Statistics Canada, 2002b). However, around 23.1% Alberta women (20.8% across all Canada) reported being depressed for at least two weeks in the past year. Women with low income and high-risk drinking habits were more likely to report depression (Statistics Canada, 2002b).

Pregnant women in northern and central Alberta were more likely to smoke than those in southern Alberta. However, the rates between the two regions were similar for rates of risky alcohol use and drug dependence. See Table 7 for more details (Northern and Central Alberta Perinatal Outreach Program, 2003; Southern Alberta Perinatal Outreach Program, 2003).

Table 7
Comparison between Northern and Southern Alberta

	Northern and Central Alberta (Northern and Central Alberta Perinatal Outreach Program, 2003)	Southern Alberta (Southern Alberta Perinatal Outreach Program, 2003).
Number of live births in 2001	21,492	16,412
% of women having live births and stillbirths to have smoked anytime during pregnancy.	Live births: 26% Stillbirths: 31%	Live births: 18% Stillbirths: 26%
Number and % of mothers identified for risky alcohol use (defined as three or more drinks on any one occasion during pregnancy, or one or more drinks per day throughout pregnancy).	418, 2%	217, 1.3%
% of women having stillbirths identified as using alcohol.	4.8%	Data not available due to very small numbers
% of women having live births and stillbirths identified as drug dependent.	Live births: 1% Stillbirths: <3%	Live births: <1% Stillbirths: Data not available

Why do Pregnant Women Drink Alcohol?

Since a sizeable portion of women drink during pregnancy, it is critical to understand the reasons behind their behaviour. According to the Best Start (2003), pregnant women drink due to many factors.

Women may be unaware that they are pregnant. They may not realize they are pregnant until the fourth week (and many in the sixth week) of their pregnancy. Until they realize they are pregnant, they continue to drink alcohol. This behaviour could negatively influence the baby's health (Floyd et al., 1999). Some women also report misperceptions about how much alcohol intake during pregnancy is acceptable and how much harm alcohol can cause. According to one Danish study (Kesmodel & Kesmodel, 2002), 76% of the women considered some alcohol intake during pregnancy to be acceptable, mostly on a weekly level. These misperceptions may be fuelled by the conflicting messages women are getting from their physicians.

Women may be drinking due to social reasons. Many women carry faulty social norms that drinking during pregnancy is widespread, and that it is okay to drink during pregnancy. Various reasons may be shaping these normative perceptions. They may personally know women who drank during pregnancy and who have children who appear outwardly to be healthy. Pregnant women may also drink because they believe the only way to socialize with their friends and participate in social events is by consuming alcohol. Drinking as a social activity and difficulty in resisting social pressure to drink was associated with drinking during pregnancy among adult women (Testa & Leonard, 1995), and among at-risk youth in American Indian populations (Ma, Toubbeh, Cline, & Chisholm, 1998a).

Finally, women may be drinking due to many psychological and environmental factors. These factors include: alcohol consumption due to addiction, alcohol as a coping mechanism (Best Start, 2003, discusses use of alcohol as a way "to cope with difficult life

situations such as poverty, violence, isolation, despair or depression" [p. 5]), lack of social support, family history of alcohol problems and partner's use of alcohol (Chang, Goetz, Wilkins-Haug, & Berman, 2000; Ockene et al., 2002). Partner's drinking habits seem to be directly related with women's habits. However, Environics Research Group study (2000) revealed that males carry several misperceptions about consequences of alcohol consumption especially during pregnancy.

From the literature review, it is less clear, and it is less likely that all women drink for all these reasons listed previously. We can only guess what groups drink for what reasons (as discussed later in the report). For now, we generally conclude that women drink during pregnancy due to either lack of awareness of alcohol consequences, due to faulty social norms, and due to certain psychological and environmental factors

Adolescents May Also Need Attention

Youth may be contributing to the FAS/FAE problem due to their misperceptions about risks of consuming alcohol during pregnancy. MacKinnon, Williams-Avery, and Pentz' (1995) study of 13-20 year olds in the US revealed that males and younger adolescents had lower perceptions about risks of alcohol. Further, 25% of high school students and 13.7% of college students reported that it was okay for a woman to drink heavily on one occasion during pregnancy.

Summary

Surveillance systems in the United States have generally reported birth prevalence rates of FAS of 0.1 to 0.7 per 1,000 live births for general populations but likely suffer from under reporting. Clinic-based studies and studies conducting an active ascertainment of FAS cases have been conducted in selective groups and FAS birth prevalence rates estimated from these studies are not representative of entire populations. This situation is particularly true of studies in Aboriginal populations that tend to target high-risk communities, yielding high incidence and prevalence estimates. Direct comparison of surveillance data in Alaska and Arizona provides the best evidence of an increased rate in Aboriginal populations (1 to 4 per 1,000 live births, a 5 to 30 fold increase compared to non-Natives). Surveillance in the United States also indicates a 5-fold rate increase in Blacks and a 2-3 fold rate increase in Hispanics compared to White non-Hispanics. Recent prevalence studies in South Africa have revealed the seriousness of the FAS problem in that country, with rates of 20-40 per 1,000 children. Estimating the prevalence of Fetal Alcohol Effects lesser than full FAS is further complicated by the difficulty to link the defects to alcohol exposure. A careful analysis of data from a seven-year follow-up of children's mental development in Seattle produced a combined prevalence estimate for FAS and ARND of 9 per 1,000.

Around 10-20% women of childbearing age drink during pregnancy. Women who are more likely to drink during pregnancy include young women, women with low education and income, and women with high-income and education. Lack of awareness of alcohol consequences, faulty social norms, and psychological, structural and environmental factors influence drinking behaviors among these women. In addition to women of childbearing age, it would be beneficial to rectify misperceptions of alcohol risks among young adolescents. The reasons that pregnant women continue to drink is an important one for later research and

these reasons along with the true prevalence of fetal alcohol effects have the ramifications on policy being formulated to address FASD-related concerns. Such policy addressing FASD is discussed next.

Chapter Eight:

Policy

The best policy for dealing with FASD is clearly prevention of alcohol consumption by pregnant women. However, much of the policy that is used to address the problem of alcohol consumption during pregnancy is based on broad-based alcohol policy generally and is not specific to the problem of drinking during pregnancy and FASD or even directed at women who drink. That said, however, much of the extant policy is still extrapolated for use in prevention programs directed at pregnant women who drink, although with only limited success. General alcohol policy, therefore, is included in this discussion as is policy specifically targeting those most at risk for producing an FASD child.

According to the 2004 Global World Health report (WHO, 2004), alcohol policy falls into three groups that are roughly analogous to the three stages of prevention. The first group of policy options is population-based. Similar to primary prevention, these policies are aimed at changing alcohol consumption at the macro level and consist of broad-based programs such as taxation and sale regulation (i.e., amount, availability, drinking location, age restrictions, and education programs). The second group of policies is comparable to secondary prevention in that they are aimed at specific alcohol-related problems and are focused and less likely to affect the non-problem drinker. An example of this type of policy are the drunk-driving programs that promote widespread random breath testing. Lastly, the third group of policies involves interventions aimed at individual drinkers who have already demonstrated difficulties, which again equates to tertiary prevention practices that address the individual in an effort to ameliorate any further difficulties in people who already are affected by drinking through treatment and rehabilitation programs.

To formulate effective policy much of what has been discussed earlier in this report will have to first be resolved. It is virtually impossible to know what policies can be or should be formulated without a thorough understanding of the true incidence and prevalence of FASD and these rates cannot be known as long as there are discrepancies among reporting practices or even among definitions.

However, even with difficulties with known prevalence or definitions, basic conclusions can and have been drawn from the literature to inform public policy. Several are cited by Barbor in a 2002 review, including that alcohol problems are highly correlated to per capital consumption; therefore, policy to reduce consumption will decrease problems related to alcohol. Barbor also notes that some literature suggests demand for alcohol is responsive to price so that price increases will correlate to declines in demand and that this also applies to heavy drinkers. Policies that affect drinking patterns through limiting access through age restrictions or geographical sale restrictions have also been shown to be effective (Barbor). Legislative interventions to reduce permitted blood alcohol levels for drivers, to increase minimum dinking age and control outlet density have also been effective in lowering alcohol related problems (Barbor). Individual approaches such as school- based intervention programs are less efficacious in curbing alcohol-related difficulties (Barbor). General alcohol policies addressing practices discussed previously can be effective in curbing problem drinking behaviour but specific policies addressing alcohol consumption during

pregnancy, when they exist at all, have been proven in this review to often be contradictory, difficult to enact, and at times even ethically suspect. An example of such broad-based policy can be seen in both Canada and the United States. Each country has policy regarding the posting of warnings about the dangers of consuming alcohol during pregnancy; Canada has warnings displayed at retail liquor outlets while the United States has warning labels on alcohol beverage containers (Miers, 1999). But, as discussed earlier, such programs have proven less than effective. Canada also has judicial guidance on recognizing FAS as a mitigating factor in sentencing and disposition (Miers, 1999).

Globally, policy regarding alcohol consumption during pregnancy is varied. Most countries prescribe limited consumption during pregnancy but the amounts vary from a high of five drinks per day being considered "safe" to UK policy suggesting one or two drinks per day being safe, to the North American recommendations of zero consumption during pregnancy. As far back as 1989, the Surgeon General of the United States advised against alcohol consumption during pregnancy (Colmorgen, 1986). The difficulty surrounding "safe" consumption levels is the lack of knowledge of specific mechanisms and timing of alcohol effects on the fetus. Some writers argue that it is better to recommend zero consumption to be safe while others argue such policies are akin to 'crying wolf' and may simply result in unnecessary stress and worry being placed on responsible women who may have consumed limited amounts of alcohol during pregnancy (Daniels & Golden, 2000). Beyond broad policy recommendations regarding alcohol consumption, there are more specific policy recommendations regarding such aspects of alcohol consumption during pregnancy such as development of prevention programs or policy directing service delivery but some countries are much further ahead than others in creating such policies.

In the United States, the Health Professions Education Partnerships Act containing the FAS Prevention and Services Program was passed in 1998. This Act was a starting point for development of a nationwide strategy to provide effective prevention, intervention, and service delivery programs (Mitchell, 1999). Canada has also developed programs to assist in creating effective policy but a 1999 report suggests Australia is lagging behind, with no agencies in Southern Australia identified as having a specific policy relating to the prevention of FASD. Countries need to develop policy regarding alcohol consumption during pregnancy as well as regarding development of prevention and service programs but a global public health message is also needed (Parry, 2000).

Often, alcohol policy takes the form of laws enacted to control those who consume alcohol through punitive measures. Such programs are seen more often in the United States than in Canada. One report suggests that at least 200 women in more than 30 states in the U.S. have been arrested and charged for drug or alcohol abuse during pregnancy (SAMHSA, 1996). Policies that prescribe punitive measures such as incarceration or the involuntary commitment of pregnant women who use alcohol or drugs during pregnancy are difficult to enact and may be unethical. Most of these laws use existing child abuse statutes and argue that by consuming alcohol a woman is putting her unborn child at risk of damage and is, in fact, abusing that child (Blank, 1996; Zeller, 1998) Because the exact mechanisms of alcohol effects on the fetus are unknown and, indeed, paternal effects have been proven to contribute somewhat, it is difficult to argue conclusively that any given woman who consumes alcohol is definitely causing damage to her unborn child. Typically, women who are charged tend to

come from a lower socioeconomic lifestyle; therefore, it becomes questionable practice to charge one pregnant woman for consuming alcohol and not all pregnant women who consume alcohol (Blank, 1996; Deville & Kopelman, 1998). Further, given the unknown contribution of paternal effects, such policies clearly show gender bias against women (Deville & Kopelman, 1999). Unfortunately, such policies, which provide punitive consequences for women who are identified as consuming alcohol during pregnancy, may lead to women not seeking care during pregnancy or aborting their pregnancy rather than risk criminal sanctions (Deville & Kopelman, 1998; Pittman, 1991; SAMHSA, 1996). Rather than implementing punitive approaches to preventing alcohol consumption during pregnancy, an environmental approach encompassing various means of restricted availability to alcohol may well be more efficacious (American Medical Association, 2002; Frohna et al., 1999).

In Canada, different provinces have attempted to address the problem of creating effective policy. The four western provinces and three territories have formed a partnership (Prairie Northern FAS Partnership), which is designed to support a coordinated strategic, women-centered plan to address FASD issues (Greaves et al., 2002). British Columbia is perhaps the leader with multi-agency task forces being created to develop strategic plans to best inform alcohol policy. In 1992, representatives from various government and health services came together to begin formulation of a strategic plan to address issues related to maternal substance use. Formulation of this group was the first step towards creating policy addressing FASD. The goals of the strategic plan were to: identify and reduce behaviors that created risks for parents and their children; reduce risk factors that compound the effects of alcohol and drug use during pregnancy (e.g., family violence, poverty, poor nutrition); improve and increase early identification of those affected by FASD; develop and support appropriate intervention programs; ensure educational strategies were targeted and the efficacy of these strategies; and finally, contribute to the development of supportive environments through public policy conducive to good health for children, families and communities (BC Community Action Guide, 1998).

The next step was to create policy recommendations. Their recommendations promote the development of policies to: address the social determinants that place women at risk for alcohol use through awareness and education campaigns; ensure training on diagnosis and best practice is in place for all health care professionals; insure that any policy that may increase barriers to access care is changed to reduce those barriers; enhance accessibility and suitability of current programs relevant to the comprehensive needs of all affected by FASD (children, youth, adults, family and support systems; and finally, work with provincial and federal ministries to share knowledge and co-ordinate funding and implementation of initiatives (BC Ministry for Children and Families, 1998, 2003). In Alberta, policy guidelines are increasingly being directed by gender and social influences on issues of substance abuse and treatment. It is becoming increasingly recognized that men and women have different drinking patterns, reasons for drinking, and alcohol impacts each differently (Poole, 2000). Research by the Alberta Alcohol and Drug Abuse Commission (AADAC) has culminated in a report that could lead to policy that is more responsive to the needs of pregnant women. For example, AADAC (2004) found substance abuse by women in their childbearing years is common as risk pattern drinking. A profile emerged of groups at higher risk but women are clearly open to changes in substance abuse behavior during pregnancy. Therefore, policy directing routine screening of all women of childbearing age

along with public awareness campaigns both broad based but also targeted at high risk groups both before and during pregnancy along with specific, focused support is recommended (AADAC).

Summary

The triangulation between models of best practice and education are intrinsically linked to policy directives. Equally, policy directives are informed by the scientific community. In the absence of empirical evidence emerging from the scientific communities, Policy makers have to make stabs in the dark. On reviewing the policies relating to FASD/FAE, it is clear there is no universal approach or dictate, and some, are dichotomously opposed from each other and sometimes ethically suspect. For instance, in Canada and the United States, warnings are posted that alert the public to the dangers of consuming alcohol during pregnancy. Such approaches have proven to be ineffective in the long run so only afford a minimal transference of knowledge to the population at large. In addition, both of these countries advice zero alcohol consumption during pregnancy whereas, other countries such as the United Kingdom suggest that one or two drinks per day is safe. The difficulty with offering safe levels of consumption clearly hinges on the lack of empirical knowledge that highlights *acceptabl*e threshold levels. One hypothesis suggests that there is a co-relationship with heavy drinking on a regular basis and binge drinking and FASD/FAE. But, these hypotheses are yet to be vindicated.

Often alcohol policy takes the form of laws that encourage punitive, and sometimes discriminatory, measures. Such models encourage a blame culture, which suggests women are solely at fault, and society, and the societal acceptance of alcohol consumption, has no part to play in the situation. Nor is there acknowledgement that one's environment is a compounding factor. Punitive models also challenge the rights of women and broader ethical considerations. Furthermore, these approaches serve to increase the levels of abortions and the women seeking health care during the antenatal period.

Worldwide policy needs to become consistent and clearly, the best policy recommendations regarding drinking by pregnant women should be geared toward prevention of FASD through abstinence. Various primary prevention initiatives have been pursued to work toward the goal of abstinence, among these, the US Government beverage labeling policy that requires warning labels on alcohol containers. The punitive approach has been followed in some jurisdictions, advocating criminal sanctions against women who drink during pregnancy. However, these polcies have not proven to be effective to date.

Chapter Nine:

Risk Factors for FASD and Alcohol Use

Certain groups appear to be more at risk than others, notably in remote, rural, and some Aboriginal communities, the incidence rates for FASD have been estimated as being higher than the rate for Canada as a whole. There is evidence that the rates are also higher in poor, inner-city communities. Others at risk include women with alcohol and drug-abuse problems. Women in lower socioeconomic groups may also be at somewhat higher risk, as the health of their offspring may be further compromised by poor nutrition and living conditions. Ways of reaching and influencing behaviour change among particular at-risk groups is needed. This need requires developing awareness, education, and support programs that are built on an understanding of the particular social, economic, health, geographic and other circumstances and needs of these groups, and utilizing the most effective and culturally appropriate communications channels and messages including families with FASD, those with FASD, Aboriginals, women in the correctional system, business and professional women, women in armed forces, lower SES women, sexually- or physically-abused women, women with other substance use problems.

Studies of the risk factors for FASD have either focused on the clinically specific and more extreme outcome of FAS or focused on a range of physical and neurodevelopmental outcomes in infants that are suspected to be affected by alcohol consumption. The methodological approaches taken to determine risk factors differ depending on the type of outcome.

Studies of Risk Factors for FAS

We have found only five studies of risk factors where FAS was the main outcome. Four of these studies have adopted a retrospective design (Bingol et al., 1987; Burd et al., 1996; Miller et al., 1995; Viljoen et al., 2002) and two are based solely on surveillance data (Burd et al., 1996; Miller et al., 1995). In retrospective studies, the exposure information may be collected several years after the pregnancy and this situation tends to lower its reliability. Recall biases may also occur when the information quality differs between the FAS cases and their mothers on the one hand, and the control infants and their mothers on the other hand. With surveillance systems, the quality and consistency of data can be low. The Cleveland NIAAA Fetal Alcohol Study (Sokol, 1981; Sokol et al., 1986) is the lone prospective study of FAS risk factors that we have found. Sokol et al. (1981, 1986) followed 8,331 pregnancies and identified 25 FAS cases. These cases were then compared to 50 controls matched on certain characteristics under a nested case-control study design. Two of the retrospective studies have a matched case-control study design (Burd et al., 1996; Viljoen et al., 2002) while Bingol et al. (1987) compared alcoholic mothers of upper-middle class and low socioeconomic status. Even with a case-control design or with samples of alcoholic mothers the number of FAS cases in these studies remains small, between 25 and 55. Sokol et al. (1986) also distinguish themselves by having performed a multivariate analysis where the effect of each risk factor is adjusted for other variables. The other reports only contain univariate analyses, where the association reported with a variable could be due to confounding by another variable.

Alcohol abuse by the mothers of FAS infants was found in all studies. Alcohol consumption being necessary for FAS to occur, the authors of the studies attribute to a denial of alcohol problems the instances where mothers of FAS infants had reported no alcohol consumption during pregnancy. Mothers of FAS infants also tended to report relatives with alcohol problems more often than mothers of controls in the study by Viljoen et al. (2002).

Given that a relatively small proportion of alcohol abusing mothers give birth to FAS infants, researchers have tried to uncover other factors that aggravate or mitigate the effects of alcohol abuse in pregnancy. Characteristics associated with low socioeconomic status such as low education (Miller et al., 1995; Viljoen et al., 2002), being unmarried (Burd et al., 1996; Miller et al., 1995), and Black race (Miller et al., 1995; Sokol et al., 1986) were found more often in FAS case mothers than in control mothers. Similarly, Bingol et al. (1987) reported a proportion of FAS of 40.5% (FAS+FAE: 71%) in the children of alcoholic mothers of low socioeconomic status but only 1% (FAS+FAE: 4.6%) in the children of upper middle-class alcoholic mothers. Black race could be the actual risk factor, but in the studies in the United States it is too strongly associated with low socioeconomic status to be able to tell them appart. The upper-middle class sample of Bingol et al. (1987) was entirely White, while the low socio-economic sample was almost exclusively Black and Hispanic. In the studies where controls were matched on ethnicity with the cases, one involving coloured South Africans (Viljoen et al., 2002) and another involving Whites and Native Americans (Burd et al., 1996), markers of socioeconomic status were nonetheless lower for cases than controls.

High numbers of pregnancies (Miller et al., 1995) and older age (Burd et al., 1996; Miller et al., 1995; Sokol et al., 1986; Viljoen et al., 2002) have been found to be associated with FAS. The correlation between age and number of pregnancies (parity) has prevented researchers from determining based on epidemiologic data whether physical changes due to aging or to repeated pregnancy are responsible for the increase in risk.

Viljoen et al. (2002) and Miller et al. (1995) also reported higher levels of smoking during pregnancy and Viljoen et al. (2002) reported lower religiosity in the mothers of FAS cases compared to the mothers of controls. These comparisons were made without adjusting for alcohol consumption. Since smoking and low religiosity are likely to be associated with alcohol consumption, it is not possible to tell whether these variables are independently associated with FAS based on the results presented. Matching of cases and controls on maternal smoking status prevented Sokol et al. (1986) from examining association with smoking in the Cleveland study.

Studies of Risk Factors for Other Fetal Alcohol Effects

While the full expression of FAS is rare, less severe adverse fetal effects of alcohol consumption during pregnancy, including physical features such as birth weight, birth length, head circumference, intrauterine growth retardation (IUGR, usually defined as a birth weight in the lowest 10 percentiles for gestational age), presence of congenital abnormalities and neurodevelopmental delay have been observed at high frequencies in the offspring of alcoholic mothers and are suspected to occur at lower levels of alcohol exposure. The higher frequency of these adverse effects permits studying risk factors prospectively in samples

exhibiting a wider range of alcohol consumption during pregnancy. All studies mentioned in this section are prospective unless noted otherwise.

Unlike FAS, an adverse outcome for each feature taken individually is not specific to alcohol consumption during pregnancy, and the first goal of research has been to determine whether alcohol use in pregnancy is actually a risk factor. In the analysis of the studies mentioned here, researchers have adjusted their estimates of alcohol effects for potential confounding factors. We found that results regarding the effect of alcohol on certain outcomes are contradictory. Bingol et al. (1987) found (retrospectively) lower birth weights, birth length and head circumference and Sokol et al. (1980) found lower birth weights and a higher proportion of IUGR in babies of alcoholic mothers compared to babies of nonalcoholic mothers, taking race into account. McCarver et al. (1997) and Jacobson et al. (2002) found associations between alcohol consumption and birth weight, birth length and head circumference for levels as low as 0.01 to 0.5 oz per day in African-American samples, and in the Loma Linda NIAAA Fetal Alcohol Study (Sokol et al., 1986) frequency of beer drinking contributed to a decrease in birth weight. By contrast, other studies found no significant association between low to moderate alcohol use and birth weight, birth length and head circumference (Walpole et al., 1990) or occurrence of low birth weight (Lundsberg et al., 1997). For IUGR, Yang et al. (2001) reported no association with alcohol consumption in a case-control study while Lundsberg et al. (1997) reported a significant protective effect of low alcohol consumption (0.01 to 0.25 oz per day), and no effect at moderate alcohol consumption levels. These finding were obtained in a large US cohort of 2,714 after adjusting for smoking, ethnicity, infant sex, mother weight, height, parity, exercise in third trimester, employment and obstetric complications. Socioeconomic status is clearly a potential confounder here, being associated with both higher birth weight and alcohol consumption during pregnancy (see Section XXX), but that confounding effect should be attenuated by the adjustment made for ethnicity and employment.

The Bayley Scales of Infant Development were used in four studies. McCarver et al. (1997) and Jacobson et al. (2002) reported a decrease in the mental development index score at 12-13 months with alcohol use during pregnancy in African-Americans while Forrest & Florey (1991) found no association between the mental and psychomotor development index scores and alcohol drinking during pregnancy in a UK sample. These authors also reported that drinking before and after pregnancy were associated with increased scores, hinting that maybe their adjustment for socioeconomic status has not removed all confounding variables. Coles et al. (2000) reported that children of women with heavy episodic drinking showed no pattern of neurodevelopmental effects throughout the first year compared to controls but did find neurodevelopmental delay in children whose mother scored high on a risk index summarizing alcohol use prior to and during pregnancy. Investigators in Seattle examined the neurological development of children selected to cover a wide range of prenatal alcohol exposure over a much longer period of 14 years (Streissguth et al., 1993). They observed a dose-response relationship between prenatal alcohol exposure and adolescent behavior and learning problems (Olson, et al., 1997) that indicated the persistence of developmental delays observed at younger ages (Streissguth et al., 1993). These findings imply that subtle neurodevelopmental effects are present in the offspring of light to moderate drinkers.

Sokol et al. (1980) examined a range of other neonatal outcomes and found a significantly higher prevalence of abnormal oral cavity, genitourinary abnormalities and hypospadias in children of alcoholic mothers compared to children of nonalcoholic mothers. Rostand et al. (1990) counted the number of craniofacial characteristics present at birth from a list of 17 features reported in FAS cases. They found higher counts of defects not only in children of alcoholics but also in children of heavy drinkers (21 drinks/week or more but no addiction) compared to a pooled group of children of abstainers and light and moderate drinkers.

This brief review shows that the literature is filled with conflicting report on the effects of low to moderate alcohol consumption during pregnancy. While negative findings do not warrant the conclusion by Walpole et al. (1990) that there is no effect of low to moderate alcohol use during pregnancy, they highlight the fact that the effects of low alcohol intake during pregnancy have not been firmly established.

The same studies looking at the effect of alcohol consumption also reported other risk factors for the above adverse outcomes. In the absence of confirmation of an effect of alcohol consumption at low and moderate levels, those factors could possibly explain the adverse outcomes in women who drink but do not abuse alcohol in pregnancy. Black or non-White race is a risk factor for low birth weight (Lundsberg et al., 1997; McCarver et al., 1997) that is also a risk factor for FAS. Low maternal pre-pregnancy weight and low weight gain during pregnancy are reported as being associated with low birth weight and IUGR by Sokol et al. (1986), Walpole et al. (1990) and Lundsberg et al. (1997) but have not been found to be associated with FAS. Smoking during pregnancy is also a risk factor according to these authors, Sokol et al. (1980) and Yang et al. (2001). Low mental and psychomotor development scores on the Bayley scales were related to low social class, low maternal age and smoking in the study by Forrest & Florey (1991). In contrast, McCarver et al. (1997) found lower mental development scores in the offspring of older women. Genetic risk factors are examined separately in a later section of this review.

Risk Factors for Alcohol Consumption During Pregnancy

The recognition that fetal alcohol exposure has adverse effects prompted research on the patterns and amounts of alcohol consumption by women during pregnancy, and the risk factors for alcohol use. Studies on alcohol consumption during pregnancy typically do not examine the offspring and are limited to interviews of the mothers-to-be. This allows such studies to be conducted by mail or phone in representative samples of pregnant women. Some studies have also been conducted in a clinical setting.

The patterns of alcohol consumption during pregnancy that have been studied are the occurrence of binge drinking, average alcohol intake, frequency of consumption, consumption above certain levels, and a clinical diagnosis of alcohol abuse. Binge drinking is considered a high-risk behavior for fetal alcohol effects and was defined in the studies reported here as consumption of five drinks or more in a single occasion. Questions on binge drinking are included in the Behavioral Risk Factor Surveillance System (BRFSS) telephone survey of the CDC in the United States. That survey has been conducted annually from 1991 to 1993 and biannually from 1995 onward. Ebrahim et al. (1999) analyzed the responses to the binge drinking question of 4,611 pregnant women aged 18 to 44 years who were part of

the BRFSS from 1991 to 1995. They found that pregnant women who were unemployed, unmarried, and currently smoking were more likely to report at least one episode of binge drinking since the beginning of their pregnancy. The other characteristics they examined were not significantly associated with the risk of binge drinking and include age, race, education level and annual household income. More recently, the CDC released a report (CDC, 2002b) including a less detailed analysis of the BRFSS data on 4,695 women from 1995 to 1999 that found the same risk factors for binge drinking as Ebrahim et al. (1999) (except for smoking, which was not examined). Ebrahim et al. (1999) also reported a significant increase in the prevalence of binge drinking among pregnant women in the United States from 0.7% in 1991 to 2.9% in 1995. If one considers the data up to 1999, the prevalence of binge drinking is relatively stable around 2.5% from 1993 to 1999 (CDC, 2002). A change in the questions asked in the survey in 1993 may partly explain the increase in reported binge drinking compared to earlier years. Kesmodel et al. (2003) surveyed 432 women in an antenatal care programme in Denmark. They found that smoking before and during pregnancy were associated with binge drinking at least once in pregnancy, in agreement with Ebrahim et al. (1999), and also that multiparous women were more likely to binge drink.

Average daily or weekly alcohol intake was the most common measure of alcohol consumption being recorded, and some researchers preferred to dichotomize that response. The 1988 National Maternal and Infant Health Survey of a probability sample of 13,417 live births and 4,772 fetal deaths in the United States is the largest study we found. Hanna et al. (1994) analyzed the rich datasets produced by the survey and found that older age and a negative attitude toward pregnancy were associated with higher alcohol intake, while Black race was associated with a lower alcohol intake. In addition, in the subgroups of Black women and women who were separated, depression tended to increase alcohol consumption during pregnancy. Floyd et al. (1999) analyzed the risk of frequent drinking during pregnancy defined as six or more drinks per week in the live birth sub-sample from the same study. They found similar risk factors as for elevated alcohol intake: older age, White race, higher education, being unmarried, smoking and infant being firstborn. In the BRFSS survey from 1995 to 1999 (CDC, 2002b), older age, being unmarried and being employed were risk factors for a similar frequent drinking outcome, but not White race and higher education.

A large probability sample of births was also conducted in New Zealand in 1990-1991 (Counsell et al., 1994). Characteristics associated with any alcohol use in pregnancy were older age, higher education, lower parity, higher socioeconomic status and European or Maori ethnicity (compared to Asians and Pacific Islanders). Among the women who reported using alcohol during pregnancy, high socio-economic status was found to be significantly associated with frequent drinking.

In the survey reported by Leonardson and Loudenburg (2003), antenatal clinics in Minnesota, North and South Dakota and Montana were sampled under a stratified design and the pregnant women attending the clinics were asked to fill out a questionnaire. A composite “high-risk” pregnancy status defined by any drinking during pregnancy or self-reported alcohol problem was used as outcome. In a multivariate analysis, Leonardson and Loudenburg (2003) found that women who were younger, had fewer children, were single, had been physically or sexually abused, smoked, used other drugs or had been in treatment

for substance abuse, had a substance abusing mate or felt sad were more likely to be at high risk for alcohol use in pregnancy. Additional demographic factors associated with high risk in a univariate analysis were low education level, low income, unemployment and Native American ethnicity. The latter was also associated with any alcohol use in pregnancy in the study by Haynes, Dunnagan and Christopher (2003).

The Alaska Pregnancy Risk Assessment Monitoring System surveyed a stratified sample of 9,733 births from 1991 to 1994 in Alaska. Perham-Hester and Gessner (1997) analyzed the responses of the 6,973 mothers who answered questions related to alcohol consumption. For any third trimester alcohol drinking, they identified as risk factors: older maternal age, marijuana or cocaine use one month before or during pregnancy, cigarette smoking during the third trimester, higher maternal education, non-Alaska native ethnicity, presence of life stressors and residence outside a community banning alcohol. When they examined regular drinking (one or more drinks per week), only older maternal age, marijuana or cocaine use and experience of domestic violence two years before or during pregnancy were significant predictors.

In addition to the results of the population surveys reported above, older age was found to be associated with average alcohol intake (Kesmodel et al., 2003), alcohol abuse (Sokol et al., 1980) and any alcohol use during pregnancy (Haynes, Dunnagan & Christopher, 2003), and appears to be a consistent predictor of alcohol use in pregnancy at all levels. However, the survey by Leonardson and Loudenburg (2003) found an opposite trend, with a higher proportion of drinking in pregnancy at younger ages, a result echoed in a report by Alberta Health and Wellness (1999). As the two studies covered contiguous geographic areas in the Prairies, this inverse relation between age and drinking may be a regional particularity. The picture is more complicated for the number of previous pregnancies, even though it is related to age. One the one hand, women of low parity are more likely to drink when the threshold is a low or moderate level of alcohol use (Counsell et al., 1994; Floyd et al., 1999; Leonardson & Loudenburg, 2003). Floyd et al. (1999) found first time mothers were particularly likely to drink before pregnancy recognition, and attribute it to the fact that a first pregnancy is more often unplanned than later pregnancies. On the other hand, women of high parity are more likely to binge drink (Kesmodel et al., 2003), to abuse alcohol (Sokol et al., 1980), or drink more frequently during pregnancy (Counsell et al., 1994). This association between high alcohol consumption and higher parity is possibly confounded with older age.

High socioeconomic status itself (Counsell et al., 1994) or related characteristics such as high education (Counsell et al., 1994; Floyd et al., 1999; Perham-Hester & Gessner, 1997), being employed (Kesmodel et al., 2003) and, in the United States, White race (Floyd et al., 1999, Perham-Hester & Gessner, 1997) are found to be associated with alcohol consumption in pregnancy at low and moderate levels, again with the exception of the report by Leonardson and Loudenburg (2003). These characteristics of high socioeconomic status are, however, not found to be associated with binge drinking, except for employment (Ebrahim et al., 1999). Being unmarried or separated is reported by most authors to be associated with the various patterns of alcohol consumption that have been examined (CDC, 2002b; Ebrahim et al., 1999; Floyd et al., 1999; Hanna et al., 1994; Leonardson & Loudenburg, 2003; Sokol et

al., 1980). Since older women are more likely to be married, being unmarried is a risk factor distinct from older age.

A positive correlation between alcohol intake prior to pregnancy and during pregnancy was reported in clinic-based studies (Hayes et al., 2002; Plant, 1984). While this was not explicitly examined in large representative samples, Leonardson and Loudenburg (2003) report that previous treatment for substance abuse is a predictor of high risk of alcohol use in pregnancy. In addition, tobacco smoking (Floyd et al., 1999; Ebrahim et al., 1999; Kesmodel et al., 2003; Leonardson & Loudenburg, 2003; Perham-Hester & Gessner, 1997; Wiemann & Berenson, 1998;) and use of other drugs (Hayes et al., 2002; Horrigan et al., 1999; Leonardson & Loudenburg, 2003; Perham-Hester & Gessner, 1997) have been associated with various patterns of alcohol use in pregnancy.

Few studies have inquired about the mood of pregnant women. In the two studies that did (Hanna et al., 1994; Horrigan et al., 1999), depression and negative attitudes toward pregnancy were found to be risk factors for alcohol use during pregnancy. History of physical or sexual abuse was associated with drinking in pregnancy in the reports by Leonardson and Loudenburg (2003), Haynes, Dunnagan, and Christopher (2003), Horrigan et al. (1999) and Perham-Hester and Gessner (1997).

Summary

Table 8 summarizes the risk factors other than alcohol consumption itself for the most commonly studied adverse effects of alcohol on the fetus and for selected patterns of alcohol consumption during pregnancy. For some variables, the same modality is associated with both adverse effects on the fetus in the presence of alcohol consumption and with higher alcohol consumption in pregnancy. This situation is the case for older age, tobacco use and being unmarried. For other variables, opposite modalities are associated with Fetal Alcohol Effect and with higher alcohol consumption. Low socioeconomic status and Black or non-White race are associated with alcohol effects while in most studies higher socioeconomic status and White race are associated with alcohol intake in pregnancy.

Table 8
Risk Factors Other Than Alcohol

Variable	FAS	Growth retardation	Neuredevelop- mental delay	Binge drinking in pregnancy	Alcohol intake in pregnancy
Age	Older	No association found	Contradictory evidence for younger and older	No association found	Older in most studies, but younger in some
Parity	Higher	No association found	No association found	Higher*	Lower
Race	Black	Black or non-White	-	No association found	White
SES	Low	-	Low	No association found	High in most studies, but low in some
Marital status	Unmarried	-	Not examined in any report	Unmarried	Unmarried
Tobacco use	Smoker*	Smoker	Smoker	Smoker	Smoker
Other substances	-	-	No association found*	-	Substance use

Table 8 provides a summary of risk factors for Fetal Alcohol Effects and alcohol consumption patterns most commonly studied. For each variable the modality or direction associated with an increased risk is indicated for each outcome. Only variables examined in a multiple studies are included in the Table; * means the variable was examined in only one study included in this report for that particular outcome; - means the variable was not examined in any study included in this report for that particular outcome.

This study has identified recognized risk factors but there remains some question about how much of the predisposition toward the risks for alcohol consumption or even FASD itself are due to physiological or genetic factors. In response to these questions, the next section examines the literature on FASD effects on human neurophysiology and genetic susceptibility to alcohol. Animal studies being carried out to determine evidence of biochemical makers and risk or protective factors are also included as these studies offer exciting possibilities for FASD prevention in human populations.

Chapter Ten:

HUMAN NEUROPHYSIOLOGY

Current studies carried out with state of the art technology are identifying the effects of alcohol consumption on the brains of FASD children in ways and with detail not possible in earlier studies.

Human FASD Effects

Brain imaging in children has shown microcephaly, as well as structural abnormalities to the cerebellum, corpus callosum and basal ganglia (Bhatara et al., 2002; Mattson & Riley, 1995; Riley, McGee, & Sowell, 2004). FASD individuals have distinctly more shape variability in the corpus callosum (Bookstein et al., 2001). A thick callosum is associated with deficits in executive function, a thin one with deficits in motor function (Bookstein et al., 2002). Deviations in the development of the vermis in the brain was the most sensitive morphological indicator of the effects of prenatal ethanol exposure in 17 children examined with magnetic resonance imaging (Autti-Ramo et al., 2002). This dysmorphology in the brain leads to poor social functioning in those affected.

There are pervasive social impacts (e.g., deficits in attachment, behavioural problems, trouble with the law, inappropriate sexual behaviour, poor parenting, etc.) as described by Kelly, Day, and Streissguth (2000). However, criminality among FASD individuals may not be due to FASD, but due to an inheritance for excessive ethanol intake, which is related to criminality (Koren, Roifman, & Nulman, 2004). There are also increased rates of attentional, memory and information processing deficits (Streissguth et al., 1999).

Genetic Susceptibility to FASD

The large variations in the rates of FAS and other Fetal Alcohol Effects in the offspring of alcohol abusing mothers from different populations and ethnic groups has led to speculation that genetic factors may affect susceptibility to FASD (e.g., Abel & Sokol, 1986). We review the evidence from human and animal studies of genetic modulation of the risk of FASD and the results of genetic epidemiology studies in humans.

In humans, evidence for the heritability of a trait is obtained by examining the recurrence risk in relatives. Twin studies are extensively used for that purpose. Twins born to alcohol abusing mothers are rare and the largest collection of twins reported in the literature in a paper by Streissguth and Dehaene (1993) comprises only 16 pairs. These authors considered three possible diagnoses: FAS, FAE and no alcohol effect. They found that 5/5 monozygotic and 7/11 dizygotic twin pairs were concordant for diagnosis. Dizygotic twin pairs discordant for diagnosis were either FAS and FAE or FAE and unaffected. They also reported that monozygotic twins had an average IQ difference of 5 points while dizygotic twins had an average IQ difference of 11 points. These results suggest that monozygotic twins are more similar than dizygotic twins, although the sample is too small to estimate heritability of FAS and FAE. Greater similarity of monozygotic twins compared to dizygotic

twins is an indication that the fetal genotype affects susceptibility to alcohol exposure during pregnancy.

Studies in rodents and avians provide further evidence of genetic effects on the susceptibility to FASD. Differences between strains of mice have been observed in the number of malformations (Chernoff, 1980), growth retardation and prenatal mortality (Gilliam et al., 1989) and in learning ability (Gilliam et al., 1987) in the offspring of dams exposed to alcohol during pregnancy. Differences in maternal blood alcohol level between certain strains provides one mechanism for these effects (Chernoff, 1980), but differences in fetal outcome have also been observed between strains with similar blood alcohol levels (Gilliam et al., 1987). Recently, researchers have sought to tease out the effects of maternal and offspring genotypes. Breeding experiments between strains of mice susceptible and resistant to alcohol exposure in pregnancy have shown that the proportion of certain types of malformations and the weight of genetically identical offspring depends on the genotype of their mother (Downing & Gilliam, 1999; Gilliam et al., 1997; Gilliam & Irtenkauf, 1990). Offspring genotype effects have been established by directly exposing offspring to alcohol. Exposure of rats pups to alcohol at days 4 to 10, the equivalent of the third trimester in humans, have revealed differences in activity levels of the rats depending on their strain (Riley et al., 1993) and exposure of chicken eggs to alcohol (Cavieres & Smith, 2000) have revealed differences in cardiac deficits between strains.

Research to identify genetic variants modulating susceptibility to FASD has started only recently and remains scarce. So far, genetic epidemiology studies have focused exclusively on genetic polymorphims (variants) in the alcohol dehydrogenase 2 (ADH2) gene, which encodes the beta subunit of the alcohol dehydrogenase enzyme of class I. That enzyme is involved in the metabolism of alcohol into acetaldehyde. Three different alleles (alternative forms) of the ADH2 gene have been found in human populations and have been labelled ADH2*1, ADH2*2 and ADH2*3. ADH2 is a candidate gene for susceptibility to FASD because the enzymatic activities of its three alleles are very different. Viljoen et al. (2001) and McCarver et al. (1997), citing work by Bosron et al. (1983), report that the enzymes encoded by the ADH2*3 and ADH2*2 alleles are > 30 fold more active than the enzyme encoded by the ADH2*1 allele.

ADH2*1 is the most common allele but the frequencies of the three alleles vary between human populations. The ADH2*3 allele is observed mostly in individuals of African descent. In African American samples, frequencies of the ADH2*3 allele ranging from 15% to 33% have been reported (Arfsten, Silbergeld, & Loffredo, 2004; McCarver et al., 1997; Stoler et al., 2002;). In Caucasian samples from the United States, Stoler et al. (2002) and Arfsten, Silbergeld, and Loffredo (2004) found that only 1-2% of the individuals were carriers of the ADH2*3 allele. McCarver et al. (1997) mention that the ADH2*2 allele is present in a majority of Orientals but has low frequency in Caucasians and Native Americans. In a sample of controls from South Africa described by Viljoen et al. (2001) as being of Khoisan-Caucasian mixed ancestry, ADH2*2 had a frequency of 11% while ADH2*3 had a frequency of 4%. The ADH2*2 allele was nearly absent from the African-American and Caucasian samples of McCarver et al. (1997), Stoler et al. (2002) and Arfsten, Silbergeld, and Loffredo (2004).

Susceptibility to FASD could be influenced by the genotype of the mother, the genotype of the fetus, or both. In most studies, researchers have genotyped both the mothers and their newborns. The ascertainment scheme must be taken into consideration when evaluating the association between genotype and infant outcomes. If mothers are ascertained, then the maternal genotype must be taken into account when evaluating association between the infant genotype and the outcome, because infant genotype depends on the maternal genotype. This approach was done by McCarver et al. (1997), but not by Stoler et al. (2002). If infants are ascertained, the infant genotype must be taken into account when evaluating association between the mother genotype and the infant outcome for the same reason, something that Viljoen et al. (2001) failed to do.

McCarver et al. (1997) measured the ADH2 genotype and alcohol intake of African-American pregnant women. They then selected a sample stratified based on alcohol intake during the periconceptional period and ADH2 genotype to make these two factors independent by design, and followed-up the infants of the selected women to measure their birth weight, birth length and head circumference, and assess their mental development at 12 months using the Bayley Scales of Infant Development Mental Development Index (MDI). They found that drinking during pregnancy was associated with a reduction in birth weight only in the absence of an ADH2*3 allele in the mother, and with a reduction in MDI at 12 months only in the absence of an ADH2*3 allele in both the mother and offspring. The interpretation that can be given to these results in that a maternal ADH2*3 allele is protective against the adverse effect of alcohol on birth weight, and either a maternal or offspring ADH2*3 allele is protective against the adverse effect of alcohol on mental development. The ADH2 genotype had no effect on birth length while a maternal ADH2*3 allele was weakly associated with larger head circumference in the sample overall, not just in women drinking during pregnancy.

The smaller proportion of small-for-gestational-age infants among carriers of the ADH2*3 allele compared to non-carriers in the study by Arfsten, Silbergeld and Loffredo (2004) further supports a protective effect of the ADH2*3 allele against growth retardation, even though the difference was not statistically significant. By contrast, the results of Stoler et al. (2002) appear to contradict the above findings: they found the ADH2*3 allele in African American pregnant women who use alcohol to be associated with Fetal Alcohol Effects, defined as the presence of any of a number of abnormalities in the newborn, including growth retardation and facial features. The use of a more heterogeneous outcome than the ones measured by McCarver et al. (1997) and Arfsten, Silbergeld, and Loffredo (2004) could explain the discrepancy in results. However, a positive association between alcohol consumption and the ADH2*3 allele exists in the Stoler et al. (2002) sample and their statistical model may not adjust properly for that confounding variable, which may have led to a spurious result.

In the study by Viljoen et al. (2001), the frequency of the ADH2*3 allele was too low to detect a significant association with FAS. They recruited children instead of mothers and their analysis is valid only for the effect of the children genotype. They reported a lower frequency of the ADH2*2 allele in the FAS cases than in controls, suggesting that this allele in the fetus is protective against the effects of alcohol.

Summary

In summary, the combined evidence from limited human twin studies and from experiments in animals indicates that the susceptibility to FASD is influenced by the genotype of both the mother and the fetus. Efforts to identify the genes responsible for the difference in susceptibility have been limited to genetic epidemiology studies of only one candidate gene, the alcohol dehydrogenase 2. The study by McCarver et al. (1997) provides the most credible evidence that the ADH2*3 allele, present in populations of African descent, is protective against some of the adverse effects of alcohol exposure during pregnancy. The report of contradictory and inconclusive findings regarding the association between the ADH2*3 allele and similar outcomes by others indicate that it is premature to conclude that carriers of that allele are protected against FASD. Caution is also required regarding the finding by Viljoen et al. (2001) that the ADH2*2 allele is protective against FAS. None of these reports has considered other genes that could explain differences in genetic susceptibility to FASD.

Chapter Eleven:

Animal Research: Mechanisms of FASD Effects

Animal research has shed considerable light on the mechanisms of FASD effects and potential strategies to prevent FASD.

Pattern and Amount of Drinking

Pattern and amount of drinking is important with regard to Fetal Alcohol Effects. With repeated doses, retarded growth, physical abnormalities, and behavioral deficits are consistent outcomes in animals (Hannigan, 1996). The peak maternal blood alcohol level is the most important determinant of the likelihood and magnitude of FASD effects (Hannigan, 1996). This outcome is true even if the overall ethanol amount consumed is less than those of more continuous drinking patterns. The mechanisms are high blood alcohol levels during critical periods of development and repeated withdrawal episodes (Maier & West, 2001).

Critical Periods

It is well established that ethanol exposure has differential effects on neurotrophic factors, apoptosis-related proteins, endogenous antioxidants and reactive oxygen species in the rat brain depending on the age at which exposure occurs (Heaton et al., 2002). A particularly critical period concerns the period of neural development. A single exposure of infant rats or mice to ethanol during synaptogenesis (mid to late pregnancy for humans) can cause developing neurons to commit suicide (die by apoptosis) on a massive scale (Olney, 2004). There is no known safe period. In fact, it has been shown that long-term alcohol exposure even *prior to conception* tends to produce lower fetal body weights (Livy, Maier, & West, 2004).

Biochemical and Neurochemical Impacts

There is a wide range of biochemical and neurochemical impacts, although some areas are more affected than others. Ethanol interferes with an array of different molecular, neurochemical and cellular events during the normal development of the brain. Some brain areas are more affected than others and, even within a given region, some cell populations are more vulnerable. The following lists the documented impacts:

- Ethanol exposure during the development of the neocortex increases natural apoptosis (suicide). These effects may be associated with ethanol-induced alterations in both neurotrophic support, and the expression of cell adhesion molecules, which may affect cell-cell interactions and cell survival.
- Ethanol disrupts radial glial and asttroglial development (Guerri, 2002; Palomo, Archer, Beninger, & Kostrzewa, 2002).
- Ethanol use during pregnancy affects cytokine synthesis. This information helps explain the mechanism by which ethanol compromises the fetal immune system, as cytokines are recognized as the principal mediators of a variety of immunologic and pathophysiologic events (Ahluvalia et al., 2000).

- Ethanol exposure programs the body to insulin resistance (Chen & Nyomba, 2004).
- Ethanol suppresses glutamate and GABA transmission (Palomo et al., 2002). When this event occurs during synaptogenesis the timing and sequence of synaptic connections is disrupted, and this situation causes millions of nerve cells to receive an internal signal to commit suicide (Farber & Olney, 2003).
- Ethanol exposure causes long-term serotonin neuron deficits in mice (Sari & Zhou, 2004).
- Ethanol alters neurosteroid levels in the developing rat brain (Caldeira et al., 2004).
- Ethanol alters the postnatal development of the spontaneous electrical activity of dopamine neurons in the ventral tegmental area (Choong & Shen, 2004).
- Neurotrophic receptor proteins in the rat hippocampus, septum, and cerebellum are decreased while levels in the cortex are increased following chronic ethanol exposure during gestation (Moore, Madorsky, Paiva, & Heaton, 2004; Palomo et al, 2002).
- Ethanol significantly reduces the expression of several key neural genes, of which, xPax6 is the most vulnerable. It is the reduced expression of this gene that plays a key role in microcephaly in tadpoles (Peng et al., 2004).
- Ethanol has the effect of decreasing the expression and immunoreactivity of vasoactive intestinal peptide in both maternal and fetal tissues (Spong et al., 2002).
- Ethanol-mediated increase in tissue protaglandin levels are correlated with growth retardation. Elevated prostaglandins appear to have teratogenic potential (Randall, Anton, & Becker, 1987).
- DNA may be an additional cellular target for the toxic effects of ethanol and DNA damage resulting from ethanol consumption may contribute to various types of ethanol-related pathologies, including liver disease, cancer, FAS, and brain damage (Brooks, 1997).
- It is possible that ethanol also has some of its neurotoxic effects through its action on muscarinic cholinergic receptor signal transduction (Costa & Guizzetti, 1999).
- It is possible that excessive activation of the receptor for glutamate, the NMDA receptor, which occurs during withdrawal, may lead to neuronal cell death (Thomas & Riley, 1998).
- One cause of ethanol-induced intrauterine growth retardation is thought to be related to increased pressor activity in the placenta, resulting in decreased oxygenation and nutrient transport to the fetus. Thromboxane is increased by ethanol exposure. This is a paracrine substance that acts as vasoregulator within the intrauterine tissues (Siler-Khodr et al., 2000).

Oxidative Stress

Studies have identified that oxidative stress is an important final common pathway for alcohol damage on the fetus. Animal models have uncovered many possible mechanisms that may underlie ethanol neurotoxicity. However, many of these mechanisms seem to converge on the production of free radicals and oxidative stress as an important underlying mechanism of FAS (reviewed in Abel & Hannigan, 1995; Cohen-Kerem & Koren, 2003; Hannigan, 1996; Henderson, Chen, & Schenker, 1999). The body constantly reacts with oxygen as part of the energy producing processes of cells. As a consequence of this activity, highly reactive molecules are produced known as free radicals. These interact with other molecules within the cell, which can cause oxidative damage to proteins, membranes and genes. The source of

their devastating actions is the oxygen molecule's unpaired electron, which makes it unstable and electrically charged. It becomes stable by interacting with the nearest available molecule. Having no prejudices, it targets lipids, proteins and DNA. Scientists have discovered that the free radical's actions can damage molecules with which they react and sometimes cause the cell's demise.

Oxidative stress is attractive as a possible mechanism for the ethanol-induced brain damage associated with FAS for many reasons. The brain processes large amounts of O_2 in a relatively small mass, and has a high content of substrates available for oxidation (i.e., polyunsaturated fatty acids and catecholamines) in conjunction with low antioxidant activities, making it extremely susceptible to oxidative damage. Ethanol can induce oxidative stress directly and indirectly. The direct effect is achieved by formation of free radicals, which leads to peroxidation of lipids, nucleic acids, and alterations of enzyme activity. Another direct effect is the formation of reactive oxygen species (ROS). These are formed as biological products of molecular oxygen reduction and they probably play a role in mediating programmed cell death (Cohen-Kerem & Koren, 2003).

Genetic Determinants

Studies have also shown there are genetic determinants of FASD but the exact mechanisms are unclear. It has been observed that different genetic strains of mice are differentially impacted by fetal alcohol exposure in terms of number of malformations (Chernoff, 1980), growth retardation and prenatal mortality (Gilliam et al., 1989) and in learning ability (Gilliam et al., 1987). Differences in maternal blood alcohol level between certain strains provides one mechanism for these effects (Chernoff, 1980), but differences in fetal outcome have also been observed between strains with similar blood alcohol levels (Gilliam et al., 1987). Recently, researchers have sought to tease out the effects of maternal and offspring genotypes. Breeding experiments between strains of mice susceptible and resistant to alcohol exposure in pregnancy have shown that the proportion of certain types of malformations and the weight of genetically identical offspring depends on the genotype of their mother (Downing & Gilliam, 1999; Gilliam et al., 1997; Gilliam & Irtenkauf, 1990). Offspring genotype effects have been established by directly exposing offspring to alcohol. Exposure of rat pups to alcohol at days 4 to 10, the equivalent of the third trimester in humans, have revealed differences in activity levels of the rats depending on their strain (Riley et al., 1993), and exposure of chicken eggs to alcohol (Cavieres & Smith, 2000) have revealed differences in cardiac deficits between strains.

Exacerbating Effects

Under nutrition, other health problems and co-drug administration tend to exacerbate the effects of ethanol exposure on the fetus. Examples of this are zinc deficiency (e.g., Keppen, Pysher, & Rennert, 1985), hypoglycemia (Mitchell et al., 1998), diabetes (Padmanabhan & Shafiullah, 2004), hypoxia (Mitchell et al., 1998), and caffeine (Hannigan, 1995).

Paternal Drinking

Paternal drinking of the father plays a role. Early research found conflicting evidence concerning whether paternal drinking had any influence on FAS (e.g., Abel, 1989; Cicero, 1994; Leichter, 1986). Recent research has found fairly consistent evidence of decreased litter size and increased prevalence of low birth weight fetuses. The evidence concerning the risk of malformations continues to be mixed. Cognitive and behavioural findings tend to be the most robust effects. These findings include learning and memory deficits, hyperactivity, and poor stress tolerance. Multiple causal mechanisms have been suggested (e.g., subtle sperm deficits, Cicero, 1994), but none seems satisfactory to explain all the findings (Abel, 2004).

Summary

Clearly, ethanol exposure has a differential effect on the neurotrophic factors. A key period is indicative during the neural development given that during rat studies a single exposure of ethanol during synaptogensis can cause developing neurons to commit suicide (die by apoptosis) on a massive scale. There is also evidence that long-term alcohol exposure prior to and including gestation leads to lower birth weights in the new born. Evidence from animal research also alludes to the fact that ethanol exposure has wide ranging impacts, which have been listed in the above text.

Oxidative stress is a further consideration given that it is a common pathway for alcohol damage on the fetus. Scientific evidence has demonstrated, thus far, that there are many possible mechanisms that underlie ethanol neurotoxicity. Several of these mechanisms relate to the convergence on the production of free radicals and oxidative stress and are, therefore, considered to be important.

Genetic determinants remain relatively unclear but there are indications that ethanol exposures on different genetic strains of mice mutate different numbers of malformations. Unfortunately there are no consistent results that provide empirical evidence to date. However, these studies do indicate a probability that genetic determinants do have an effect on the developing fetus.

When considering other mediating factors, nutrition, other health problems, and co-drug administration have some correlationship with the effects of ethanol on the developing fetus. Whilst these issues relate to maternal ethanol exposure during pregnancy, attention should also be given to paternal alcohol consumption. Early research found conflicting evidence, nonetheless, contemporary studies have shown that there is an effect.

Evidently, animal studies do provide a framework for understanding and can act as a benchmark for human studies to be undertaken in particular fields.

Chapter Twelve

PROTECTIVE FACTORS

Exogenous supplementation with certain substances may limit ethanol teratogenesis.

Antioxidants

The body is equipped with natural antioxidants that control free radical molecules and mend damage. These include the enzymes superoxide dismutase (SOD), glutathione peroxidase, and catalase. Other molecules that contribute to antioxidant activity are vitamins such as vitamin C, vitamin E, and beta-carotene helps to detoxify certain harmful free radicals. Thus, it seems plausible that additional antioxidant supplementation may mitigate the effects of alcohol. Applying antioxidant treatment in pregnancy is not a new concept. Human clinical trials have demonstrated that women who are at high risk for pre-eclampsia benefit from vitamin C or E (Chappell et al., 1999).

Vitamins

Antioxidant treatment (vitamin E) during ethanol exposure reliably reduces the neurotoxic effects of ethanol in rats (Cohen-Kerem & Koren, 2003; Heaton et al., 2000b; Marino et al., 2004; Mitchell, Paiva, & Heaton, 1999a). This effect is also the case in the presence of ischemia (i.e., decreased blood flow) as noted by Mitchell, Paiva, and Heaton (1999b). Similarly, embryonic exposure to exogenous Vitamin E attenuates ethanol-induced decreases in endogenous levels of vitamin E in both embryonic chick brains and liver and also partially attenuates ethanol-induced reductions in brain neuron densities (Miller et al., 2003). It is not a panacea, however, as is does not prevent all of the behavioral impairments associated with FAS (Marino et al., 2004).

Folic Acid

Folic acid is another antioxidant that mitigates oxidative stress caused by ethanol exposure in rats (Cano et al., 2001; Garcia-Rodriguez et al., 2003). Here again, total protection is not typically obtained (Burdan, 2002).

Flavonoids

Silymarin is a flavonoid found in the milk thistle plant. Silymarin/phospholipid compounds mitigate against ethanol-induced behavioral deficits in pregnant rats (Abascal, Herbalist, & Yarnell, 1993; Busby et al., 2002; Edwards et al., 2000). It specifically has been shown to protect against impacts to spatial working memory (Neese et al., 2004), social learning deficits (Reid et al., 1999), left paw preference, and corpus callosum impacts (Moreland, La Grange, & Montoya, 2002). This compound has been previously shown to protect against ethanol-induced hepatotoxicity.

Neurotrophic Nerve Growth Factors

Nerve growth factor (NGF) is the prototype for the neurotrophin family of polypeptides, which are essential in the development and survival of certain sympathetic and sensory neurons in both the central and peripheral nervous systems. Neurotrophic factors also are capable of making damaged neurons regrow their processes in a test tube and in animal models.

Endogenous overexpression of neurotrophic nerve growth factors (under the control of the glial fibrillary acidic protein promoter) can protect the developing mouse brain from the neurotoxic effects of ethanol (Heaton et al., 2000a).

Exogenous administration appears to have similar effects. Both brain-derived neurotrophic factor (BDNF) and glial cell line-derived neurotrophic factor (GDNF) have been shown to provide protection (Bradley et al., 1999). Similarly, basic fibroblast growth factor (bFGF) and insulin-like growth factor I or II (IGF-I, IGF-II) improve neuronal viability when exposed to ethanol (Zell et al., 1999). Nerve growth factor tended to have a moderate ameliorative effect on the ethanol neurotoxicity in septal cultures, and was slightly effective in the hippocampal cultures, whereas basic fibroblast growth factor was protective for both (Heaton et al., 1994). The phosphatidylinositol 3-kinase (PI3-K) signaling pathway is strongly implicated in the underlying neuroprotective effects of neurotrophic factors against ethanol toxicity (Heaton, Kim, & Paiva, 2000).

These protective effects also occur in the presence of co-existing conditions. Nerve growth factor provides extensive neuroprotection against ethanol + hypoxia as well as ethanol + hypoglycemia, whereas brain-derived neurotrophic factor only affords protection against ethanol + hypoglycemia (Mitchell et al., 1998).

Peptides

Exogenous administration of certain peptides (NAPVSIPQ, SALLRSIPA) ameliorates the effects of ethanol on the developing fetus in mice (Brenneman et al., 2004). This includes preventing fetal death in the case of NAPVSIPQ (Spinney, 1999; Spong et al., 2001) and antagonizing ethanol-retarded brain growth deficits and underdevelopment of the forebrain and midline neural tube (Spinney, 1999; Zhou et al., 2004). The mechanism in the case of SALLRSIPA is agonism of activity-dependent neurotrophic factor (ADNF). However, the underlying mechanism is presumably due to antioxidant properties (Spinney, 1999; Spong et al., 2001). Pituitary adenylate cyclase-activating polypeptide (PACAP) has also shown to be a potent protective agent against ethanol-induced neuronal cell death in rats (Vaudry et al., 2002).

Nutrients

Natural nutrients (e.g., Ca, taurine, homotaurine, aminobutyric acid) demonstrate protective action against the impaired ionic transfer in the placenta caused by ethanol (Guiet-Bara, 1988). Zinc deficiency potentiates the teratogenic effects of ethanol, and, therefore, nutritional intervention for alcoholic women during pregnancy might reduce effects of FASD (Keppen, Pysher, & Rennert, 1985). Indeed, there is evidence of this effect (Seyoum & Persaud, 1991; Tanaka, Inomata, & Arima, 1983).

Other Substances

Gangliosides are a family of ubiquitously expressed membrane-spanning glycolipids, which have neuroprotective effects against ethanol neurotoxicity; therefore, exogenous gangliosides may have a role in reducing or preventing FASD (Hungund & Mahadik, 1993). It is proposed that ganglioside's neuroprotective effects against ethanol neurotoxicity involve protection and restoration of plasma membrane structure and, thereby, its function.

Prenatal ethanol exposure significantly decreases fetal brain phosphatidylcholine and phosphatidylethanolamine. Tuna oil prevented the decrease in these substances and, therefore, may be of use in pregnant women (Burdge et al., 1997).

Ethanol exposure is associated with abnormal development of the serotonergic system. Buspirone appears to prevent or reverse most of the ethanol-associated developmental abnormalities in serotonin reuptake sites (Jung-Ae & Druse, 1996). Similarly, the protective effects of ipsapirone (a serotonin agonist) have been demonstrated in the offspring of ethanol-fed dams (Tajuddin & Druse, 2001).

Enriched Environments

Raising FASD rats in enriched environments can improve the outcome. Post-weaning environmental enrichment can improve behavioral performance in FASD rats, although there tends to be persistent impairment in neuronal plasticity (Hannigan & Berman, 2000).

Summary

Much has been said about protective factors in the process of ethanol decomposition in the human body. The body is equipped with natural antioxidants that control free radical molecules and can assist in the reparit of cellular damage. Other molecules are said to act as an antioxidant such as vitamis, folic acid, flavonoids, peptides, and nutrients. The state of a woman’s health at the point of, and throughtout, the gestational period is of significance. Further, scientific enquiry is required to clearly establish the relationship between the mediating effect of antioxidant agents and ethanol damage to the fetal development.

Chapter Thirteen

Social Change Strategies: Defining Education, Marketing, Law, and Community-Based Programs

Social marketing is "the use of marketing principles and techniques to influence a target audience to voluntarily accept, reject, or abandon a behavior for the benefit of individuals, groups, or society as a whole" (Kotler, Roberto, & Lee, 2002, p. 5). Social marketing attempts to manage behavior by offering benefits and reducing costs in exchange for the desirable behavior (Rothschild, 1999). While social marketing makes use of advertising and other communication methods, it is much more. Through extensive use of research, social marketing requires the understanding of target individuals and competition. It then offers attractive products and rewards, and it overcomes barriers, so target individuals and communities have the opportunity, the ability, and the desire to perform the new behaviours. Social marketers do not restrict their campaigning to mass media vehicles alone, but achieve their objectives by involving the community to bring about societal change at the grass root level. Social marketing has been used in a variety of contexts such as promoting alternative transportation to reduce driving after drinking, safe sex behaviors, increased fruit and vegetable intake, breast cancer screening, and the use of environmentally friendly products (Deshpande & Basil, in press).

The marketing approach can be contrasted with two other approaches to social change: education and law (Rothschild, 1999). *Education* attempts to elicit behaviours using informational messages (most often using mass media). Education informs "but cannot deliver benefits," (Rothschild, p. 25) and thus in order to be effective, "education requires the target to initiate the quest for the benefit and solicit voluntary compliance" (Rothschild, p. 25). *Law* "involves the use of coercion to achieve behavior change in a nonvoluntary manner (e.g., military conscription) or to threaten with punishment for noncompliance or inappropriate behavior (e.g., penalties for littering)" (Rothschild, p. 25). *Law* can also influence transactions "through free market mechanism (by the use of price subsidies or increases via taxes)" (Rothschild, p. 25).

In addition to education, marketing, and law, social change agents also employ a variety of community-based interventions to promote desirable behaviors. Apart from changing individual behaviors, community-based programs also focus on "modifying community structures, processes, and policies" (Baker & Brownson, 1999, pp. 8-9). These community-based strategies allow change agents to tailor an approach that will meet community and individual needs, and to provide opportunity to local communities to influence program development, implementation, and evaluation (Baker & Brownson, 1999, p. 9). Due to the nature of their activities, community-based social change strategies tend to be comprehensive and multidisciplinary in nature.

Behaviour change is the end goal of any social change campaign. Each social change campaign has its strengths and weaknesses (Rothschild, 1999). Education campaigns are appropriate for those individuals who change their behaviours once they become aware of the

positives of the desired behaviour or the negatives of the current behaviours. Such individuals, it is assumed, have the motivation, opportunity and ability to behave in the desired direction. Legal approaches (taxes, etc.) are appropriate when individuals lack motivation to behave despite possessing awareness, ability, and opportunity. By using the legal stick of fines and punishments, social change agents can influence these individuals to switch their behaviours. Marketing approach is appropriate when targeting individuals lack environmental opportunity to behave desirably. Finally, community-based campaigns are appropriate when multiple factors influence current behaviours.

Impact of Education Campaigns

Education campaigns that use mass media or mass mailings have had limited success in promoting preventive behaviors on women's drinking habits. Such campaigns employ persuasive messages to persuade target individuals, but do not provide support services to change behavior. Examples are presented next.

A mass communication campaign highlighting the risks of smoking and drinking during pregnancy did not change alcohol consumption habits in two Danish cities (Odense and Aalborg). In a study reported by Olsen, Frische, Poulsen, and Kirchheiner (1989), the campaign managers carried out several communication activities between 1984 and 1987. They aired commercials in local cinema halls, local radio and newspapers, ran several reports about the campaigns, and conducted a healthy food preparation program on local television station. They also distributed brochures on smoking, drinking, and eating habits, and used stickers in several public places. Results showed that pregnant women were more knowledgeable of the risks of drinking and were motivated to reduce their alcohol consumption before and after the media campaign, but this self-reported awareness and motivation did not translate into actual behaviours. About 18-19% pregnant women in both cities continued to binge drink. The average number of drinks per week for the pregnant women continued to be around 1.5-1.8 drinks.

Inadequacy of education-only campaigns was also revealed by the Donnelley, Mowery, and McCarver (1998) study. Inner-city African American mothers of one-year old infants received mass mailings (A booklet called "Growing Up Drug Free: A Parent's Guide to Prevention") informing parents about alcohol, tobacco and drugs as well as providing tips on how to talk to children about drugs. The distribution of brochures failed to improve parental knowledge on prevalence of alcohol abuse among children. The campaign managers felt that face-to-face interactions might have been more effective in spreading awareness and correcting misperceptions than mere mass mailings.

Authors came across a couple of media campaigns in the literature, but did not find information on awareness and behavior change among individuals who were exposed to these campaigns:

- Best Start organization attempted to introduce alcohol and pregnancy signs among restaurants and bars in Wawa area, Canada (Burgoyne, 1998). The signs had the message, "Drinking alcohol during pregnancy can cause birth defects." Eighty-seven percent of the restaurants and bars that were approached during the campaign agreed to participate in this initiative.

- Minnesota Department of Health and others sponsored Minnesota Media Campaign, which was aired on TV and radio during 1994-95. The campaign attempted to promote alcohol-free pregnancy by promoting awareness regarding alcohol consumption, and by challenging alcohol use norms during pregnancy (Health Education Quarterly, 1996).

Unlike education-only campaigns, personalized education campaigns integrated with other strategies have had some success in influencing behaviors, although in certain instances. Such campaigns were successful in the US to influence adolescents, but not adult pregnant women in Latin America.

The STARS (Start Taking Alcohol Risk Seriously) – a model SAMHSA program[3] - is designed to prevent alcohol use among middle school and junior high school youth. The program uses a range of communication channels (media and non-media) to match the prevention content specific to alcohol initiation risk and protective factors. Another model SAMHSA program, the Family Matters Program, has been working to reduce the prevalence of tobacco and alcohol use among children 12 to 14 years of age. The intervention includes distribution of four booklets delivered to the home with a follow-up telephone interview of parents and children conducted by trained health educators. The booklet contains lessons and activities designed to teach parents to deal with substance abuse of their children. Unlike the study presented previously (by Donnelley, Mowery, & McCarver, 1998) where mass distribution of brochures was the only intervention, Family Matters Program combines distribution of booklets with personal interaction. This added activity seems to have worked, and has led to reduction in adolescent cigarette smoking and alcohol use.

A Latin American study (Belizan, Barros, Langer, Farnot, Victoria, Villar, & the Latin American Network for Perinatal and Reproductive Research, 1995) tested the influence of face-to-face home intervention among at-risk pregnant women. The intervention was delivered by trained social workers or obstetrics nurses and included psychological support and health education on 'health-related habits, alarm signs, hospital facilities, antismoking and anti-alcohol programs, and a reinforcement of adequate health services utilization for the pregnant woman and a support person.' Similar to other education campaigns, the experiment group reported better knowledge about the alarm signs for at-risk baby, but failed to report change in prenatal outcome, health-related behaviour, or utilization of health facilities. Short period of intervention (three months) and structural constraints may have led to campaign failure.

May and Hymbaugh (1989) report an attempt to increase community awareness of FAS consequences for Native Americans and Alaska natives. The campaign distributed a variety of educational materials that discussed numerous topics: alcohol and pregnancy, guidance for male partners, advice for Indian women for a safer pregnancy, etc. The local community workers had success in imparting FAS information to a variety of audiences (prenatal groups, school children, and community groups). However, the study did not report whether the increase in awareness led to change in behavior.

[3] All model SAMHSA programs were retrieved (early 2004) from the World Wide Web: www.modelprograms.samhsa.gov

Warning Labels[4]

Warning labels on alcoholic drink containers are required by law in many countries and regions. United States introduced a warning label in 1989 and requires placing it on all alcohol beverage containers being sold or distributed in the country (Caprara & Koren, 2004). It reads as follows:

> "GOVERNMENT WARNING: According to the Surgeon General, women should not drink alcoholic beverages during pregnancy because of the risk of birth defects."

In Canada, warning labels on alcoholic containers have been required by law in the Yukon province since 1992 (Yukon Liqour Commission, 2002) and in Northwest Territories (Caprara & Koren, 2004). At the time of writing this report, warning labels are not required in Alberta province. The English label required by Yukon government (the label also includes a French version) reads as follows:

"WARNING: Drinking alcohol during pregnancy can cause birth defects."

Going by the content, warning labels like most other media-based education campaigns use fear appeals by highlighting the adverse fetal consequences of drinking alcoholic beverages.

Warning labels on alcoholic beverage containers seem to work conditionally, as summarized below:

Continued exposure to warning messages about alcohol consumption increases awareness (Marin, 1997) and conversations about drinking during pregnancy (Kaskutas & Graves, 1994). Labels also provide the advantage of keeping awareness steady over time. Other sources do not offer that advantage. Awareness from advertisements, and signs and posters is high when these campaigns are in effect. However, it declines once the campaigns are taken off. This finding was revealed by Kaskutas et al. (1998) study. When they conducted the study in 1994, pregnant women reported to have seen the warning messages on container labels (42%), advertisements (58%) and signs and posters (17%), and to have had conversation about drinking (58%). Women likely to become pregnant reported to have seen the warning messages on container labels (60%), advertisements (63%) and signs and posters (17%), and to have had conversation about drinking (69%). However, when these numbers were compared over a five-year period (since 1989), the exposure levels had gone down for all sources except for exposure to container labels. However, alcohol consumption levels among pregnant women during the same five-year period remained pretty similar. In other words, sustained awareness from labels did not translate into reduced alcohol consumption among all women.

[4] Although warning labels on alcohol containers are treated as legal approaches, they ought to be treated as a part of education campaign from the target individual's perspective.

Labels may cause light drinkers to abstain, but not heavy drinkers (Hankin, Sloan, Firestone, Ager, Sokol, Martier, & Townsend, 1993; Kaskutas, 1995). Similarly, according to the Hankin, Firestone, Sloan, Ager, Sokol, and Martier (1996) study of inner city high-risk African American women, labels seem to positively influence drinking habits among nulliparous women but not among multiparous women.

Exposure to warning labels leads to increased awareness of consequences of drinking during pregnancy. However, the exposure levels are not consistent. Six months after the introduction of warning labels, groups that are more likely to have seen the labels are younger women (30 years and younger), heavy drinkers, the more educated, and men. Other groups (e.g., women 30 years and older, periconceptional and life-time abstainers, and non-risk drinkers) may be less effectively reached by these labels (Graves, 1993; Greenfield, Graves, & Kaskutas, 1999; Hankin, Sloan, Firestone, Ager, Sokol, & Martier, 1996; Kaskutas & Greenfield, 1992). This creates an interesting situation where labels influence light drinkers but they are less likely to see them. The reverse is true for heavy drinkers.

Exposure to warning labels runs into other challenges. First, women tend reveal a large number of false positive responses (Hankin et al. 1993; Graves, 1993; Marin, 1997). Second, there tends to be a four-month lag (Hankin et al, 1993) or more (Greenfield, et al., 1999; Marin, 1997) between enactment of law and increase in awareness of the label, and influence on conversations.

Warning Labels and Mass Communication Messages

Overall, education campaigns seem to be work more effectively when used in combination with other strategies, rather than used alone. A Kaskutas and Graves (1994) study supports this assertion. Exposure to three different messages (warning posters in restaurants and bars, warning labels on alcohol products, and mass media campaigns) among 18 to 40 year old women led to more conversations about drinking during pregnancy and reduction in alcohol consumption.

Legal Approaches

Other than the warning labels, a variety of legal approaches have been employed. These approaches include: mandatory distribution of brochures to applicants for a marriage license (e.g., it is required by law in the states of Wisconsin, Oregon, and Rhode Island [Ris, 1988])[5], taking away child custody from pregnant addicts (Jessup & Roth, 1988), restriction on where alcohol products can be advertised, and so on. Information on effectiveness of these measures was not available in the literature review.

Community-Based Programs

Community-based approaches have been successfully employed in a variety of settings and in targeting a variety of populations. In some cases, these attempts take a multidisciplinary approach combining one or more of these techniques: campaigns

[5] Similar to warning labels, receipt of brochures when registering for marriage license is a piece of communication for the target individual.

correcting normative perceptions, fear appeals, marketing, skill-enhancement, motivational interviewing, counseling, etc. A summary of campaigns organized by target audience is provided below.

Programs targeting pregnant women:

Using an approach where women determine their own abstinence goals seems to be effective. Chang et al. (2000) conducted a study to test the effect of a brief intervention (identifying drinking goals while pregnant, reason for the goal, risk situations for drinking, and alternatives to alcohol) among women in their 16th week of gestation. Current drinkers, who named abstinence as their goal, reduced subsequent prenatal alcohol use. Setting abstinence as a goal was associated with concern for personal and baby's health and positive social support.

Similarly, motivational interviewing seems to work. In a study with small sample size (N = 42), Handmaker et al. (1999) reported that motivational interviewing persuaded both light- and heavy-drinking pregnant women to reduce their alcohol consumption more effectively than providing them informative reports or literature.

Programs to target substance abusing women:

To help overcome substance abuse, programs using case management method, using multidisciplinary drug treatment, and building life-skills seem to be working. However, programs enhancing social skills, restructuring social network, and providing individualized social support fail to report positive influence.

Four US-based model SAMHSA case management programs seem to have been successful in lowering substance abuse:

- Project LINK integrates counseling services (clinical services and case management services) with maternal-child health system.
- The Parent Child Assistance Program (P-CAP) program uses a case management approach and a paraprofessional home visitation model for extremely high-risk substance abusing women.
- The SISTERS program uses a comprehensive paraprofessional case management model for substance abusing pregnant and postpartum women receiving detoxification treatment services.
- "Perinatal care program" is a comprehensive program for poor inner-city women who have delivered cocaine-tainted babies. The program addresses both mother's addiction and child development.

Drug treatment and counselling programs have shown positive results. According to Svikis, Golden, Huggins, Pickens, McCaul, Velez, Rosendale, Brooner, Gazaway, Stitzer, and Ball (1997), infants of drug-abusing pregnant women belonging to the group that received multidisciplinary drug treatment along with obstetrical care services were half as likely to require neonatal intensive care unit hospitalization. The Wheel project (Ashery, Wild, Zhao, Rosenshine, & Young, 1997) used an individual as well as group counselling intervention approach to reduce HIV-related sexual and drug risk behaviours among low-income, low-education women. When subjects who received the two interventions were combined into a single group, alcohol use declined by 18.3% following the interventions.

Skills-enhancement programs have shown mixed results. On one hand, life-skills training (e.g., communication skills, assertion skills, problem solving) with booster sessions reduced alcohol use among heavy as well light drinkers (Connors & Walitzer, 2001). On the other hand, social skills training and social network restructuring program failed to prevent drug use among high-risk pregnant (30% were either pregnant or parenting), multiethnic, low income female adolescents (14-19 year old) any more effectively than normative education strategy (Palinkas, Atkins, Miller, & Diane, 1996).

Youth-based programs to prevent or reduce alcohol use:

The youth-based programs described below employ a multi-disciplinary approach and report positive influence on their drinking behaviors. All programs described below (unless otherwise noted) are model SAMHSA programs.

The Project SUCCESS (Schools Using Coordinated Community Efforts to Strengthen Students) provides in-school services in order to prevent and reduce substance abuse among high risk, multi-problem high school adolescents, 14 to 18 years of age. The services include normative and preventive education, counselling and skills training, problem identification and referral, community-based processes and environmental approaches. The Too Good for Drugs (TGFD) program is a comprehensive K-12 school-based prevention program that teaches children skills that builds social competence and autonomous problem solving. Project Northland uses a multilevel community wide approach such as the use of home team, and peer led social influences curricula to prevent and reduce alcohol use among 6^{th}, 7^{th} and 8^{th} graders in a rural community (Perry, Williams, Veblen-Mortensen, Toomey, Komro, Anstine, McGovern, Finnegan, Forster, Wagennar, & Wolfson, 1996).

First Nations individuals

One program (the Baby SAFE program – a model SAMHSA program) was found in the literature review that uses a multidisciplinary approach among First Nations individuals in Hawaii and has been successful in decreasing the incidence and prevalence of drug or alcohol use among pregnant women and improving birth outcomes for these women. In this program, counselling staff provides in-person or by phone, counselling, education and support services.

Healthcare professionals

Healthcare professionals have the power to effectively communicate with women at the interpersonal level the information on risks (Gordis, 1992; Kesmodel & Kesmodel, 2002) and the promotion of screening tests and counselling sessions (Kloehn, Miner, Bishop, & Daly, 1997). These interactions may be more effective than a mass media campaign in promoting responsible alcohol consumption during pregnancy (Minor & Bernice, 1982).

However, several misperceptions exist among healthcare professionals. According to Diekman, Floyd, Decoufle, Schulkin, Ebrahim, and Sokol (2000) healthcare professionals, especially those graduating before warning labels were introduced, are unclear about the adverse effects of alcohol use during pregnancy, and about the effective methods for screening and counseling women who report alcohol use during pregnancy. Similar findings have been reported in Canada (Environics Research Group, 2000).

Proposed Prevention-Based Social Change Campaigns

Previous research reveals that women who drink before and during pregnancy constitute a sizeable number (e.g., if 10% of 622,000 18-44 year old women of childbearing age in Alberta drink during pregnancy, this translates into around 62,200 women who could put their babies at risk). Further, these women differ by their demographic and psychographic makeup (e.g., women earning low and high income continue to drink during pregnancy) and the reasons why they consume alcohol. Due to both size and individual differences, it becomes necessary to group these women into homogeneous segments. Previous research has also revealed that certain social change strategies work under certain conditions. Depending on the profile of the target group appropriate social change attempts should be carefully selected and targeted. For example, education campaigns and warning labels are appropriate among low-risk drinkers, community-based approaches are appropriate among women who face economic hardships and struggle with addictions, and marketing approach is appropriate for those men and women who drink for social reasons.

In the following section, target individuals are divided into segments and appropriate social change strategies are proposed for each segment to promote abstinence during and before pregnancy. Table 9 summarizes this discussion.

Table 9
Summary of proposed social change campaigns

Segment #	Target	Primary Objective	Strategy
1	*Women who drink during pregnancy (Best Start, 2003)* • Sub-segment 1: 30+ high-income earning women, • Sub-segment 2: Young, poor, unemployed or depressed, and indulge in substance abuse.	• Sub-segment 1: Change social norms; offer alternatives to socialize, promote skills to resist social pressure, • Sub-segment 2: Promote skills to resist social pressure; offer support services; promote skills to overcome addiction	• Sub-segment 1: Marketing approach (to promote alcohol-free social clubs) • Sub-segment 2: Comprehensive community-based programs
2	*Women who might be pregnant but do not realize it.*	Promote abstinence by spreading awareness and reminding consequences of drinking; change social norms	Media campaigns, warning labels, point of sale posters
3	*Adolescents (<18 years)* • Sub-segment 1: Low-risk drinkers • Sub-segment 2: Those who drink to socialize • Sub-segment 3: Those who indulge in substance abuse	• Sub-segment 1: Create awareness about consequences of drinking during pregnancy • Sub-segment 2: Offer alternatives to socialize • Sub-segment 3: Change social norms; promote skills to resist social pressure, Promote skills to overcome addiction	• Sub-segment 1: In-school curriculum, media campaigns, warning labels, point of sale posters, • Sub-segment 2: marketing campaigns (example, after-school programs) • Sub-segment 3: Comprehensive community-based programs

Segment #	Target	Primary Objective	Strategy
4	*Healthcare professionals*	Correct misperceptions about consequences of drinking during pregnancy, seek cooperation to proactively teach women about the consequences	Personalized communication
5	*Male partners of pregnant women*	Increase awareness about consequences of drinking during pregnancy; correct misperceptions, and promote responsible drinking when woman partner is pregnant	Education (to teach about consequences), and marketing campaigns (to promote alcohol-free social clubs)

Segment One: Women Who Drink During Pregnancy

Women in this segment drink during pregnancy. Specifically, Best Start (2003, p. 5) has identified two groups that may be at risk:

a. Women who are over 30 and have "successful careers" are most likely to report that they consumed alcohol during their last pregnancy.
b. Women who use other substances, have low self-esteem, who are young, poor, unemployed or depressed are at high risk as they may need substantial care and support in order to address their alcohol use.

These women include mothers who have previously delivered FAS babies.

Women in this segment who regularly consume four or more drinks per week are less likely to believe that alcohol use during pregnancy is harmful (Environics Research Group, 2000). In general, women in this segment do not respond positively to message-only campaigns. Messages such as 'Alcohol is bad for your baby,' or 'Just Say No' may not work. To motivate behaviour change, they have to be exposed to more comprehensive approaches that remind and reinforce abstinence.

The professional well-educated women (sub-segment 1) who are aware of consequences but who continue to drink during pregnancy (Ingle, Owen, Jones, Perry, & Cassidy, 1994) may be doing so due to social reasons. For these women, drinking may be satisfying their need to socialize, and may be perceived as the social norm. For this sub-segment, it may be appropriate to use a social marketing approach and provide alcohol-free alternatives to socialize. The second sub-segment could be approached with community-based programs that use counselling, case management, and multi-disciplinary treatment approach to reduce substance abuse.

Segment Two: Women Who do not Realize they are Pregnant

This segment comprises women who are not aware that they are pregnant yet. They are also more likely to quit drinking once they realize that they are pregnant. Women in this segment are also more likely to drink occasionally and at lighter levels. Primary prevention campaigns may be necessary for these women since they realize they are pregnant only in the fourth week (and many in the sixth week) of their pregnancy. This behaviour could negatively influence baby's health (Floyd et al., 1999). Second, most women in this segment are aware of alcohol consequences on the fetus, but they are not clear about the exact disabilities caused due to FAS. They are also not clear as to how much intake of alcohol is harmful (Environics Research Group (2000). Keeping these two factors in mind, the authors recommend exposing women to these two messages: (1) promote responsible drinking behaviour just before getting involved in sexual relationships, and (2) rectifying misperceptions about exact nature of alcohol consequences.

Previous research has shown that this segment responds well to message campaigns (Hankin, Sloan, et al., 1993; Kaskutas, 1995). Increase in awareness among these women seems to have a positive influence on their behavior. It may be appropriate to remind this segment using warning labels, media campaigns, point of sale campaigns, and distribution of brochures to applicants of marriage license. However, two suggestions are recommended: (1) It is important to ensure synergy among these communication elements (Kaskutas & Graves, 1994), and (2) While fear-based appeals are normally used to communicate drinking consequences, health communication literature has shown that these appeals are more effective when they are supplemented with positive solutions (Witte, 1992).

Box 1: Should Alberta Government introduce warning labels?

Canadian Medical Association (2000) in its policy document has urged the Canadian government to introduce warning labels on alcoholic beverages. While Yukon and North West Territory governments require warning labels on alcohol drink containers, such is not the case in Alberta. Should Alberta government introduce warning labels like the US, and the two Canadian provinces? From the US studies, we are aware that warning labels have had limited success. Warning labels are useful to dissuade low-risk drinkers from getting involved in risky behaviors (Hankin, Sloan et al., 1996) and are potentially effective in changing the culture of drinking, similar to the change in attitudes toward drinking and driving or smoking (Caprara & Koren, 2004). But labels have failed to influence heavy drinkers. While labels effectively influence drinking habits of light drinkers, not heavy drinkers the reverse is true when it comes to 'exposure' to labels. Heavy drinkers are more likely to see the warning labels than light drinkers. Finally, Canadians favor initiatives to inform public about the risks of alcohol use through warning labels on containers (Anglin, Kavanagh, & Giesbrecht, 2001; Environic Research Group, 2000;).

If the Alberta government decides to introduce warning labels on alcoholic beverage containers, they should keep in mind that labels work only with low-risk drinkers, who at the same time may be less likely to see them. In order to ensure a better exposure to alcohol consequences among low-risk drinkers, warning labels could be introduced, but they should also be supplemented with other media. However, in order to ensure a better exposure to alcohol consequences among high-risk drinkers, marketing and community-based approaches ought to be supported.

Segment Three: Adolescents (<18 years)

Three kinds of campaigns could be employed to prevent alcohol use among adolescents. Similar in-school strategies have been recommended by Ma et al. (1998b).

Message Campaigns for Low-Risk Drinkers:

Adolescents in this segment are associated with high self-esteem and motivation for good health. They are also likely to drink responsibly once they become aware of consequences. These individuals could be persuaded to abstain from alcohol by being exposed to a variety of communication campaigns including school curriculum, media campaigns, warning labels, point of sale posters, and so on. Currently, Alberta Children's Services (2004) in partnership with Alberta Learning and AADAC is running a youth awareness campaign with a tag line "Alcohol and Pregnancy Don't Mix." The campaign intends to educate youth about FAS, and to promote abstinence during pregnancy. The campaign includes two 30-second radio commercials, posters and transit interior ads. The campaign is likely to work with such adolescents.

Marketing Campaigns for Those Who Drink in Order to Socialize:

Individuals who drink for social reasons may respond positively to marketing campaigns that offer attractive alternatives to socialize. After-school programs demanding active involvement in social and physical activities, and youth mentoring programs could be promoted.

Community-Based Campaigns for High-Risk Drinkers:

Programs using a multi-disciplinary approach and involving parents, teachers and trained personnel could be employed in order to deal with high-risk individuals (those already practicing risky behaviors, or those who are children of alcoholics, etc.).

Segment Four: Healthcare professionals

Healthcare professionals (e.g., physicians, midwives, and general practitioners) have power to influence women's drinking behaviors. While creating education campaigns targeting women, this group could be used as endorsers or spokespersons.

In addition, there is a need to create a dialogue with these individuals. According to the Environics study (2000), four findings about healthcare professionals were revealed. These findings along with our recommendations are discussed below.

1. Healthcare providers send a variety of messages to women about how much alcohol should be consumed before and during pregnancy. To ensure that the message of abstinence gets consistently passed on to women, health professionals should be contacted using personal communication techniques.
2. Healthcare providers cite lack of time as an important reason for not discussing alcohol consumption with women of childbearing age. To overcome time barriers, health providers could be provided with brochures that discuss alcohol effects. Each time a woman of childbearing age visits a health clinic, providers could hand out this brochure and reduce his/her time involvement.
3. Majority of healthcare providers (60%) "believe a registry of specialists for consultation, referral resources, and clinical practice guidelines would be helpful in their practice." This database needs to be developed (if not already in existence) and made available to the providers.

4. A few providers (25%) expressed a desire for "training in addictions counseling, assistance with diagnosis, or access to information via telemedicine." Necessary help should be offered to these select few providers.

Segment Five: Male partners

Partner alcohol use is a good predictor of alcohol use during pregnancy, especially among adolescents (Wiemann & Berenson, 1998). To ensure that male partners (in a heterosexual relationship) exhibit responsibility in their own drinking habits while their female partner is pregnant, two types of campaigns could be employed. (1) Education campaigns would create awareness about the effects of alcohol use on fetus development and the importance of providing support to their partners to abstain from alcohol during pregnancy. (2) Over and above education, a marketing campaign would promote alcohol-free alternatives to socialize for the couple during pregnancy.

Social marketing campaigns clearly must be addressed at directed segments of the population to be effective. A critical component of successfully directing programs to a segment of the population is to understand that population and such understanding must address different cultural perspectives such as those addressed below.

Summary

Education-only campaigns increase awareness, but have limited influence on changing behaviors. However, warning labels increase awareness, but influence behaviours of only the light drinkers, not the heavy drinkers. When used in synergy, media-based campaigns and warning labels have a greater influence on changing behaviors. Community-based comprehensive social change programs have a good record of influencing at-risk women drinkers and youth. No marketing-only campaign was found in our literature review.

Chapter Fourteen:

Culture

Streissguth (1994), a leader in the field of FASD, espouses "FAS is not an Indian problem per se; it is an alcohol problem. . . . FAS is a major public health problem in cultures that have problems with alcohol abuse" (p. 45). From the literature selected for this review, cultural perspectives were discussed in 41 articles. All articles were retained because of the varied perspectives that they contributed to the understanding of the relationships between culture and FASD. Of these 41 articles, 19 present research reports.

Incidence of FASD Among Native Populations

Bray and Anderson (1989) provide a critical synopsis of three case studies conducted in British Columbia and the Yukon Territories. They address the epidemiological evidence suggesting FAS occurs more frequently in Native populations. Given data from three case studies, the authors question the epidemiological evidence, for instance:

- Lack of published research in Canada limits drawing conclusions that there is a higher incidence of FAS among Native, compared to Non-Native, populations.
- The application of standard diagnostic criteria across cultures in problematic (e.g., facial features).
- The use of IQ tests to assess CNS dysfunction requires caution (e.g., "Indian children often score better in Performance IQ tests, which require a holistic/visual spatial problem-solving strategy, rather than Verbal IQ tests [Bray & Anderson, 1989, p. 43]).

The authors ask several key questions (e.g., related to: consumption rates; attitudes; support programs; fertility rates, parity, and age at childbearing; and risk factors) regarding the need to understand Native populations and the incidence of FAS. They conclude that more research is essential to delineate the true picture of FAS among Native populations.

O'Connor and Whaley (2003) "examine the prevalence rates of prenatal alcohol consumption . . . [and] identify variables associated with post-conception drinking in low-income minority women" (p. 773). The Public Health Foundation Enterprises (PHFE) Women, Infants, and Children (WIC) Program, serving over 316,000 pregnant women infants, and children monthly in 55 centres in Southern California, is "mandated to screen prenatal clients for use of alcohol and other drugs" (p. 774). The researchers randomly selected 12 WICs, with caseloads over 3,000 monthly, for participation in this study. Using a cross-sectional survey, they assessed alcohol use rates, demographic variables, and alcohol risk status rates among 826 (71.3% response rate) pregnant women enrolled in the PHFE WIC Program. From the self-administered screener, O'Connor and Whaley learned that "30% of the white non-Hispanic women, 29.4% of the black non-Hispanic women and 20% of the Hispanic women" (p. 777) reported drinking post-conception. The best predictor of post-conception alcohol consumption was the woman's high-risk drinking score as measured by the TWEAK. For instance, the "the mean TWEAK scores were higher in the

postconception drinking group (mean = 1.43 [1.16]) than in the abstinent group mean (mean = 0.29 [0.77]) (t = 14.09, 620 df, p < .001)" (p. 778). Importantly, the TWEAK scores were also higher for those women who continued to drink even after learning that they were pregnant. O'Connor and Whaley conclude that it is important to screen "low-income minority pregnant women in a community setting so interventions can begin to prevent FAS and related conditions" (p. 773).

Rhodes, Gingiss, and Smith (1994) investigate "the frequency and intensity of drinking by pregnant African-American, Hispanic, and White adolescents and the extent to which these young women maintain, reduce, or quit their drinking during pregnancy. . . . [and] examine the role of background characteristics and the influence of peers, sexual partners, family members and mentors on young women's drinking patterns during pregnancy" (p. 556). The convenience sample included 183 pregnant adolescents. The researchers utilized several key instruments: demographic data, substance use, Youth Risk Behavior Survey (YRBS [Kolbe, 1993]) and the National Survey of High School Seniors (NSHSS [Johnston, O'Malley, & Bachman, 1988]). The mean age of the sample was 16 years, with a range between 11 and 19 years. The sample described their ethnicity as: African-American – 89 (48.5%), Hispanic – 78 (42.5%), and White -16 (9%). The authors note that adolescents are more likely to continue drinking throughout their pregnancy when their environments provide easy access to alcohol and their partners and peers are drinking and using drugs. However, adolescents who had strong parental and mentor support were less likely to consume alcohol while pregnant. Also, adolescents with support were more likely to resume their education postnatally. White adolescents, compared to African-American and Hispanic adolescents, were most likely to maintain their drinking behaviours since becoming pregnant and following delivery.

Literature Reviews

May and Moran (1995) note the common use of the term "Indian" or "American Indian," to reflect indigenous peoples rather than the term "Native American." They also report "as of 1993, there were 341 federally recognized tribes and another 111 tribal groups seeking federal recognition" (May & Moran, 1995, p. 289). This literature review makes a significant contribution in highlighting primary, secondary, and tertiary prevention efforts among Indian peoples. In particular, May and Moran describe the application of a theoretical framework as well as the development of policies and laws.

Theory

Few researchers employ a theoretical framework in their quest to understand FASD. Ferguson (1970, cited in May & Moran, 1995) is a notable exception. She applied the stake theory to a sample of Navajo Indians while exploring chronic alcoholism. Essentially, the stake theory suggests that individuals who have a stake in society tend to conform to society's norms and, therefore, they are less likely to engage in "deviant" behaviours such as alcohol misuse. In this research, Ferguson explained that stake in a Navajo society was reflected by involvement in traditional activities such as stock raising, silversmithing, and

farming in a family context. In comparison, stake in Western society was demonstrated by individuals holding a permanent job or engaging in activities whereby Navajos and non-Navajos were treated equally. Regarding chronic alcoholics, Ferguson discovered that individuals with a stake in either the Navajo society or the Western society fared better than individuals with no stake in either society. Interestingly, those individuals with a stake in both Navajo society and Western society experienced the greatest success.

Policies and Laws.

In 1953, each tribal group was given authority to regulate alcohol within its own borders. About two-thirds of the Indian tribes in the US espouse a policy of prohibition. However, the evidence depicting negative effects of alcohol misuse speak to the ineffective nature of this approach. Indeed, some policies appear to stimulate alcohol-abusive behaviour. Despite the negative outcomes associated with some policies, several researchers have evaluated a variety of policy approaches and deemed them to be effective. May and Moran (1995) observe that "the non-Indian literature on policy-oriented primary prevention is extensive and may inform health promotion among Indians as well" (p. 296). This comment is couched in the need to ensure that prevention programs focus on the specific Indian culture and engage the community in health promotion activities.

Primary Prevention in a Cultural Context

In some instances, researchers investigate the knowledge levels of the public, including potential mothers, and other researchers explore the knowledge levels among health care professionals. To be effective, primary prevention strategies need to be community based, multifaceted, and comprehensive (Parker-Langley, 2002).

Knowledge Among Public

Shostak and Brown (1995) explore "the kinds and amounts of information possessed by urban American Indians in Southern California regarding the effects of alcohol on the developing fetus" (p. 39). These authors present a good overview of the diagnostic criteria, health issues, and incidence rates for general and Native populations. Based upon 20 studies conducted in Australia, North America, and Europe, they suggest a worldwide rate of 1.9/1000 live births compared to "1/690 live births among Navajo tribes, to 1/495 live births among Pueblo tribes, to 1/102 live births among Plains tribes" (Shostak & Brown, p. 45). Shostak and Brown also note that Bill H. R. 1322, proposed March 7, 1992, and the Comprehensive Fetal Alcohol Syndrome Prevention Act, proposed in 1994, have not yet been implemented into law. In their study, they distributed a 53-item survey to 76 American Indian adults (e.g., some were undergraduate and graduate students in university, others were residents in an alcoholism treatment facility, others were members of a church, and others were recipients of a newsletter). Part one of the survey included 31 statements about alcohol consumption, effects of alcohol on the fetus, and expectations for the future. Part two of the survey included demographic information. Shostak and Brown describe the sample as primarily female (68%), 27 years of age (median), single (70%), educated (i.e., 88% had a high school diploma and 22% had a college degree). Further, 12 (16%) subjects had lived on

a reservation. Two subjects indicated that they had an FAS child and two subjects indicated that they had FAS. Three important findings were noted:

- Subjects had the highest level of information regarding alcohol consumption during the prenatal period.
- Subjects were also informed about the effects of alcohol on the fetus.
- Female subjects had "significantly higher mean correct percentage than male respondents for the Expectation for the Future section [i.e., 64% vs 51% correct respectively" (Shostak & Brown, p. 56).

Shostak and Brown note the "powerful negative stereotypes" (p. 56) about children with FAS. They comment about the effect of "The Broken Cord" by Michael Dorris and the television movie based on this book. They conclude that prevention programs are effective in increasing knowledge levels in certain areas. Further, they observe that there are more noticeable FAS programs available in rural and reservation areas than in urban areas.

In a northern Manitoba community, Williams and Gloster (1999) explore the knowledge level, age and gender differences, and relationship between knowledge about FAS and the frequency of drug and alcohol use among a Native population. A nonrandom sample of 466 Natives, proportionately representative of 21 reserves, participated in interviews. Williams and Gloster report that only 18% of the 242 women indicated no use of any kind of drugs during their pregnancy. Further, although 80% of the sample indicated that the fetus could be adversely affected by alcohol, only 36% had heard of FAS, both percentages are lower than those reported in the US. Perhaps not surprisingly, teenagers, compared to other age groups, report higher levels of smoking and alcohol use during pregnancy. Also, 61% of the sample (i.e., more men than women) believed that there was a safe amount of alcohol that could be consumed without any adverse effects. They conclude that education remains a key, but is insufficient alone, in arresting the use of alcohol during pregnancy.

Ma, Toubbeh, Cline, and Chisholm (1998) explain a three-phase project to develop substance abuse prevention products for purposes of eliminating, or at least, reducing, the incidence of FAS among Native American adolescents. In the first phase, Ma et al. (1998) conduct a needs assessment from the perspective of prevention professionals. It was anticipated that the outcome of this assessment would be recommendations to facilitate the development of appropriate FAS prevention materials.

In a related article, Ma, Toubbeh, Cline, and Chisholm (1998) determine the needs and strategies for developing a culturally appropriate FAS prevention curriculum for Native American middle school students. The researchers randomly selected 163 students in grades six through eight from four middle school sites and representing 60 tribal affiliations. Their findings indicate a high percentage (74%) of these adolescents indicated that their parents and immediate family members are the most influential individuals in their lives. However, 40% indicate that they do not talk with their parents about alcohol and 71% indicate their parents would respond negatively to them drinking alcohol. Further, 91% noted that alcohol could be harmful to their health and 82% knew that alcohol could cause birth defects. More

males than females drank alcohol (57% vs. 51%, respectively). Perhaps their knowledge derived from previous exposure to educational courses about the use of alcohol and drugs as reported by 61% of the females and 43% of the males. Although challenging, Ma et al. (1998b) conclude that:

> A multi-faceted prevention and intervention program in FAS must be age-, gender-, and culture-sensitive; must reflect shared responsibility between adolescent males and females; must be rooted in the family, community, and the school; and must use media avenues appropriate to adolescents. (p. 135)

In a recent article, Kvigne and colleagues (2003) describe the characteristics of Northern Plain Indian mothers who have children with FAS. They derive these characteristics from two studies: Study 1 includes 43 case mothers whose children had FAS compared to 86 control mothers and Study 2 includes 35 randomly selected mothers whose children had one to four criteria compared to 70 different control mothers. The findings include the following:

- "Maternal drinking, maternal age, intentional injuries, depression, and sexual abuse were the strongest predictors of FAS" (Kvigne et al., p. 299), using logistic regression analysis.
- "Mothers of children of FAS had significantly higher levels of alcohol consumption and a more serious alcohol addiction than mothers of children who has some characteristics of FAS" (Kvigne et al., p. 299).
- Terms such as alcohol-related neurodevelopmental disorder and alcohol-related birth defects are used today rather than Fetal Alcohol Effects (FAE).
- Mothers of children with FAS (Study 1) had significantly higher rates of alcohol abuse and referral for alcohol treatment than mothers whose children has some characteristics of FAS.

Kvigne and colleagues conclude the following:

- "Health care providers should routinely screen, promptly intervene, and refer for treatment all women of childbearing age who have problems associated with substance abuse, such as depression, sexual abuse, and injuries" (Kvigne et al., p. 302).
- Intervention is critical for women who have an FAS child, because successive children with FAS demonstrate even more dramatic effects.
- There is a need to use systematic screening for maternal alcohol use early in routine prenatal care.
- A prenatal health assessment tool, approved by the Indian Health Services and Centres for Disease Control and Prevention, is available online at: http://forms.psc.gov/forms/HIS/his.html
- Physicians need to be updated regarding effective methods for screening and counselling women for alcohol abuse.

Zhand and Klein (1997) conducted research to "provide information on self-reported needs, problems, and concerns for American Indian women who are pregnant or parenting

and at risk for heavy or problem substance use" (p. 121). The sample included 290 women who were 15 years of age or older, living in rural and urban settings. Of the 290 women, the sample included 171 (59%) who were "screened in" and completed the long interview, indicating that they met the threshold for risk for heavy or problem alcohol or drug use. The remaining 119 women completed the short interview. In total, 91 tribes were represented (i.e., 35 California and 57 out-of-state tribes). The major findings included the following:

- 72% of American Indian/Alaska Native women were "screened" as at risk of heavy or problem substance use versus 39% for comparable agency clients. This finding demonstrates frequent, although not daily use of large quantities of alcohol or bingeing.
- Over 50% reported recent marijuana use.
- Over 30% reported recent methamphetamine use.
- A somewhat larger proportion of rural versus urban American Indian women reported use of marijuana and methamphetamines.
- A positive finding was the reported decreased use of alcohol and drugs during pregnancy.
- Parenting women also faced legal interventions regarding their children, with substance abuse being the issue.
- Other findings: relative disinterest in formal substance abuse treatment, belief that one can decrease use independently, and common lack of desire to completely cease use.
- The major finding regarding the service needs assessment was the emphasis on economic concerns (e.g., education and vocational training, job placement, housing and transportation assistance, food and income support, and help with health care.
- Overall, there was an emphasis on cultural and spiritual needs noted by the women in this study.

Knowledge Among Health Care Providers

Plaisier (1989) describes the effect of FAS prevention efforts among American Indian communities in northern Michigan. Thus, "Indian health workers were interviewed regarding their FAS prevention programs. Childbearing Indian women were interviewed regarding their knowledge and attitudes about drinking alcohol during pregnancy" (p. 17). The provision of educational resources was well received by all communities. Despite participating in a workshop on FAS, health workers acknowledged a basic understanding of FAS concepts, a lack of information about current research findings, and a discomfort in discussing substance abuse with clients. Of the 29 women interviewed, 83% had heard about FAS and 50% could identify a characteristic of FAS. Of note, 11 of the 29 women reported that when physicians asked about drinking, they responded "no" and the topic was dropped. Plaisier concludes that information about FAS is reaching women and health care providers in these northern communities although aggressive outreach initiatives are needed to support women who are socially isolated; educational efforts alone are insufficient. Importantly, "traditional cultural patterns can support the development of a strong Indian women's health program" (Plaisier, p. 16) for purposes of improving their own health care.

Secondary (Cultural) Prevention

Secondary prevention may be described as efforts intended to identify and arrest the use of alcohol, and other substances, at an early stage. Thus, research efforts focus upon epidemiological surveys and prevention programs in various settings (e.g., schools, clinics, and communities).

Kaskutas (2000) discusses the Developing Effective Educational Resources (DEER) project for purposes of creating culturally appropriate consumption measures, collecting epidemiological data, and assessing responses to health warnings. Surveys were returned by 321 subjects (74% response rate): 102 American Indian, 185 African American, and 34 Whites in southern (n = 225) and northern (n = 96) California. Interviews were conducted matching ethnicity for most subjects. There were no ethnic differences in terms of exposure, understandability, believability, and self-report of health warning influences. Interestingly, pregnant drinkers reported that these health-warning messages made them feel negative toward themselves. Moreover, women in this study who tended to drink at risk levels were less likely to report seeing warning labels. African American women had less accurate knowledge about FAS than American Indian women. Unfortunately, heavier drinkers of all ethnicities were less likely to realize that "it was never too late to reduce drinking during pregnancy" (p. 1249). Hence, Kaskutas recommends changes in campaign strategies and interventions to provide factual information to help at-risk women reduce their drinking during pregnancy.

At the request of the Dene Cultural Institute, Kowalsky and Verhoef (1999) "examine the concerns and beliefs of people about FAS and FAE" (p. 150). With the aid of a contact person, the researchers developed culturally sensitive guidelines for gaining entry into an Aboriginal community. Using a snowball technique, one researcher collected data through participant observation and interviews with 50 service providers, community resource personnel, and individual community members. Regarding concern expressed by professionals about the prevalence of FAS/FAE, Kowalsky and Verhoef report "diagnosed FAS is only the tip of the iceberg and that FAE is much more prevalent than FAS" (p. 153). Although service workers expressed a belief that health professionals were knowledgeable about FAS/FAE, this belief was not borne out in reality. Among the community resource personnel, foster parents displayed their emotions, both sadness and anger, because "they perceived no effective way to prevent women from inflicting what they believed to be an unnecessary life sentence on innocent children" (Kowalsky & Verhoef, p. 156). Significantly, the emotional and spiritual pain demonstrated by foster parents was not evident among professionals. Kowalsky and Verhoef explain:

> Alcohol abuse has been an attempt to fill the void of lost spirit and culture. Long-term alcoholism has had a negative effect, as is evidenced by the high prevalence of sexual abuse, physical abuse, family discord, and, ultimately, FAS/FAE. (p. 161)

The lack of community support hinders the willingness of individuals to acknowledge the prevalence of FAS/FAE and prevents the completion of the healing circle process. The researchers conclude that the learning disability of the young (i.e., those FAS/FAE children)

resulted in a spiritual agony among community members who realized the loss of their language, traditions, and way of life.

Li, Olsen, Kvigne, and Welty (1999) describe the effect of a maternal substance use-screening program. They surveyed 20 clinics within the Aberdeen Area Indian Health Services, which includes 19 tribes. The sample includes 30 prenatal clinic staff members working within the 20 clinics and 235 women whose medical chart was reviewed regarding their first prenatal visit. Li et al. report that the protocol for using the self-administered questionnaire (SAQ), which included items from the revised T-ACE (Tolerance, Annoyance, Cut down, and Eye opener), was not always followed. For instance, although the SAQ was administered, the health care provider did not always score the tool. Interestingly, multiparous women were less likely to be given the SAQ. The researchers conclude that training is necessary for successful implementation of any screening tool. Further, confidentiality needs to be addressed so that information pertaining to substance use screening is available for use by all health care providers. Li et al. speak to the needs regarding implementation of a successful screening tool but do not speak to issues of American Indian pregnant women in this article.

Corse, McHugh, and Gordon (1995) describe their findings subsequent to the implementation of an innovative model to prevent and treat substance abuse among pregnant women. Using a naturalistic research design (i.e., participant observation and interviews) over a period of 18 months, they explore caregiver attitudes and behaviour to substance-using women who attend a nurse-midwife managed clinic in Philadelphia. Participants in the research include seven nurse-midwives who work full-time in the clinic. The client base includes: 53% Caucasian, 39% African American, 5% Southeast Asian, and 3% other ethnic groups. The majority (63%) of the clients are unmarried, 62% are on Medical Assistance, 50% have less than high school education, and the majority of the clients (43%) are between 19 and 24 years of age. The model encompasses two key elements: a focus on the goals of an expanded nurse-midwife role and teamwork as well as staff training relevant to the effects of substances of abuse on women and the developing fetus. The education provided in this model resulted in changed attitudes and behaviours toward clients. The model effected three structural changes: retaining substance-using women in clinic; implementing a care coordinator model, which increased continuity of care, heightened sense of responsibility and commitment, and intensified the relationship, lengthened appointment time, and increased standard of prenatal visits; and adding on-site services to address addiction and other life issues.

Streissguth (1994) provides a comprehensive review of FAS/FAE. She describes several successful programs that have been initiated in support of American and Canadian Indians. As an example, she identifies the 3-P Program for preventing Fetal Alcohol Syndrome: public awareness, professional education, and provision of services. She states, “the motivation to prevent FAS arises from the knowledge of its devastating consequences to the health of the community’s children and to the vitality of the tribal culture” (Streissguth, p. 61). To that end, Streissguth (1994) suggests that one of the most effective strategies for preventing FAS is to identify individuals with FAS in the community. Unfortunately, there is no research reporting “the usual mode of play therapy, insight-oriented therapy, or group

therapy are particularly effective with patients with FAS/FAE" (Streissguth, 1994, p. 75). Further, Streissguth explains that one program was deemed successful because emphasis was placed on *prevention* rather than alcoholism or social work. Recognizing that culturally relevant interventions tend to be the most effective, Streissguth (1994) concludes:

> FAS is preventable. FAS prevention is as simple as getting women to stop drinking during pregnancy and as complex as changing community attitudes about drinking and alcohol abuse. . . . A single community, acting on its own volition (but using state and federal resources through key public health and safety workers), can use local action to bring about significant community change from alcohol to sobriety. (pp. 62-63)

Parker-Langley (2002) suggests several shortcomings in research pertaining to secondary prevention. She notes the following concerns:

- Small sample sizes.
- Lack of comparison groups.
- Poorly explained interventions.
- Interventions not sensitive to cultural context.
- Limited follow-up approach prohibits assurance of any lasting effect of intervention.

Tertiary (Cultural) Prevention: Policy

Parker-Langley (2002) suggests that strategies for intervention through alcohol control policies or laws have been tried for the past 165 years. Unfortunately, unless these strategies originate with the Indian people themselves, they will be unsuccessful.

Lauzon, Gregoire, Gliksman, McKay, and Douglas (1998) discuss the Mattagami First Nation's policy to reduce harm from alcohol. The authors describe the development and implementation of a policy to govern the use of alcohol specific to the Mattagami, an Aboriginal community in northeastern Ontario. The policy was adopted June 7, 1993. Rules pertaining to where alcohol can and cannot be served, youth attendance limitations, need for posters, training of servers and door monitors, need of non-alcoholic beverages, designated driver programs, no drinking by workers putting on the event, and restriction of the numbers of drinks that can be obtained at one time are identified. One poster specifies a warning to expectant mothers about the potential harmful effect of alcohol to babies. Lauzon et al. suggest the effect of this policy includes the reduction in fights and fewer underage individuals being served alcohol. Although positive results are reported regarding the legalized use of alcohol, few tribal councils (i.e., only five) have implemented this legislative option.

Hamilton and Snyder (1995) provide an overview of a training program that was established to meet the objective of the Indian Health Care Improvement Act (P. L. 102-573, section 708). The objective was "to reduce the rate of FAS to one per 1000 live births among

American Indians and Alaska Natives" (Hamilton & Snyder, 1995). Awareness of this project led to increased requests for training.

Another example pertains to a resolution put forth by the Montana-Wyoming Tribal and Urban Grassroots Coalition of Chemical Dependency Counselors (Kopera-Frye, Tswelnaldin, Streissguth, & LaDue, 1994). During a training session, 18 counsellors developed a resolution to be put to the Tribal Action Plan, recommending that all communities be encouraged to adopt all or some of the resolution. Essentially, this resolution speaks to the need for training for all individuals who interact with persons affected by FAS. Further, counsellors identified the need to ensure that pregnant substance-abusing women are priorized for treatment, that screening processes be implemented, and that the outcomes of the screening was not to be used for purposes of prosecution. Several positive initiatives ensued following the presentation of this resolution, such as an increase in the number of training sessions, the opening of a Women's Healing Centre for purposes of providing residential alcohol treatment for pregnant women, and the development of a newsletter.

The Substance Abuse and Mental Health Services Administration (SAMHSA, 2004) has six legislative mandates, one of which is to study adaptations of innovative clinical interventions and service delivery improvement strategies. The second mandate pertains to the creation of a national inventory of FASD specific programs. In 2002, the SAMHSA sponsored site visits to 10 tribal groups and five health centers for purposes of gathering information about local practices regarding FASD. The findings reflect the pervasive incidence of FASD among the American Indian/Alaska Native population (i.e., three times the rate among non-Natives). Findings include the following:

- Only one tribe has a paid FASD coordinator.
- Only one of the cities has an FASD clinical coordinator.
- No tribe provides coordinated services.
- Services for children relate to the Head Start Program.
- No services for adults unless they are severely disabled.
- Most sites had evidence of good programs.

These findings speak to the need "to identify support, and promote effective prevention practices and to build capacity" (p. 3). Interestingly, the Indian Health Services provided such a service. However, this role and responsibility was transferred to Indian communities through self-governance initiatives. Unfortunately, without the financial and educational support, focuses on FASD and practices to enhance prevention have been lost.

Tertiary (Cultural) Prevention: Research

Tertiary prevention acknowledges the existence of the *problem* and, hence, strategies to reduce or eliminate the long-term effects of that problem. Thus, tertiary interventions are typically identified as *treatments* (e.g., clinical screening programs).

Grant, Ernst, and Streissguth (1999) describe the "administrative components of an effective home visitation program for high-risk alcohol and drug-abusing mothers" (p. 1), identifying effective principles and practices. They position their Parent-Child Assistance Program (P-CAP) within a model of paraprofessional advocacy based on relational theory. Two major theoretical concepts are motivation as a process and ambivalence about change. The framework of the P-CAP intervention was also influenced by harm reduction theory. Given the assumption underlying this theory, the goal is to support clients to move along the continuum from excessive use of alcohol and drugs to abstinence. Importantly, the P-CAP, a three-year program, was recognized as one of a few successful intervention programs nationwide, which received federal funding. Paraprofessional advocates have overcome difficult life experiences similar to those of their clients and, therefore, can inspire hope. Moreover, they represent diverse ethnic backgrounds (e.g., African American, Caucasian, Native American, and Pacific Islander). This advocacy approach serves two major functions: the paraprofessionals serve as role models and provide practical assistance in the client's home as well as ensuring clients have access to various community services. Essentially, the P-CAP acknowledges the significant role of paraprofessionals working independently to support a generally non-compliant high-risk group of mothers. The success of this Program is evident in the ongoing funding provided by private sources, the strong retention of advocates, and the success of the clients.

Kvigne, Bad Heart Bull, Welty, Leonardson, and Lacina (1998) "examine the maternal and prenatal factors that were associated with alcohol use in the study that validated the screening tool" (p. 214). The data collection procedures include several activities: prenatal patients complete a self-administered questionnaire, public health nurses review the information provided on the questionnaire with the patient, a nurse researcher interviews each patient, and review of medical charts occurs post delivery to obtain any information regarding alcohol-related injuries or illness. Of the 177 American Indian women participating in the study, 44% did not drink during pregnancy and 56% did consume alcohol while pregnant. Further, of those women who did drink during pregnancy, only 23% reported this information on the questionnaire; the rest were detected during the interview and medical chart review. Among those women who drank alcohol while pregnant, "the only statistically significant difference was binge drinking before pregnancy which was reported by 87.8 per cent of the former group and 65.5 per cent of the latter group ($p = 0.01$)" (p. 215). Other findings indicate that women who drank were 24 years of age at delivery, single (77%), had less education (11 years), and less access to transportation. These women tended to drink more alcohol per occasion with binge drinking being the usual pattern of drinking before and during pregnancy. Women who drank alcohol while pregnant tended to smoke cigarettes and lived with a partner who also drank alcohol. Kvigne and colleagues conclude that there is a need for programs and especially those programs that encourage family support as well as the need for treatment opportunities for women and their children.

Parker-Langley (2002) espouses that the success of prevention programs among Indian communities, and perhaps all culturally diverse communities, rests upon two factors: researchers must truly adopt an "insider's view" of these communities and communities must commit to both structural and programmatic changes necessary to realize long-term positive

outcomes if future generations are to benefit. Streissguth (1994) places the responsibility to prevent FASD with every individual, when she states:

> Every person in every community has the responsibility to do what he or she can to safeguard the next generation; to prevent FAS, and to help create an environment that will be safe and healthy for our children, both before and after birth. It is respect for life. It is essential for our continuity. (p. 77)

Summary

There is a common perception that the incidence of FASD/FAE is greater among Aboriginal peoples. Indeed, many statisitics confirm this assumption. Several governmental initatives invested significant resources (i.e., people and funds) in an effort to combat alcohol abuse among Aboriginal peoples. In some instances, Aboriginal communities have successfully implemented policy to control alcohol consumption within their territories. Unfortunately, these efforts have not always been sustained.

Further, Aboriginal peoples have been the focus of many research activities. For instance, researchers endeavoured to determine the knowledge base of health care professionals, pregnant women as well as school children. Overall, the knowledge base is lower than one would like given the devastating effects of FASD/FAE. Nonetheless, there are a few programmes that appear to promote positive health outcomes.

Chapter Fifteen:

Best Practices: Education

While practice is often associated within a clinical setting, education is also critical as part of prevention and intervention. Clearly, health care professionals cannot address issues such as FASD without substantive knowledge and skill and the ability to work within a inter-disciplinary approach, as previously stated. Therefore educational models of necessity need to reflect the knowledge needs of health care professionals and the main stream teaching community. After all only when health care professionals and teachers are well prepared themselves can they provide the essential preventative and intervention strategies for client populations and their significant others. The literature examined in our review addresses educational programmes from clinical and school-based perspectives. Furthermore, a focus is given to the need for educating the educators to ensure they are competent to practice.

Health Care Professionals Educational Needs

Recently developed training centers for FASD diagnosis and intervention for healthcare workers appears very promising (Sharpe et al., 2004). These training centers develop practical methods of disseminating FASD curriculum through a variety of means, each aimed at specific FASD knowledge or training needs and for varying occupations within the FASD support teams. National experts within the United States and Canada are involved in the curriculum creation and ongoing evaluation of this multilayered and multi-disciplinary approach to FASD training. This work is to be commended because the lack of knowledge and preparation of Health care workers and teachers were often cited regarding failure to diagnose and effectively treat FASD.

Clinic-Based Programmes

Clinic-based programs are one area in which knowledge of women can be assessed and, if needed, addressed through information sharing and through follow-up support.. Self-help programs, cognitive therapies and behavioural management programs have been effective in reducing problem drinking (Murphy-Brennan & Oei, 1999). Providing educational materials and information is more likely to occur in clinics where the staff is educated and has received FASD training (Bowden & Rust, 2000). Unfortunately, information dissemination and advice alone does not ensure alteration of behaviour of a social drinker, nor, does that enable early identification of a problem drinker, two key factors that are associated with primary prevention of FASD. To overcome such a dilemma it is important to pair reading or media-based text with counseling. This approach will help to address the predisposing, reinforcing, and enabling factors that place certain individuals at risk.

Clinics also provide opportunities to holistically assess and diagnose women for alcohol consumption and their children for FASD. Moving on such a perspective The Fetal Alcohol Syndrome Diagnostic and Prevention Network at the University of Washington has developed a diagnostic model to be used in the clinical setting in which mothers of FASD

children are very receptive to interventions (Streissguth, 1998). Within these setting opportunities encourage the inclusion of Inter-professional practice that can diagnose, support, intervene and educate FASD affected families.

Another area of good practice that has been identified within the literature is ***Breaking the Cycle program at the Toronto Hospital for Sick Children*** (Avner & Koren, 2004; Leslie & Roberts, 2001). In this practice setting, pregnant and parenting women attend the clinic program with their children to obtain follow-up support and identification of factors that impact with, and on, FASD effects. Part of the model has recognized the need to operate within a blame-free and a non-judgmental framework given that FASD evokes inherent shame, guilt and fear associated with alcohol use in pregnancy. This program is an offshoot of the Motherisk clinic, a large teratogen-information service in which staff counsel over 100 individuals per day regarding exposure to: alcohol, drugs, chemicals, and radiation and infections during pregnancy (Gladstone, Levy, Nulman, & Koren, 1997; Koren et al., 1996).

The success of all clinical based programmes is associated with the ability to increase awareness in females during their child-bearing years. ***The Healthy Touch***, an interactive multimedia program, aimed at alcohol education for low-income pregnant women, was found to be effective (i.e., 81% of women said they learned why they should abstain from alcohol; Kinzie, Schorlng, & Siegal, 1993). This program provides factual information related to the effects of alcohol consumption during pregnancy. It also addresses existing health-related beliefs, models behaviour changes, and personal control over one's behaviour.

School-Based Programmes

School based educational programs dealing with alcohol consumption, FASD effects and general drug and alcohol knowledge were identified with prevention of FASD (Bowden & Rust, 2000; Murphy-Brennan & Oei, 1999). Changes in student attitudes and behaviour were more likely to occur in community-based programs rather than teacher directed classes (Murphy-Brennan & Oei, 1999). For example a community approach to teenage alcohol reduction targeted grade six students with interventions ranging from parental involvement and education, social curriculum in school, peer leadership, and community wide task forces (Perry et al., 1996). Results of this approach uncovered a decreased onset and prevalence of alcohol consumption for students involved in this program.

Increasing public awareness by beginning with preadolescent knowledge of alcohol consumption and pregnancy will likely affect beliefs and behaviour changes. Primary prevention strategies that nurses or healthcare workers could adopt when working with the adolescent population is to teach them about FASD, inquire about alcohol use, educate both males and females in a co-ed environment and encourage contraceptive use for those who are planning to become sexually active (Allard-Hendren, 2000). Assessing students' knowledge of alcohol effects prior to educational sessions will assist in determining their needs, beliefs and current behaviours (Haemmerlie, Merz, & Nelson, 1992).

The literature draws attention to the fact that school aged students should be provided with educational programs that incorporate; (i) risk factors, (ii) effects of alcohol use for young adolescents, (iii) protective factors, that include active involvement in social and

physical activities and, (iv) the need to strengthen the family relationship and environment (Ma, Toubbeh, Cline, & Chisholm, 1998b). Such an approach serves to inform the next generation of parents with a body of knowledge and understanding to the dangers of alcohol consumption and the production of a healthy child (children) and family. Education in its various forms is indeed necessary to all members of society if FASD/FAE is truly to be eradicated.

Gaps in Research and/or Practice

So far we have highlighted areas of good practice from and educational perspective, but the literature also suggests that several gaps in our knowledge of effective educational and clinical practices persist. For instance there was a distinct lack of:

- Substance-abuse services within communities, in particular for prenatal substance abuse, including services for women only (Leslie & Roberts, 2001).
- Coordination of services and interagency best practices.
- Knowledge regarding the FASD population in Canada, provincially or within specific communities.
- Knowledge among adolescents and the general population, including pregnant women and men about the causes of FASD and the consequences of alcohol abuse.
- Trained staff, especially qualified individuals for purpose of diagnosis, assessment, interventions and family support.

Summary

The focus of educational practice pertains to initially ensuring a knowledge base among health care professionals in all disciplines and members of the teaching profession. Given the emphasis on the literature on the lack of preparation of these professionals attention needs to be directed to the development of curricula and education and training opportunities that fully reflect contemporary and evidence based knowledge. The needs of women and children are severely affected when the professional workforce lacks the competence to assess, diagnose and treat FASD/FAE. In turn informed professionals are best positioned to effect change in clinic and school based programmes. Indeed, it is encouraging to realize that several programmes are deemed to be effective such as the *Breaking the Cycle Programe in Torronto,* and *FAS Diagnostic and Prevention Network in Washington.* Nonetheless, several gaps are noted and further research is required.

Chapter Sixteen

Best Practice Evidence Review

Barriers to Screening

Research studies have repeatedly proven that health care providers fail to evaluate the alcohol and drug consumption behaviors of clients on a regular basis. Specific references were made to the fast paced medical office environment and personal discomfort with asking difficult questions. In addition, health care providers expressed concern about the ethics surrounding raising issues when the community lacks the necessary services to treat identified addictions (Donovan, 1991).

Brief Alcohol Screening Questionnaires

Minimization and denial are integral aspects of alcoholism. A number of simple screening tools have been reported to be sensitive in identifying women requiring a thorough assessment of their intake of alcohol and/or drugs. Researchers recommend that professionals administering the CAGE, TWEAK and AUDIT to women of childbearing age suggest the use of lower cut points (Becker, 2001). A two-item conjoint screen for alcohol and drug use (TICS) was found to detect 80% of substance abuse in young and middle-aged patients (Brown et al., 2001). With consideration to providing professionals with mechanism for comfortably engaging the patient in a discussion of their intake of alcohol, researchers developed and tested the "one question" screening tool for women, which asked "When was the last time you had more than four drinks in a day?" Researchers compared the CAGE and the single question format, assessing the frequency of use, patient/clinician comfort and the patient's engagement in the change process. They concluded that both clinicians and patients were comfortable with both tools and that the two instruments were equal in their ability to engage the patient (Vinson et al., 2004).

Universal Family Stress Assessment Screening

The Healthy Families America model incorporates a multi-faceted screening process that identifies women who are prenatal or postpartum, which may place their child at risk. Using the Kempe Family Stress Assessment Checklist as an initial screening tool, factors are identified that are known to be associated with less than optimal birth and child development outcomes. While the use of alcohol and drugs during pregnancy is specifically addressed, the format also identifies homelessness, domestic violence, food insecurity and isolation as essential issues to be considered.

Identification of High Risk Groups

Women with alcohol problems commonly seek help through their primary care physician. Clinicians often treat the symptoms of alcoholism without diagnosing the disorder. Women with alcohol disorders may present with multiple complaints, none specific to alcoholism. This group has a higher incidence of mental illness, panic or phobia disorders, eating disorders, PTSD and victimization (Becker, 2001). Women who give birth to children with FAS/FAE have similar characteristics. They tend to be older, have higher parity, lack of

prenatal care, mental health issues, alcohol-related injuries and are frequent drinker with binge drinking behaviors (Railton, 2003).

Comprehensive Alcohol Screening Instruments

A more detailed assessment of the client's alcohol and drug use can be accomplished through the use of research-based instruments such as the Addictions Index Severity Scale (ASI) or the Substance Abuse Severity Scale Inventory (SASSI). Women who fear losing custody of their children may disclose their true substance use in small portions. The ability of the clinician to establish an atmosphere of trust of mutual respect is paramount in the attainment of a true picture of the client's addiction history.

Physical Findings

As a stand-alone screening tool, the physical examination is the least specific. The detection of the odor of alcohol on the breath of the client indicates that they generally have a blood alcohol level (BAC) greater than 125mg/dl. A subtle odor indicates a BAC between 75-125mg/dl (Becker, 2001). Physical findings, which can be associated with chronic alcohol use, include the presence of unexplained bruising, spider angiomas, palmar erythema, and distal extremity hair loss and poor dental health. Of particular focus should be the evaluation of a client's complaints of insomnia, which is frequently associated with alcohol consumption (Brower, 2003). In addition, a careful assessment of the client's nutritional status is warranted (Abel, 1994).

Brief Focused Intervention Models

Treatment Models

FAS Prevention Models
Maternal Focus
Child Focus
Family Focus
Child Abuse Prevention
Mental Health/Addictions (Dual Diagnosis)
Culturally based
Judicially based

Home Visitation Models

The literature describes a long history of programs being applied to improve outcomes for children and families. A number of programs with nurse home visitors were studied where the goals were to reduce maltreatment of children, improve health (birth weight, developmental outcomes), decrease parent criminal behavior, subsequent pregnancies, use of welfare, etc. Many of the studies reported improved outcomes with a range of program input (number of visits/hours spent with the mothers/children). More needs to be known about the level of intensity that is adequate. How many visits or hours and what breadth of intervention (identify and address the whole scope of family need) is necessary?

The results from one 20-year study (Eckenrode et al., 2000) have been frequently cited as evidence of the potential of home visitation programs to prevent a host of societal ills and have consequently increased the popularity of this modality. The result has been an expansion of home visitation and a large variety of home visitation models being implemented, including Parents as Teachers, Healthy Families America, Early Head Start, etc.

The Eckenrode et al. study identified that outcomes did not improve for those children living in homes where domestic violence was present and not addressed (Eckenrode et al., 2000). Another study of early intervention with adolescent mothers determined that "greater efforts need to be directed toward preventing repeat pregnancy and return to substance use following childbirth in at-risk adolescent mothers" (Koniak-Griffen et al., 2002). Although some of these studies identified reduction of alcohol consumption as a goal, they did not specifically name reduction of FASD as a goal. Ebrahim (1999) comments:

> The recent increases in reported rates of binge drinking among pregnant women highlight the need for measures to sensitize women about the dangers of binge drinking during pregnancy. Efforts to reduce alcohol consumption among teenagers and young women may benefit from concerted programs focusing on family planning and the prevention of polydrug use and sexually transmitted diseases.

One of the limitations of the data collection in the research has been client report rather than factorial design. The lack of hard evidence leaves the reader without a conviction about study conclusions, especially in relation to alcohol and polydrug use (Koniak-Griffin et al., 2002).

The literature clearly indicates that a majority of women (particularly adolescents) will stop or decrease their alcohol consumption when pregnant either of their own volition, or as a result of education or awareness campaigns or short health care provider interventions.

For the group of women who continue to consume alcohol at a risky rate during pregnancy, a greater intensity of intervention is needed if FASD is to be prevented. The literature describes targeted programming with the goal of addressing the needs of this group of women in regard to reducing harm or preventing FASD.

Project CHOICES Intervention Research Group described positive outcomes with women at-risk of having a child with FASD (alcohol use and lack of contraceptives). This intervention was designed to be used prior to contraception. It consists of screening for risk, inviting voluntary participation, four, manual-guided motivational interviewing sessions, one contraceptive counselling session and a six-month follow-up. They discovered at the end of the six-month follow-up that of the 75.3% of the women who participated in the follow-up, 68.5% of them were no longer at risk of having an alcohol exposed pregnancy according to client report. The investigators recommended additional study using a randomized controlled trial.

The Pregnancy and Health Program was established at the Washington State University Medical School in 1978. This project developed three major divisions: (1) public

education and professional training, (2) client services, and (3) evaluation. The client services division developed a program model called Parent Child Assistance Program **(P-CAP)** or **Birth to Three** to address the needs of this higher-risk group. The goal of this model is to provide a range of interventions at a level of intensity that reduces the risk of having a child with FASD. The intensive level of service is flexible and responsive to client growth allowing for adequate interventions depending on the circumstances.

Typically, during the first year of participation, the client receives at least one and one-half hours of time with her advocate in addition to transportation for essential appointments or services to access health care, housing, financial support, food, counselling, detoxification, treatment, etc. The focus of intervention is stabilization.

The transition to the second stage of program participation occurs when the woman has achieved some degree of stability. The focus at this point is to assist her in achieving some movement toward longer-term goals and to help her keep connected to those formal and informal community resources that help her maintain sobriety and stability. Typically, during the second year (or stage) of service the woman receives visits of one and one-half hours every second week, again, with the addition of support for meeting needs as described previously. The third and final year of inclusion in this program focuses on independence, allowing for the advocate to support the woman through crisis if they arise, regular check-in visits of once a month and phone contact where indicated.

The P-CAP model uses paraprofessional Home Visitation model. From the P-CAP program, discussing use of paraprofessional advocates:

> The skills that professionals and paraprofessionals bring to the intervention are distinctly different, but complementary and both have advantages. Professional credentials may instill trust on the basis of their expertise. On the other hand, paraprofessionals with shared cultural and some common experiences may understand clients in a way that allows them to gain access and build rapport with women who might otherwise be unapproachable.

Advocates form supportive, mentoring relationships with the women on their caseloads. The caseloads are small (12-15) to allow for the intensity of the intervention.

The P-CAP model was adapted to address the special needs associated with clients with suspected or confirmed FASD. Specifically; program goals over three years included diagnosis, family planning, PDD services, alcohol or drug treatment and aftercare, protective environment, and a solid network of community service providers. Finally, advocates attempted to locate long-term mentors for clients (Grant et al., 1997, 1999, 2002). While specific evidence was not found in the literature to suggest that mothers with FASD may never be successful parents within a supported environment, the P-CAP model provides a set of goals that may be adequate to address the unique needs of this target group. More outcome evaluation is needed.

In Alberta, the P-CAP model has been adopted and implemented in Edmonton, Calgary, Lethbridge and on the Blood Reserve in Southern Alberta.

In Lethbridge, additional elements include the following: infant nutrition, infant and child developmental stimulation, assessment and referral, positive parenting practices, umbrella of the Health Region (screening, assessments, referrals, consultations, CARE services, fleet vehicles, office space, connection with other health professionals (PHNs, Sexual Health, etc.).

Characteristics of Successful Home Visitation Programs

Intensive level of service that is responsive to client growth
First year: 1.5 hours or more per week (excluding transportation time)
Second year: Visits every other week
Third year: Transition to monthly and as needed visits
Lifelong community based support for clients with FASD
Relationship based
Trust
Perseverance
Role model
Strength based
Non-judgmental
Client/family centered
Help client get appropriate services (based on client goals)
Starts with the basics
Food, shelter, safety, transportation
Financial support/prenatal care/family planning
Diagnosis for Adults strongly suspected to have FASD
Addictions treatment
Social Services (advocacy)
Legal support (advocacy)
Domestic violence support
Counseling
Keep client connected with children in care

Gaps

- Soft data regarding actual substance use of clients (self-report versus laboratory screening).
- Lack of research into programming impact on the protective factors of preconception and perinatal intervention programs (nutrition, harm reduction, ultrasound research, etc.).
- Inclusion of male partner in programming.

- Lack of hard research into the environmental factors, which could be proven to enhance brain growth from birth to three, as well as minimize the development of secondary disabilities.
- Diagnostic criteria based on hard data versus self-report.
- Strength based neuropsychological testing for FASD affected clients.
- Family inpatient addictions program (possible research into the drinking habits of children of alcoholics who are included in addiction treatment programming throughout the lifespan).
- Ultrasound.

Summary

Best practice evidence review clearly illustrates the fact that most health professionals do not have either the knowledge or skill to undertake a comprehensive screening that identifies women at risk whilst using alcohol during pregnancy. Ethical concerns were also highlighted with respect of identifying women at risk and then having insufficient services to address the health needs of the woman and her family. However, some view this situation as a professional cop out rather than a real need to serve the health needs of women and prevent a human tragedy, a child with FASD/FAE. Our review identified useful screening questionnaires such as CAGE, TWEAK, AUDIT, and The Universal Family Stress Assessment Tool. However, all the literature pointed to the malady of the use of self-report. Other screening tools involve labaoratory analysis such as the Substance Abuse Severity Scale (SASSI), which can identifiy the levels of ethanol in the blood. Further, physical examinantions can also reveal other disease or functional loss due to the abuse of alcohol.

The real dilemma professionals have is gaining trust from women that operate in a framework of mutual respect. Mutual trust then allows for a true record of alcohol consumption been disclosed. Such a model is essential in the client-professional relationship but is so often absent.

Our review demonstrated that home visitation models are effective and there is evidence that there is improvement in health outcomes for the child, the mother and the family. Enabling the family to become involved in maternal alcohol reduction is a powerful key to success. However, the literature states we need more evaluation. It is also clear that there are some gaps in the literature (previously identified) that should assist in professional's aquiring best practice from an evidence-based position.

Chapter Seventeen:

Conclusion

This literature review has undertaken a methodological approach towards the retrieving of scientific articles and the grey literature in an attempt to synthesize the evidence pertaining to FASD/FAE prevention. Our methodological section clearly illustrates the approach taken, which includes our rigor and robustness in not only retrieving the articles, but also in applying a critical and standardized approach towards the reading and synthesizing of the information. In our quest to seek out the evidence, it became evidently clear that few random controlled trials in the prevention arena had been undertaken to date. Most of the literature relates to the grey area, and whilst this literature may be useful in raising awareness to certain issues pertaining to FASD, it does not allow for empirical knowledge to be formulated and, thus, sufficiently inform policy, practice, or education.

We explored prevention from the angles of Primary, Secondary and Tiertiary perspectives, and used the frameworks of interpretations defined by the World Health Organisation (1998). Further, we have incorporated an historical overview to clearly show that the knowledge of the consumption of alcohol on the fetus has been known throughout the ages. There are biblical and religious reference, social policy, and, in addition, some early animal investigations to establish empirical evidence. Most of these studies were concluded during the early 20th C and little more information was established until 1973 when Jones and Smith identified that children born of women who drank alcohol had a series of characteristics that were all similar. They were one of the first people to clasify and codify these characteristics into a medical label known as Fetal Alchol Syndrome (FAS), recently changed to FASD/FAE to better reflect the complexity of the diagnosis.

From those early observations and identification of the characteristics associated with FAS the modern day literature has identified further elements that we have catalogued under the auspices of: cognitive, physical, and affective domains. With such a constellation of characteristics, assessment and diagnosis is difficuilt for the modern day health professional. In our diagnostic chapter, we have illustrated, from the literature, a selection of tools and instruments that are used in contemporary practice. These instruments and tools have, in themselves, been called in to question relating to their efficacy and effectiveness in assisting in the diagnosis.

Through the process of our reading we established that the literature was devoid of substantive theoretical frameworks to support, or, negate the emerging evidence. We have, therefore, drawn from seminal pieces of work from a variety of disciplines and have expanded on the notions of: prevention, lifestyle as a health construct, coping strategies, health beliefs, health locus of control, social marketing, the stake theory within an Aboriginal focus, social support and social networks, and empowerment as a health coping strategy. These theories should inform and shape practice and educational models, and provide a framework through which policymakers can substantiate their policy directives. For example; health belief changes cannot occur effectively over a sustained period if health professionals

do not ascertain the women's health belief systems in a cultural and sensitive framework. Nor can we ascertain social support from a positive perspective if their current support systems are neither identified nor understood. Evidence emerging from social marketing strategies must be heeded if future mistakes are to be avoided. Therefore, in our social marketing chapter, we have identified from the literature positive and negative approaches.

There is much debate and attention given both in the media and within the political rhetoric that the incidence and prevalence of FASD/FAE is on the increase and many cases are undetected that should be notified. There is a universal problem with recording the incidence and prevalence, chiefly, because (i) there are differences in recording prevalence and incidence, (ii) there is no universal standard, (iii) diagnosis is difficuilt with the new born, (iv) low data quality, and, (v) retrospective studies used as a predominate method. The demographic data are equally flawed in that Aboriginal peoples are more likely to be assessed for their maternal alcohol consumption and users of illegal substances than other population groups. Through our review we tried to unravel the reasons why women drink during pregnancy, firstly, and an important point to make, is that women may not know they are pregnant particularly in the first few months when the animal literature tells us that ethanol consumption has a profound effect on the developing fetus. Secondly, alcohol consumption is a social activity and peer pressure is very strong. Thirdly, women may be drinking due to psychological and environmental factors.

While we have alluded to the fact there are many reasons why women drink alcohol, this situation is further exacerbated due to the polarized views emerging from the global policy literature. We have identified that Canada and the USA have adopted a zero acohol positon whilst other countries' political statements suggest one or two drinks a day is within safety boundaries. These controversial views emerge because there is no definitive information relating to human threshold levels. Further, policy directives are often inter-related to laws and offer a punitive position, which appears to have some ethical dimensions and challenges and portrays women in a blameful and degrading manner. This approach does not encourage openness, nor helps to enage the client and health professional in a trusting and respectful working relationship.

It is clear from reading the literature that there are risk factors that are associated with FASD. It is suggested that certain groups are more at risk than others, such as the Aboriginal people. Whilst this statement is challenging, we need to consider this in light of their scoicioeconomic standing and their nutritional status and environemental factors. In Table 7, we have correlated risk factors other than alcohol such as age, parity, race, SES, marital status, tobacco use, and other susbstance abuse. All of these factors, the literature suggests are important co-morbid factors in the risk analysis. Clearly, risk assessment is subjected to further investigatory work been undertaken to arrive at a more definitive position.

Current attention is given to expanding knowledge and understanding through exploration of the effects of alcohol on the brains of FASD children in ways that is only made possible now due to advancements in technology. Evidence from brain imaging clearly supports that children with FASD have microcephaly, as well as structural abnormalities to the cerebellum, corpus callosum, and basil ganglia. Further to this, work is ongoing to

establish a genetic susceptibility towards FASD. Heritability of trait is obtained by examining the recurrence of risk in relatives. Twin studies provide some explanations towards this concept. However, intergenerational understanding of alcohol consumption would provide further information. What is established is that the susceptibility to FASD is influenced by the genotype of both the mother and fetus. In addition, there appears to be some exacerbating effects, which have been recognized and discussed within the literature. For example: ethanol use during pregnancy affects cytokine synthesis; ethanol exposure programmes the body to insulin resistance; and DNA may be damaged, which contributes to organ dysfunction.

Emphasis in the literature is clearly focused on maternal alcohol drinking and the effect on the fetus. But there are some earlier studies that have recognized paternal alcohol consumption and the effect on the sperm and, recently, there are suggestions of an indicative relationship between neonates being born with low birth weight. Notwithstanding, there is a need for further investigations into the effect of paternal alcohol consumption and possibly any other drug ingestion that may have a co-morbid effect and including tobacco.

Recent literature has questioned any mediating or protective factors that are associated with alcohol consumption and FASD. These include antioxidants as natural phenomena and other molecules such as vitamins, folic acid, micro-nutrients and other substances such as gangliosides.

Prevention strategies include an educational element aimed at all levels, including populations, communities, school children, other students, health professionals, and targeted at women at high risk. Social marketing directs energy and resources at educating populations, communities and groups of individuals through a variety of media modalities. Unfortunately, evidence suggests that such approaches through labeling, posters etc. have a limited and short-term effect. Other approaches are linked to mandatory legal distribution of brochures and on products to offset any responsibility of the manufactures. Labeling of the dangers of cigarette packages is a clear example of this approach. But this tactic does not take into the account the addictive nature of substances. More successful marketing approaches are those that engage the community in taking responsibility for the health and well-being of the community and individuals who reside in the community. With respect of FASD programs can these can target women at risk, or those who are known to be pregnant. To be effective over the longer term programs need to be directed at the lifestyle and living conditions of individuals within a cultural context. In this way education programs can be more informative and bring about lasting changes that improve health outcomes. In our Social Marketing chapter we have compiled a Table that summarizes proposed social change campaigns, that is, best start programs; changes that relate to social norms; awareness raising with the young; and correct misconceptions of health professionals.

We have so far alluded to the notion of culture and how important this aspect is with regard to altering lifestyle behaviours. Our review, clearly illustrates how there is a fundamental lack of knowledge of cultural diversity amongst the health professional community. Such a dilemma is a clear barrier to achieving successful health outcomes. Researchers have commented that the success of progams related to the Aboriginal

communities need to have an insider's worldview of the situation and the communities themselves must adopt attitudes that support structural and programmatic changes necessary to realize long-term positive outcomes if future generations are to benefit.

The onus rests with health professionals who we expect will have had the necessary training and education to be truly competent to practice. Sadly, the literature repeatedly draws our attention to the fact that health professionals, the judicial system, and teachers are unprepared to work in the field of FASD. Most lack common understanding of the concept as well as a knowledge base as to what models of practice can best influence positive health outcomes. Whilst we can believe this is wholly unacceptable practice, as mentioned earlier, there is confusion centered on the quality of the diagnostic tools, and advice on what are the best tools and instruments to use in clinical practice for screening, assessment, and diagnosis. Furthermore, there are barriers to effective screening that are fundamentally due to the pace of medical consultations and the lack of opportunity to explore difficult questions. More effective, and more costly, are home visitation models that do improve health outcomes for women, their affected children and their immediate family members. The literature has identified characteristics that support home visitation programs such as, intensive level of service that is responsive to client growth, lifelong community based support for children with FASD; trust and respectful relationship between client and health professional; perseverance, role model; client- and family-centered approach within a blame-free environment.

Our review has examined a range of concepts that underpin prevention regarding FASD. Whilst there is some growth in the knowledge base there remains a lot of areas that require further investigation from which increased knowledge can more effectively inform the public, health professionals, practice, education, and policy. Animal studies clearly inform us of the dangers of ethanol effects; however, these investigations need to be further examined in the human form. Genetics will also provide future knowledge and a greater understanding as will evaluation of best practice models. The last four decades has provided a benchmark for more rigorous scientific enquiry to take place; however, we believe that the next decade will direct the research areas specifically required so that we can truly state we have sound empirical evidence from which we can prevent and treat FASD/FAE. This being said, we hope that more effective and robust marketing strategies (that will be evaluated) will have such an impact that no child inthe next decade will be born with FASD/FAE, thus preventing a human tragedy.

Recommendations for Education

- Education is required to inform health professionals on the positive and negative health coping strategies that can serve to prevent secondary and tertiary interventions.
- Health professionals require a comprehensive understanding of, and the necessary skill to undertake, assessment and diagnosis of FASD/FAE.
- Health professionals need to understand theoretical frameworks that underpin best practice and prevention intervention strategies.
- Health professionals need to be able to work inter-professionally, and within inter-agency frameworks, so as to provide the best possible health outcomes for their clients, and their families.
- Health professionals need to explore the health beliefs of clients and their families to improve health outcomes, through compliance with the necessary behavioural changes.
- Health professionals need to have knowledge of prevalence and incidence gathering modalities so that they can contribute to the data gathering of incidence and prevalence of FASD/FAE.
- Health professionals require knowledge of protective factors such as antioxidants and the role that they play in the prevention of FASD/FAE.
- Health professionals require a sound grasp of social marketing theories and their effectiveness and efficiency in preventing FASD/FAE.
- Health Promotion and Health Education should be the central focus of health professional's work. In essence, health prevention is everyone's business.
- Education relating to the effects alcohol consumption has on the fetus should be undertaken in all schools, with adolescent women and men, communities and wider population groups, using a multi-media approach that is culturally sensitive.
- Cultural diversity issues must be understood and applied in clinical practice.
- Education within the clinical setting can be most effective, therefore, health professionals must seize every opportunity to inform the public and target meaningful education to women at risk.
- Revise basic health curricula to include knowledge, skill, understanding and experience of FASD/FAE.
- Provide training and knowledge for all health care professionals about related support and resources available to them in treating FASD. This training could include support and resources geared to diagnose, intervention and prevention of FASD.

Recommendations for Practice

- Practice models or instruments should incorporate coping assessment as part of their screening approach.
- Use a multi-service surveillance system to develop and offer appropriate services to children and their families including prevention strategies for women in child-bearing years.

- National pooling of FASD research findings and best practices for dissemination should be applied in the practice setting as part of evidence-based strategy.
- Health educators in a variety of clinical settings should unite to come in contact with women of child-bearing age in order to disseminate information related to alcohol consumption during pregnancy.
- Highlight the need for early diagnosis of FASD/FAE so that appropriate interventions can be established over the life of the child.
- Prevention programs for women should provide a range of services such as brief interventions, counselling, social support, prenatal classes, parenting classes, and mental health services.
- Incorporate evidence from scientific enquiries to provide a framework for evidence-based practice.
- Develop good inter-agency and multi-professional working relationships and communication systems using a client-centred electronic record system.
- Include male partner in programming.
- Empower communities to assist in prevention programs.
- Further examination to establish biochemical markers.

Recommendations for Research

- Evaluation of promising prevention programs.
- Studies utilizing large, representative Native populations to measure factors that put women at risk such as: socioeconomic factors, environment, cultural norms, health beliefs, nutritional state, and social support systems.
- Investigations into the ways in which women and families cope with FASD/FAE measuring those that have a positive health outcome and those that produce negative ones.
- Development or adaptation of health coping assessment tools used with FASD.
- Multigenerational research to determine the influence of family circumstances and habitus.
- Assess the reasons why women do not engage in treatment programs.
- Evaluate the effectiveness of antioxidants as a protective substance to alcohol abusing pregnant women (there have been no human trials to date).
- Undertake research to establish the true nature of mediating factors such as genetic make-up, nutritional state of the women, and their physical, psychological, social, and spiritual wellness.
- Undertake research to evaluate the effectiveness, efficiency, and economy of programs in practice.
- Explore the effects of various environmental factors.
- Further examine the threshold limit of alcohol consumption pre-conceptual, during pregnancy and through the period of lactation.
- Explore the relationship of FASD and binge drinking through longitudinal and cross population studies.

- Explore factors that could put Canadian Native women at risk for producing children with FASD. Bray and Anderson (1989) recommend that research be conducted to assess the consumption of alcohol among Canadian Native women in specific communities.
- Use larger samples and cross-sections of Native populations to enhance generalizability. Shostak and Brown (1995) also note the need to include Indians who reside on reservations and those who live in rural areas.
- The psychometrics of instruments need to be assessed. This information supports the identification of appropriate tools to be used and gives credibility to the findings (Shostak & Brown, 1995).
- What are the effects of early diagnosis and intervention, loving and knowledgeable family, and educational opportunities on children? The effects of these situations remain unexplored (Shostak & Brown, 1995).
- Increase accessibility to all forms of family planning. Kvigne et al. (2003) note that Northern Plains Indian women tend to choose sterilization as a means of birth control, in large measure, due to a lack of accessibility to other forms of family planning.
- Ensure educational materials are written at an appropriate reading level. Given the educational level of many Native women is lower than non-Native women, educational materials need to be appropriate or many women will not have access to basic information (Kvigne et al., 2003).
- Larger samples and comprehensive assessments are needed to determine the variables that may predispose women to continue drinking once pregnancy is known. O'Connor and Whaley (2003) suggest that variables might include: education about risks of drinking during pregnancy, role of drinking behaviour of significant others, personal characteristics of those women who continue to drink (e.g., depression), and the use of other teratogens such as nicotine.
- Develop campaigns and interventions to provide factual information to help at risk women reduce their drinking during pregnancy. Kaskutas (2000) describes women's responses to health-warning messages is less than anticipated. In fact, many women said the warnings made them feel negative about themselves, rather than conveying an important message about drinking behaviours.
- Treatment outcomes are needed. Streissguth (1994) indicates that the effectiveness of play therapy, insight-oriented therapy, or group therapy have not been identified through research. Nor is there any research on the efficacy of medications for treating persons with FAS/FAE.
- Examination of co-morbid substance use effects in relationship to the development of FASD/FAE.
- Evaluation of the effectiveness of various screening tools through which a universal approach can be had.
- Evaluation of home visitation models over a longitudinal period.
- Scientific collection of data needs to be the basis of evaluation rather than the use of client self-report.
- Dissemination of scientific evidence should reach practitioners working in the field so they can critique its usefulness, and if appropriate apply in the practice setting.

- Examine the relationship between the advantages and disadvantages of using professional advocates versus para-professional advocate.
- Health professionals need to understand and undertake Holistic Risk Analysis.

Recommendations for Policy

- Policy should encourage and support informal and formal *positive* social support.
- Establish a universal policy directive that supports zero alcohol tolerance during pregnancy and throughout the period of lactation, if supported by sound evidence.
- Invest resources in programs to eliminate the occurrence of FASD. Shostak and Brown (1995) suggest that professionals unite in supporting one another in the prevention of FAS. This recommendation is also supported by Kvigne et al. (1998).
- Include all community stakeholders when developing health promotion programs for American Indians (May & Moran, 1995). This recommendation is important given the desire of most, if not all, Aboriginal peoples to be self-governing. Although sharing information from successful programs is appropriate, it is recognized that programs need to be adapted for use in any community.
- Use existing policies that have been shown to be effective for purposes of developing policies for American Indians (May & Moran, 1995). This notion is supported by most researchers who readily acknowledge that success of policy and laws regarding use of alcohol in any commuity are dependent upon direct and committed community involvement.
- Establish a standard approach to diagnosis and measuring prevalence that impacts policy, for example, compulsory reporting of FASD cases to inform a centralized database system.
- Invest resources in programs to eliminate the occurrence of FASD/FAE such as women-specific alcohol programs, culturally sensitive programs, and education.
- Reconsider punitive approaches that are neither ethical, nor, effective.
- Focus on the fact that FASD is not a women's issue, it is one in which society at large must take active responsibility.
- Policy and resource allocation for developing competence in FASD for health care workers.
- Direct curricula in schools, health curricula to address FASD/FAE in a comprehensive manner commiserate with levels of understanding.
- Direct resources to fully implement effective and efficient Marketing Strategies and Other Preventative Models.

References

Abel, E. L. (1984). Is Fetal Alcohol Syndrome a new discovery? In E. L. Abel (Ed.), *Fetal Alcohol Syndrome and Fetal Alcohol Effects*. Author.

Abel, E. L. (1989). Paternal behavioral mutagenesis. *Neurotoxicology, 10*(3), 335-345.

Abel, E. L. (1995). An update on incidence of FAS: FAS is not an equal opportunity birth defect. *Neurotoxicology & Teratology, 17*(4), 437-443.

Abel, E. L. (1998). Fetal Alcohol Syndrome: The 'American Paradox.' *Alcohol and Alcoholism, 33*(3), 195-201.

Abel, E. L. (2004). Paternal contribution to Fetal Ethanol Syndrome. *Addiction Biology, 9*(2), 27-133.

Abel, E. L., & Hannigan, J. H. (1995). Maternal risk factors in Fetal Alcohol Syndrome: Provocative and permissive influences. *Neurotoxicology and Teratology, 17*(4), 445-462.

Abel, E. L., & Sokol, R. J. (1986). Maternal and fetal characteristics affecting alcohol's teratogenicity. *Neurobehavioral Toxicology and Teratology, 8*, 329-334.

Abascal, K., Herbalist, J. D., & Yarnell, E. (1993). The many faces of Silybum Marianum (Milk Thistle). *Alternative and Complementary Therapies, 9*(5), 251-255.

Ahluvalia, B., Wesley, B., Adeyiga, O., Smith, D. M., Da-Siulva, A., & Rajgura, S. (2000). Ethanol modulates cytokine secretion and synthesis in human fetus: An in vivo and in vitro study. *Ethanol, 21*, 207-213.

Alberta Alcohol and Drug Abuse Commission. (2004). *Windows of opportunity: A statistical profile of substance use among women in their childbearing years in Alberta.* Executive Summary. Edmonton, AB, Canada: Author.

Alberta Children's Services. (2004). *Youth awareness campaign: 2004 Youth Fetal Alcohol Spectrum Disorder Prevention Campaign.* Retrieved November 18, 2004 from http://www.child.gov.ab.ca/whatwedo/fas/page.cfm?pg=Youth%20Awareness%20Campaign

Alberta Health and Wellness, & Alberta Medical Association. (2002). *Alberta reproductive health: Pregnancies and births*. Edmonton, AB, Canada: Author.

Alderson, P., Green, S., & Higgins, J. P. T. (Eds.). (2003). *Cochrane reviewers' handbook 4.2.2.* Retrieved February 18, 2004 from http://www.cochrane.org/resources/handbook/hbook.htm

Allard-Hendren, R. (2000). Alcohol use and adolescent pregnancy. MCN, *25*(3), 159-162.

American College of Obstetricians and Gynecologists (1994). Substance abuse in pregnancy. *Int J Gynaecol Obstet., 47*(1),73-80.

American Medical Association. (2002). The environmental approach to prevention: Implementing alcohol policies. Retrieved July 18, 2004 from http://www.alcoholpolicysolutions.net.

Anderson, J. M., Wiggins, S., Rajwani, R., Holbrook, A., Blue, C., & Nig, M. (1995). Living with a chronic illness: Chinese-Canadian and Euro-Canadian women with diabetes – Exploring factors that influence management. *Social Science Medicine, 41*, 181-195.

Anglin, L., Kavanagh, L., & Giesbrecht, N. (2001). Alcohol-related policy measures in Ontario: Who supports what and to what degree? *Canadian Journal of Public Health, 92*(1), 24-2.

Antonucci, T. C. (1985). Personal characteristics, social support and social behaviour. In B. E. Shanas (Ed.). *Aging and social sciences.* New York: Van Nostrand Reinhold.

Appelbaum, M. G. (1995). Fetal Alcohol Syndrome: Diagnosis, management and prevention. *Nurse Practitioner, 20*(10), 24-36.

Arfsten, D. P., Silbergeld, E. K., & Loffredo, C. A. (2004). Fetal ADH2*3, maternal alcohol consumption, and fetal growth. *International Journal of Toxicology, 23*(1), 47-54.

Arling, G. (1987). Strain, social support, and distress in old age. *Journal of Gerontology, 42*(1), 107-113.

Arnold, M. S., Butler, P. M., Anderson, R. M., Funnell, M. M., & Feste, C. (1995). Guidelines for facilitating a patient empowerment program. *The Diabetes Educator, 21*(4), 308-312.

Ashery, R. S., Wild, J., Zhao, Z., Rosenshine, N., & Young, P. (1997). The Wheel Project Women helping to empower and enhance lives. *Journal of Substance Abuse Treatment, 14*, 113-121.

Astley, S. J., & Clarren, S. K. (1995). A Fetal Alcohol Syndrome screening tool. *Alcoholism: Clinical and Experimental Research, 19*(6), 1565-1571.

Astley, S. J., & Clarren, S. K. (2001). Measuring the facial phenotype of individuals with prenatal alcohol exposure: Correlations with brain dysfunction. *Alcohol and Alcoholism, 36*(2), 147-159.

Astley, S. J., Bailey, D., Talbot, C., & Clarren, S. K. (2000). Fetal Alcohol Syndrome (FAS) primary prevention through FAS diagnosis: II. A comprehensive profile of 80 birth mothers of children with FAS. *Alcohol and Alcoholism, 35*(5), 509-519.

Autti-Ramo, I., Autti, T., Korkman, M., Kettunen, S., Salonen, O., & Valanne, L. (2002). MRI findings in children with school problems who had been exposed prenatally to ethanol. *Developmental Medicine in Child Neurology, 44*(2), 98-106.

Avner, M., & Koren, G. (2004). Breaking the Cycle – A unique model for FASD research. *Journal of Family and Society Institute, 2*(3), 2.

Baker, E. A., & Brownson, C. A. (1999). Defining characteristics of community-based health promotion programs. In R. C. Brownson, E. A. Baker, & L. N. Novick (Eds.), *Community-based prevention: Programs that work*. Gaithersburg, MD: Aspen.

Bandura, A. (1986). *Social foundations of thought and action: A social cognitive theory*. Englewood Cliffs, NJ: Prentice-Hall.

Barbor, T. F. (2002). Linking science to policy. The role of international collaborative research. *Alcohol Research and Health, 26*(1), 66-74

Basford, L., Nyatanga, L., & Dann, K. (2001). *Empowerment within the field of mental health care.* ISBN: 090143719 0. The University of Derbyshire.

Bearer, C. F., Jacobson, J. L., Jacobson, S. W., Barr, D., Croxford, J., Molteno, C. D., Viljoen, D. L., Marais, A., Chiodo, L. M., & Cwik, A. S. (2003). Validation of a new biomarker of fetal exposure to ethanol. *The Journal of Pediatrics, 143*, 463-469.

Becker, K. L., & Walton-Moss, B. (2001). Detecting and addressing alcohol abuse in women. *Nurse Practitioner, 26*(10), 19-23.

Belizan, J. M., Baros, F., Langer, A., Farnot, U., Victoria, C., & Villar, J. (1995). Obstetrics. Impact of health education during pregnancy on behaviour and utilization of health resources. *American Journal of Obstetricians, 173*(3), 894-899.

Best Start. (2003). *Keys to a successful alcohol and pregnancy communication campaign. A manual by Best Start: Ontario's Maternal, Newborn and Early Child Development Resource Centre.* Toronto, ON: Author.

Betzner, A., Luxenberg, M., Evered, S., & Rainey, J. (2001). Changing harmful habits. Assessing Minnesota's nurses' screening practices and training needs regarding women's alcohol and tobacco use. Minnesota Department of Health, Family Health Division. Retrieved 8/4/04. http://www.health.state.mn.us/fas/reports/habits/changing_harmfulhabits.pdf

Bhatara, V., Lovrein, F., Kirkeby, J., Swayze, V., Unruh, E., & Johnson, V. (2002). Brain function in Fetal Alcohol Syndrome assessed by single photon emission computer tomography. *South Dakota Journal of Medicine, 55*(2), 59-62.

Bingol, N., Schuster, C., Fuchs, M., Iosub, S., Turner, G., Stone, R. K., & Gromisch, D. S. (1987). The influences of socioeconomic factors on the occurrence of Fetal Alcohol Syndrome. In *Children of alcoholics.* The Haworth Press.

Blank, R. H. (1996). Mandating outpatient treatment for pregnant substance abusers: Attractive but unfeasible. *Politics and the Life Sciences, 15*(1), 49-50.

Blaxter, M. (1990). *Health and lifestyles.* London: Routledge.

Bookstein, F. L., Sampson, P. D., Streissguth, A. P., & Connor, P. D. (2001). Geometric morphometrics of corpus callosum and subcortical structures in the fetal-ethanol-affected brain. *Teratology, 64*(4), 4-32.

Bookstein, F. L., Streissguth, A. P., Sampson, P. D., Connor, P. D., & Barr, H. M. (2002). Corpus callosum shape and neuropsychological deficits in adult males with heavy fetal ethanol exposure. *NeuroImage, 15,* 233-251.

Bosron, W. F., Magnes, L. J., & Li, T-K. (1983) Human liver alcohol dehydrogenase: ADH Indianapolis results from genetic polymorphisms at the ADH2 gene locus. *Biochemical Genetics, 21,* 735-744.

Bourdieu, P. (1984). *Distinction.* Cambridge, MA: Harvard University Press.

Bowden, R., & Rust, D. (2000). A review of Fetal Alcohol Syndrome for health educators. *Journal of Health Education, 31*(4), 231-237.

Bray, D., & Anderson, P. D. (1989). Appraisal of the epidemiology of Fetal Alcohol Syndrome among Canadian Native peoples. *Canadian Journal of Public Health, 80,* 42-45.

Bradley, D. M., Beaman, F. D., Moore, D. B., Kidd, K., & Heaton, M. B. (1999). Neurotrophic factors BDNF and GDNF protect embryonic chick spinal cord motoneurons from ethanol neurotoxicity in vivo. *Developmental Brain Research, 112,* 99-106.

Brenneman, D. E., Spong, C. Y., Hauser, J. M., Abebe, D., Pinhasov, A., Golian, T., & Gozes, I. (2004). Protective peptides that are orally active and mechanistically nonchiral. *The Journal of Pharmacology and Experimental Therapeutics, 309,* 1190-1197.

British Columbia Ministry for Children and Families. (1998*). Community Action Guide: Working together for the prevention of Fetal Alcohol Syndrome.* Victoria, BC: Adult Addiction Services.

British Columbia Ministry for Children and Families (2003). *Fetal Alcohol Spectrum Disorder: A strategic plan for British Columbia.* Victoria, BC: Author.

Brooks, P. J. (1997). DNA damage, DNA repair, and ethanol toxicity A review. *Alcoholism: Clinical and Experimental Research, 21*(6), 1073-1082.

Brower, K. J. (2003). Insomnia, alcoholism and relapse. *Sleep Medicine Review, 7*(6), 523-539.

Brown, S. A., D'Amico, E. J., McCarthy, D. M., & Tapert, S. F. (2001). Four-year outcomes from adolescent alcohol and drug treatment. *Journal of Studies on Alcohol, 62*(3), 381-388.

Burd, L., Cox, C., Fjelstad, K., & McCulloch, K. (2000). Screening for Fetal Alcohol Syndrome: Is it feasible and necessary? *Addiction Biology, 5,* 127-139.

Burd, L., Cotsonas-Hassler, T. M., Martsolf, J. T., & Kerbeshian, J. (2003). Recognition and management of fetal alcohol syndrome. *Neurotoxciology and Teratolgy, 25,* 681-688.

Burd, L., Martsolf, J. T., Klug, M. G. & Kerbeshian, J. (2003). Diagnosis of FAS: a comparison of the Fetal Alcohol Syndrome Diagnostic Checklist and the Institute of Medicine Criteria for Fetal Alcohol Syndrome. *Neurotoxicology and Teratology, 25*, 719-724.

Burd, L., Martsolf, J. T., & Klug, M. G. (1996). Children with Fetal Alcohol Syndrome in North Dakota: A case control study utilizing birth certificate data. *Addiction Biology, 1*, 181-189.

Burdan, F. (2002). Folic acid isn't sufficient for prevention of congenital defects. Report from the 42nd Teratologic Society Conference. *Ginekol Pol, 73*(12), 1247-1249.

Burdge, G. C., Wright, S. M., Warner, J. O., & Postle, A. D. (1997). Fetal brain and liver phospholipid fatty acid composition in a guinea pig model of Fetal Ethanol Syndrome: Effect of maternal supplementation with tuna oil. *Nutritional Biochemistry, 8,* 438-444.

Burgoyne, W. (1998). Alcohol and pregnancy signs for restaurants and bars. *Best Start Resources.* Retrieved July 19, 2004 from the World Wide Web: www.beststrat.org/ resources/alc_reduction/ alc_preg_signs.html

Burton, R. (1621). *Anatomy of melancholy.* London: Colophon.

Busby, A., LaGrange, L., Edwards, J., & King, J. (2002). The use of a silymarin/phospholipid compound as a fetoprotectant from ethanol-induced behavioral deficits. *Journal of Herbal Pharmacotherapy, 2*(1), 39-46.

Byrne, C. (1998). Facilitating empowerment groups: Dismantling professional boundaries. *Issues in Mental Health Nursing, 9,* 55-71.

Caldeira, J. C., Wu, Y., Mameli, M., Purdy, R. H., Li, P. K., Akwa, Y., Savage, D. D., Engen, J. R., & Valenzuela, C. F. (2004). Fetal alcohol exposure alters neurosteroid levels in the developing rat brain. *Journal of Neurochemistry, 90*(6), 1530-1539.

Cano, M. J., Ayala, A., Murillo, M. L., & Carreras, O. (2001). Protective effect of folic acid against oxidative stress produced in 21-day postpartum rats by maternal-ethanol chronic consumption during pregnancy and lactation period. *Free Radical Research, 34,* 1-8.

Caprara, D., & Koren, G. (2004). To label or not to label: The pros and cons of alcohol warning labels in pregnancy. *Journal of FAS International, 2,* 1-3.

Cavieres M. F., & Smith, S. M. (2000). Genetic and developmental modulation of cardiac deficits in prenatal alcohol exposure. *Alcoholism: Clinical & Experimental Research. 24*(1), 102-109.

Centers for Disease Control and Prevention. (1997). Atlanta, Georgia surveillance from multiple sources 1981-1989 0.10 NA 285538 Unknown

Centers for Disease Ccontrol and Prevention. (2002). Alcohol use among women of childbearing age - United States, 1991-1999. *MMWR, 51*(13), 273-276.

Chang, G. (2001). Alcohol-screening instruments for pregnant women. *Alcohol Research and Health, 25*(3), 204-209.

Chang, G., Goetz, M. A., Wilkins-Haug, L., & Berman, S. (2000). A brief intervention for prenatal alcohol use: An in-depth look. *Journal of Substance Abuse Treatment, 18,* 365-369.

Chang, G., Wilkins, L., Berman, S., & Goetz, M.A. (1999). The TWEAK: Application in a prenatal setting. *Journal for the Study of Alcohol, 60,* 306-309.

Chang, G., Wilkins-Haug, L., Berman, S., & Goetz, M.A. (1999). Brief intervention for alcohol use in pregnancy: A randomized trial. *Addiction, 94*(10), 1499-1508.

Chappell, L. C., Seed, P. T., Briley, A. L., Kelly, F. J., Lee, R., Hunt, B. J., Parmar, K., Bewley, S. J., Shennan, A. H., Steer, P. J., & Poston, L. (1999). Effect of antioxidants on the occurrence of pre-eclampsia in women at increased risk: A randomized trial. *Lance, 354,* 810-816.

Chen, L., & Nyomba, B. L. G. (2004). Whole body insulin resistance in rat offspring of mothers consuming alcohol during pregnancy or lactation: comparing prenatal and postnatal exposure. *Journal of Applied Physiology, 96,* 167-172.

Chernoff, G. F. (1980). The Fetal Alcohol Syndrome in mice: Maternal variables. *Teratology, 22*(1), 71-75.

Choong, K., & Shen, R. (2004). Prenatal ethanol exposure elaters the postnatal development of the spontaneous electrical activity of dopamine neurons in the ventral segmental area. *Neuroscience, 126*(4), 1083-1091.

Cicero, T. J. (1994). Effects of paternal exposure to alcohol on offspring development. *Alcohol Health & Research World, 18*(1).

Clarren, S. K., & Astley, S.J. (1998). Identification of children with fetal alcohol syndrome and opportunity for referral of their mothers for primary prevention – Washington, 1993-1997. *Morbidity and Mortality Weekly Report, 47*(40). **01492195, 10/16/98,**

Clarren, S., & Smith, D. W., (1978). The fetal alcohol syndrome. *New England Journal of Medicine, 298,* 1063-1067.

Cobb, S. (1976). Social support as a moderator of life stress. *Psychosomatic Medicine, 38,* 300-314.

Cockerham, W. (1995). *Medical Sociology*. Englewood Cliffs, New Jersey: Prentice Hall.

Cockerham, W., Rutten, A., & Abel, T. (1997). Contextualizing contemporary health lifestyles: Moving beyond Weber. *Sociological Quarterly, 38,* 342.

Cohen, S., & Syme, S. (1985). Issues in the study and application of social support. *Social support and health.* Orlando, FL: Academic Press.

Cohen, S., & Wills, T. (1985). Stress social support, and the buffering hypothesis. *Psychological Bulletin,* **XXX**, 310-357.

Cohen-Kerem, R., & Koren, G. (2003). Antioxidants and fetal protection against ethanol teratogenicity. *Neurotoxicology and Teratology, 25*(1), 1-9.

Cole, C. (2001). Fetal alcohol exposure and attention: Moving beyond ADHD. *Alcohol Research and Health, 25*(3), 199-203.

Coles, C. D., Kable, J. A., Drews-Botch, C., & Falek, A. (2000). Early identification of risk for effects of prenatal alcohol exposure. *Journal of Studies on Alcohol, 61*(4), 607-616.

Colmorgen, G. H. C. (1986). Prevention of Fetal Alcohol Syndrome. *Delaware Medical Journal, 58*(8), 544-545.

Connors, G. J., & Walitzer, K. S. (2001). Reduced alcohol consumption among heavily drinking women: Evaluating the contributions of life-skills training and booster sessions. *Journal of Consulting and Clinical Psychology, 69,* 447-456.

Corse, S. J., McHugh, M. K., & Gordon, S. M. (1995). Enhancing provider effectiveness in treating pregnant women with addictions. *Journal of Substance Abuse Treatment, 12*(1), 3-12.

Costa, L. G., & Guizzetti, M. (1999). Muscarinic cholinergic receptor signal transduction as a potential target for the developmental neurotoxicity of ethanol. *Biochemical Pharmacology, 57*(7), 721-726.

Counsell, A. M., Smale, P. N., & Geddis, D. C. (1994). Alcohol consumption by New Zealand women during pregnancy. *The New Zealand Medical Journal, 107*(982), 278-281.

Coyne, J. C., & DeLongis, A. (1986). Going beyond social support: The role of social relationships in adaptation. *Journal of Consulting and Clinical Psychology, 54,* 454-460.

Craig, H. M., & Edwards, J. E. (1983). Adaptation in chronic illness: An eclectic model for nurses. *Journal for Advanced Nursing, 8*(5), 397-404.

Cutrona, C., Russell, D., & Rose, J. (1986). Social support and adaptation to stress by the elderly. *Psychology and Aging, 1,* 47-54.

Daniels, C., & Golden, C. (2000). The politics of paternity: Foetal risks and reproductive harm. In M. Freeman (Ed), *Law and Medicine.* New York: Oxford.

Danis, R. P. (1981). Pregnancy and alcohol. *Current Problems in Obstetrics and Gynecology, 4*(6), 2-48.

Dahrendorf, R. (1979). *Life chances.* Chicago, IL: Chicago University Press.

De Ridder, D., Depla, M., Severens, P., & Malsch, M. (1997). Beliefs on coping with illness: A consumer's perspective. *Social Science and Medicine, 44*(5), 553-559.

Deshpande S., & Basil, M. (in press). Lessons from research on social marketing for mobilizing adults for positive youth development. In E. Gil Clary, & Jean E. Rhodes, (Eds.), *Mobilizing adults for positive youth development: Lessons from the behavioral sciences on promoting socially valued activities.* Minnesota, MN: Search Institute.

DeVille, K. A., & Kopelman, L. M. (1998). Moral and social issues regarding pregnant women who use and abuse drugs. *Obstetrics and Gynecology Clinics of North America, 25*(1), 237-254.

DeVille, K. A., & Kopelman, L. M. (1999). Fetal protection in Wisconsin's revised child abuse law: Right goal, wrong remedy. *Journal of Law, Medicine and Ethics,* 27, 332-342.

Diekman, S. T., Floyd, L., Decoufle, P., Schulkin, J., Ebrahim, S. H., & Sokol, R. J. (2000). A survey of obstetrician-gynecologists on their patient's alcohol use during pregnancy. *Obstetrics and Gynecology, 95*(5), 756-763.

Donne, J. (1624). *From whom the bell tolls. Meditation XVII.*

Donnelley, F. M., Mowery, J. L., & McCarver, D. G. (1998). Knowledge and misconceptions among inner-city African-American mothers regarding alcohol and drug use. *American Journal of Drug and Alcohol Abuse, 24*(4), 675-683.

Donovan, C. L. (1991). Factors predisposing, enabling and reinforcing routine screening of patients for preventing Fetal Alcohol Syndrome: A survey of New Jersey physicians. *Journal of Drug Education, 21*(1), 34-42.

Downing C., & Gilliam, D. (1999). Cytoplasmic factors do not contribute to a maternal effect on ethanol teratogenesis. *Behavior Genetics, 29*(1), 31-39.

Duimstra, C., Johnson, D., Kutsch, C., Wang, B., Zentner, M., Kellerman, S., & Welty, T. (1993). A Fetal Alcohol Syndrome surveillance pilot project in American Indian communities in the Northern Plains. *Public Health Reports, 108*(2), 225-229.

Ebrahim, S. H., Diekman, S. T., Floyd, R. L., & Decoufle, P. (1999). Comparison of binge drinking among pregnant and nonpregnant women, United States, 1991-1995. *American Journal of Obstetrics and Gynecology, 10*(1 Pt 1), 1-7.

Eckenrode, J., Ganzel, B., Henderson, C. R. Jr., Smith, E., Olds, D. L., Powers, J., Cole, R., Kitzmand, H., & Sidora, K. (2000). Preventing child abuse and neglect with a program of nurse home visitation: The limiting effects of domestic violence. JAMA, 284(11), 1385-1391.

Edwards, J., La Grange, L., Wang, M., & Reyes, E. (2000). Fetoprotectivity of the flavanolignan compound siliphos against ethanol-induced toxicity. *Phytotherapy Research, 14,* 517-521.

Egeland, G. M., Perham-Hester, K. A., Gessner, B. D., Ingle, D., Berner, J. E., & Middaugh, J. P. (1998). Fetal Alcohol Syndrome in Alaska, 1977 through 1992: An administrative prevalence derived from multiple data sources. *American Journal of Public Health,* 88(5), 781-786.

Elderton, E. M., & Pearson, K. A. (1910). *First study of the influence of parental alcoholism on the physique and ability of the offspring,* (2nd. Ed.). London: Dulau and Company.

Environics Research Group. (2000). *Awareness of the effects of alcohol use during pregnancy and Fetal Alcohol Syndrome: Results of a national survey.* Ottawa, ON: Health Canada.

Farber, N. B., & Olney, J. W. (2003). Drugs of abuse that cause developing neurons to commit suicide. *Developmental Brain Research, 147,* 37-45.

Felton, B. J. (1990). Coping and social support in older people's experiences of chronic illness. In *Stress and coping in later-life families*. New York: Hemisphere.

Feste, C., & Anderson, R. M. (1995). Empowerment: From philosophy to practice. *Patient Education and Counselling, 26,* 139-144.

Floyd, L. R., Decoufle, P., & Hungerford, D. W. (1999). Alcohol use prior to pregnancy recognition. *American Journal of Preventive Medicine, 17*(2), 101-107.

Folkman, S., & Lazarus, R. S. (1985). If it changes it must be a process: A study of emotion and coping during three stages of a college examination. *Journal of Personality and Social Psychology, 48,* 150-170.

Forrest, F., & Florey, C. du V. (1991). The relation between maternal alcohol consumption and child development: The epidemiological evidence. *Journal of Public Health Medicine, 13*(4), 147-255.

Fox, D. J., & Druschel, C. M. (2003). Estimating prevalence of Fetal Alcohol Syndrome (FAS): Effectiveness of a passive birth defects registry system. *Birth Defects Research Part A Clinical Mol. Teratology,* 67(9), 604-608.

Friere, P. (1973). *Education for critical consciousness*. New York: Seabury Press.

Frohna, J. G., Lantz, P. M., & Pollack, H. (1999). Maternal substance abuse and infant health: policy options across the life course. *The Milbank Quarterly, 77*(4), 531-571.

Garcia-Rodriguez, S., Arguelles, S., Llopis, R., Murillo, M. L., Machado, A., Carreras, O., & Ayala, A. (2003). Effect of prenatal exposure to ethanol on hepatic elongation factor-2 and proteome in 21 day old rats: Protective effect of folic acid. *Free Radical Biology & Medicine, 35*(4), 428-437.

Giddens, A. (1991*) Modernity and self identity: Self and society in late modern age.* Stanford University Press.

Gilliam, D. M., & Irtenkauf, K. T. (1990). Maternal genetic effects on ethanol teratogenesis and dominance of relative embryonic resistance to malformations. *Alcoholism: Clinical & Experimental Research, 14*(4), 539-545.

Gilliam, D. M., Kotch L. E., Dudek B. C., & Riley, E. P. (1989). Ethanol teratogenesis in selectivity bred long-sleep and short-sleep mice: A comparison to inbred C57BL/6J mice. *Alcoholism: Clinical & Experimental Research, 13*(5), 667-672.

Gilliam, D. M., Mantle, M. A., Barkhausen, D. A., & Tweden, D. R.. (1997). Effects of acute prenatal ethanol administration in a reciprocal cross of C57BL/6J and short-sleep mice: Maternal effects and nonmaternal factors. *Alcoholism: Clinical & Experimental Research, 21*(1), 28-34.

Gilliam, D. M., Stilman, A., Dudek, B. C., & Riley, E. P. (1987). Fetal Alcohol Effects in long- and short-sleep mice: Activity, passive avoidance, and in utero ethanol levels. *Neurotoxicology & Teratology, 9*(5), 349-357.

Gladstone, J., Levy, M., Nulman, I., & Koren, G. (1997). Characteristics of pregnant women who engage in binge alcohol consumption. *Canadian Medical Association, 156*(6), 789-794.

Golka, K. (2004). Carbohydrate-deficient transferring (CDT): A biomarker for long-term alcohol consumption. *Journal of Toxicology & Environmental Health, 7*(4), 319-337.

Gordis, E. (1992). From the National Institutes of Health. *Journal of American Medical Association, 268,* 3183.

Grant, T., Ernst, C. C., McAuliff, S., & Streissguth, A. P. (1997). The difference game: Facilitating change in high-risk clients. *Families in Society, 78*(4), 429-432.

Grant, T., Streissguth, A. P., & Ernst, C. C. (2002). Benefits and challenges of paraprofessional advocacy with mothers who abuse alcohol and drugs and their children. *Zero to Three, 23*(2), 14-20.

Grant, T., Ernst, C. C., & Streissguth, A. P. (1999). Intervention with high-risk alcohol and drug-abusing mothers: 1. Administrative strategies of the Seattle model of paraprofessional advocacy. *Journal of Community Psychology, 27*(1), 1-18.

Graves, K. L. (1993). An evaluation of the alcohol warning label: A comparison of the United States and Ontario, Canada in 1990 and 1991. *Journal of Public Policy and Marketing, 12*(1), 19-29.

Greaves, L., Poole, N., & Cormier, R. (2002). Fetal Alcohol Syndrome and women's health: Setting a women-centered research agenda. Vancouver, BC: The British Columbia Centre of Excellence in Women's Health.

Greenfield, T. K., Graves, K. L., & Kaskutas, L. A. (1999). Long-term effects of alcohol warning labels: Findings from a comparison of the United States and Ontario, Canada. *Psychology & Marketing, 16*(3), 261-282.

Guerri, C. (2002). Mechanisms involved in CNS dysfunctions induced by prenatal ethanol exposure. *Neurotoxicity Research, 4*(4), 327-335.

Guiet-Bara, A., Bara, M., Durlach, J., & Pechery, C. (1988). Ethanol effect on the ionic transfer through isolated human amnion. I. Preventive and antagonistic actions of some nutrients and of their synthetic congeners. *Ethanol, 5*, 63-71.

Habbick, B. F., Nanson, J. L., Snyder, R. E., Casey, R. E., & Schulman, A. L. (1996). Foetal Alcohol Syndrome in Saskatchewan: unchanged incidence in a 20-year period. *Canadian Journal of Public Health, 87*(3), 204-207.

Haemmerlie, F. M., Merz, C. J., & Nelson, S. (1992). College vs. junior high school student's knowledge of alcohol as a teratogen. *Psychological Reports, 71,* 809-810.

Hall, B. D., & Orenstein, W. A. (1975). Noonan's phenotype in an offspring of an alcoholic mother. *Lancet, 1,* 680.

Hamilton, C., & Snyder, L. (1995). Preventing Fetal Alcohol Syndrome. *The HIS Primary Care Provider, 20*(4), 57-60.

Handmaker, N. S., Miller, W. R, & Manicke, M. (1999). Findings of a pilot study of motivational interviewing with pregnant drinkers. *Journal of Studies on Alcohol, 60,* 285-287.

Hankin, J. R., Firestone, I. J., Sloan, J. J., Ager, J. W., Sokol, R. J., & Martier, S. J. (1996). Heeding the alcoholic beverage warning label during pregnancy: Multiparae versus Nulliparae. *Journal of Studies on Alcoho, (March),* 171-177.

Hankin, J. R., Sloan, J. J., Firestone, I. J., Ager, J. W., Sokol, R. J., Martier, S. S., & Townsend, J. (1993). The alcohol beverage warning label: When did knowledge increase? *Alcoholism: Clinical and Experimental Research, 17*(2), 428-430.

Hankin, J. R., Sloan, J. J., Firestone, I. J., Ager, J. W., Sokol, R. J., Martier, S. S., & Townsend, J. (1996). Has awareness of the alcohol warning label reached its upper limit? *Alcoholism: Clinical and Experimental Research, 20*(3), 440-444.

Hanna, E. Z., Faden, V. B., & Dufour, M. C. (1994). The motivational correlates of drinking: Smoking, and illicit drug use during pregnancy. *Journal of Substance Abuse, 6*, 155-167.

Hannigan, J. H. (1995). Effects of prenatal exposure to ethanol plus caffeine in rats: Pregnancy outcome and early offspring development. *Alcoholism: Clinical and Experimental Research, 1* (1), 238-246.

Hannigan, J. H. (1996). What research with animals is telling us about ethanol-related neurodevelopmental disorder. *Pharmacology, Biochemistry & Behavior, 55*, 489-499.

Hannigan, J. H., & Berman, R. F. (2000). Amerlioration of fetal ethanol-related neurodevelopmental disorders in rats: Exploring pharmacological and environmental treatments. *Neurotoxicology & Teratology, 22*, 103-111.

Hayes, M. J., Brown, E., Hofmaster, P. A., Davare, A. A. Parker, K. G., & Raczek, J. A. (2002). Prenatal alcohol intake in a rural, Caucasion clinic. *Family Medicine, 34*(2), 120-125.

Haynes, G., Dunnagan, T., & Christopher, S. (2003). Determinants of alcohol use in pregnant women at risk for alcohol consumption. *Neurotoxicology and Teratology, 25*, 659-666.

Heaton, M. B., Kim, D. S., & Paiva, M. (2000). Neurotrophic factor protection against ethanol toxicity in rat cerebellar granule cell cultures requires phosphatidylinositol 3-kinase activation. *Neuroscience Letters, 291*, 121-125.

Heaton, M. B., Mitchell, J. J., & Paiva, M. (2000a). Overexpression of NGF amerliorates ethanol neurotoxicity in the developing cerebellum. *Inc. Journal of Neurobioogy, 45*, 95-104.

Heaton, M. B., Mitchell, J. J., & Paiva, M. (2000b). Amerlioration of ethanol-induced neurotoxicity in the neonatal rat central nervous system by antioxidant therapy. *Ethanolism: Clinical and Experimental Research, 24*(4), 512-518.

Heaton, M. B., Paiva, M., Madorsky, I., Mayer, J., & Moore, D. B. (2002). Effects of ethanol on neurotrophic factors, apoptosis-related proteins, endogenous antioxidants, and reactive oxygen species in neonatal striatum: Relationship to periods of vulnerability. *Developmental Brain Research, 140*(2), 237-252.

Heaton, M. B., Paiva, M., Swanson, D. J., Walker, D. W. (1994). Responsiveness of cultured septal and hippocampal neurons to ethanol and neurotrophic substances. *Journal of Neuroscience Research, 39*, 305-318.

Hendersen, G. I., Chen, J. J., & Schenker, S. (1999). Ethanol, oxidative stress, reactive aldehydes, and the fetus. *Frontiers in Bioscience, 4*, D541-D550.

Holahan, C. J., Moos, R. H., Holahan, C. K., & Brennan, P. L. (1997). Social context, coping strategies, and depressive symptoms: An expanded model with cardiac patients. *Journal of Personality and Social Psychology, 12*(11), 1100-1106.

Horrigan, T. J., Schroeder, A. V., & Schaffer, R. M. (1999). The triad of substance abuse, violence, and depression are interrelated in pregnancy. *Journal of Substance Abuse Treatment, 18*, 55-58.

House, J. S., & Kahn, R. L. (1985). Measures and concepts of social support. In S. Cohen & S. L. Syme (Eds.), *Social support and health*. San Francisco: Academic Press.

Hungund, B. L., & Mahadik, S. P. (1993). Role of gangliosides in behavioural and biochemical actions of ethanol: Cell membrane structure and function. *Alcoholism: Clinical and Experimental Research, 17*(2), 329-339

Hutchison, C. (1999). Social support: Factors to consider when designing studies that measure social support. *Journal of Advanced nursing, 29*(6), 1520-1526.

Ingle, D., Owen, P., Jones, L., Perry, S., & Cassidy, S. (1994). Alcohol consumption and Fetal Alcohol Syndrome – Alaska 1991 and 1993. *Journal of American Medical Association, 271,* 422-423.

Jacobson, J. L., & Jacobson, S. W. (1999). Drinking moderately during pregnancy. *Alcohol Health and Research World, 23*(11), 23-35.

Jacobson, S. W., Chiodo, L. M., Sokol, R. J., & Jacobson, J. L. (2002). Validity of maternal report of prenatal alcohol, cocaine, and smoking in relation to neurobehavioral outcome. *Pediatrics, 109*(5), 815-825.

Jellineck, E. M., & Jolliffe, N. (1940). Effects of alcohol on the individual. *Q. J. Stud. Alcohol, 1,* 110.

Jenkins R., & Bursih, T. (1995). Health clcus of control: Chemotherapy related distress and response to behavioural intervention in cancer patients. *Psychology and Health, 10,* 463-475.

Jessup, M., & Roth, R. (1988). Clinical and legal perspectives on prenatal drug and alcohol use: Guidelines for individual community response. *Medicine and Law, 7,* 377-389.

Jewell, N. (2004). Statistics for epidemiology. Chapman & Hall.

Johnston, L. D., O'Malley, P. M., & Bachman, J. G. (1988). *Illicit drug use, smoking, and drinking by American's high school students, college students, and young adults*. Washington, DC: US Government Printing Office.

Johnson, J. E. (1998). Stress, social support, and health in frontier elders. *Journal of Gerontological Nursing, 24*(5), 29-35.

Jones, K. L., & Smith, D. W. (1973) Recognition of the fetal alcohol syndrome in early infancy. *Lancet, 2,* 999-1001.

Jones, K. L., & Smith, D. W. (1976). The fetal alcohol syndrome. *Teratology, 12,* 1.

Jones, K. L., Smith, D. W., Ulleland, C. N., & Steissguth, A. P. (1973) Pattern of malformation in offspring of chronic alcoholic mothers. *Lancet, 1,* 1267-1271.

Jung-Ae, K., & Druse, M. J. (1996). Protective effects of maternal buspirone treatment on serotonin reuptake sites in ethanol-exposed offspring. *Developmenal Brain Research, 92,* 190-198.

Kaskutas, L. A. (1995). Interpretations of risk: Scientific information in the development of the alcohol warning label policy. *The International Journal of the Addictions, 30,* 1519-1548.

Kaskutas, L. A. (2000). Understanding drinking during pregnancy among urban American Indians and African Americans: Health messages, risk beliefs, and how to measure consumption. *Alcoholism: Clinical and Experimental Research, 24*(8), 241-250.

Kaskutas, L. A., & Graves, K. (1994). Relationship between cumulative exposure to health messages and awareness and behavior-related drinking during pregnancy. *American Journal of Health Promotion, 9,* 115-124.

Kaskutas, L. A., & Greenfield, T. K. (1992). First effects of warning labels on alcoholic beverage containers. *Drug and Alcohol Dependence, 31,* 1-14.

Kaskutas, L. A., Greenfield, T. L., Mija, E., & Cote, J. (1998). Reach and effects of health messages on drinking during pregnancy. *Journal of Health Education, 29,* 11-17.

Kelly, S. J., Day, N., & Streissguth, A. P. (2000). Effects of prenatal ethanol exposure on social behavior in humans and other species. *Neurotoxicology and Teratology, 22,* 143-149.

Keppen, L. D., Pysher, T., & Rennert, O. M. (1985). Zinc deficiency acts as a co-teratogen with ethanol in Fetal Ethanol Syndrome. *Pediatric Research, 19*(9), 944-947.

Kesmodel, U., & Kesmodel, P. S. (2002). Drinking during pregnancy: Attitudes and knowledge among pregnant Danish women, 1998. *Alcoholism: Clinical and Experimental Research, 26*(10), 1553-1560.

Kesmodel, U., Kesmodel, P. S., Larsen, A. & Secher, N. J. (2003). Use of alcohol and illicit drugs among pregnant Danish women, 1998. *Scandinavian Journal of Public Health, 31,* 5-11.

Kinzie, M. B., Schorling, J. B., & Siegal, M. (1993). Prenatal alcohol education for low-income women with interactive multimedia. *Patient Education and Counseling, 2,* 51-60.

Kirby, M., Bruce, I., Radic, A., Coakley, D., & Lawlor, B.A. (1997). Mental disorders among the community-dwelling elderly in Dublin. *British Journal of Psychiatry, 171,* 369-372.

Kloehn, D., Miner, K. J., Bishop, D., & Daly, K. (1997). Alcohol use in Minnesota Extent and cost. *Public Health Report, 80,* 26-29.

Kolbe, L. J. (1993). An epidemiological surveillance system to monitor the prevalence of youth behaviors that most effect health, chronic disease, and health promotion (I-V). *1990-1991. Youth risk behavior surveillance system.* Washington, DC: US Department of HHS, PHS, CDC, National Center for Chronic Disease and Health Promotion.

Koniak-Griffin, D., Anderson, N. L. R., Brecht, M. L., Verzemnieks, I., Lesser, J., & Kim, S. (2002). Public health nursing care of adolescent mothers: Impact on infant health and selected maternal outcomes at 1 year postbirth. *Journal of Adolescent Health, 30,* 44-54.

Kopera-Frye, K., Tswelnaldin, P., Streissguth, A. P., & LaDue, R. A. (1994). Preventing FAS by empowering Native American chemical dependency counselor. *IHS Primary Care Provider, 19*(4), 66-69.

Koren, G., Koren, T., & Gladstone, J. (1996). Mild maternal drinking and pregnancy outcome: Perceived versus true risks. *Clinica Chimica Acta, 246,* 155-162.

Koren, G., Roifman, I., & Nulman, I. (2004). Hypothetical framework: FASD and criminality causation or association? The limits of evidence-based knowledge. *JFAS Int, 2*, e6.

Kotler, P., Roberto, N., & Lee, N. (2002). *Social marketing: Improving the quality of life* (2nd ed.). Thousand Oaks, CA: Sage.

Kowalsky, L. O., & Verhoef, M. J. (1999). Northern community members' perceptions of AS/FAE: A qualitative study. *The Canadian Journal of Native Studies, XIX*(1), 49-168.

Kvigne, V. L., Bad Heart Bull, L., Welty, T. K., Leonardson, G. R., & Lacina, L. (1998). Relationship of prenatal alcohol use with maternal and prenatal factors in American Indian women. *Social Biology, 45*(3/4), 214-222.

Kvigne, V. L., Leonardson, G. R., Borzelleca, J., Brock, E., Neff-Smith, M., & Welty, T. K. (2003). Characteristics of mothers who have children with Fetal Alcohol Syndrome or

some characteristics of Fetal Alcohol Syndrome. *Journal of the American Board of Family Practice, 16*(4), 296-303.

Laireiter, A., & Baumann, U. (1992). Network structures and support functions: Theoretical and empirical analyses. In H. O. F. Veiel, U. Baumann, et al (Eds.), *The meaning and measurement of social support* (pp. 33-35). (The series in clinical and community psychology). New York: Hemisphere.

Lanza, A. F., & Revenson, T. A. (1993). Social support interventions for rheumatoid-arthritis patients – The cart before the horse. *Health Education Quarterly, 20,* 97-117.

Lauzon, R., Gregoire, T., Gliksman, L., McKay, I., & Douglas, R. R. (1998). Mattagami First Nation's policy to reduce alcohol-related harm. *The Canadian Journal of Native Studies, XVIII*(1), 37-48.

Lazarus, R. S. (1981). The stress and coping paradigm. In C. Eisdorfer, D. Cohen, A. Kleinman, & P. Maxim (Eds.), *Models for clinical psychopathology* (pp. 177-214). New York: Spectrum.

Lazarus, R. S. (1993). Coping theory and research: Past, present and future. *Psychosomatic. Medicine,* 55(3), 234-247.

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal and coping*. New York: Springer.

Leichter J. (1986). Effect of paternal ethanol ingestion on fetal growth in rats. *Growth, 50*(2), 228-33.

Lemoine, P., Harousseau, H., Borteyru, J.P., & Menoet, J.C. (1968) Children of alcoholic parents: Anomolies observed in 127 cases. *Quest Medicale, 21,* 476-482.

Leonardson, G. R., & Loudenburg, R. (2003). Risk factors for alcohol use during pregnancy in a multistate area. *Neurotoxicology and Teratology, 25,* 651-658.

Leslie, M., & Roberts, G. (2001). Enhancing Fetal Alcohol Syndrome (FAS) – Related interventions at the prenatal and early childhood stages in Canada. *Executive Summary: Breaking the Cycle Program and the Canada Centre on Substance Abuse.* Toronto, ON: Mothercraft.

Li, C., Olsen, Y., Kvigne, V., & Welty, T. (1999). Implementation of substance use screening in prenatal clinics. *South Dakota Journal of Medicine, 52*(2), 59-64.

Liebermann, M. A. (1986). Social supports – The consequences of psychologizing: A commentary. *Journal of Consulting and Clinical Psychology, 54,* 461-465.

Lipowski, Z. J. (1970). Physical illness, the individual and the coping processes. *Psychiatry in Medicine, 1*(3), 91-102.

Livy, D. J., Maier, S. E., & West, J. R. (2004). Long-term alcohol exposure prior to conception results in lower fetal body weights. Birth Defects Research. Part B. *Developmental and Reproductive Toxicology, 71*(3), 135 - 41.

Loock, C. A. (1990) Targeting high-risk families: Prenatal alcohol/drug abuse and infant outcomes. Sunny Hill Hospital for Children. University of British Columbia: Vancouver, BC.

Lopez, M. A., & Mermelstein, F. J. (1987). *Stress, coping and adaptation to chronic illness in hospitalized elderly: Predictors of treatment outcome*. Department of Psychology and Social Sciences. Chicago: Rush University Press.

Lundsberg, L. S., Bracken, M. B. and Saftlas, A. F. (1997) Low-to-moderate gestational alcohol use and intrauterine growth retardation, low birthweight, and preterm delivery. *AEP, 7*(7), 498-508.

Ma., G. X., Toubbeh, J., Cline, J., & Chisholm, A. (1998). Native American adolescents' views of Fetal Alcohol Syndrome prevention in schools. *Journal of School Health, 8*(4), 131-136.

MacKinnon, D., P., Williams-Avery, R., & Pentz, M. (1995). Youth beliefs and knowledge about the risks of drinking while pregnant. *Public Health Reports*, *110,* 754-763.

Maier, S. E., & West, J. R. (2001). Drinking patterns and ethanol-related birth defects. *AlcoholResearch & Health, 25*(3), 168-174.

Maillard, T., Lamblin, D., Lesure, J. F., & Fourmaintraux, A. (1999). Incidence of Fetal Alcohol Syndrome on the southern part of Reunion Island (France). *Teratology,* 60(2), 51-52.

Majewski, E., Michaelis, R., Moosman, K., et al. (1975). Low birthweight dwarfism: The Dubowitz syndrome. *Z. Kinderheilkd, 120*, 283.

Manne, S. L., & Zautra, A. J. (1990). Couples coping with chronic illness: Women with rheumatoid arthritis and their healthy husbands. *Journal of Behavioral Medicine, 13*(4), 327-342.

Marin, G. (1997). Changes across 3 years in self-reported awareness of product warning messages in a Hispanic community. *Health Education Research: Theory & Practice, 12*(1), 103-116.

Marino, M. D., Aksenov, M. Y., & Kelly, S. J. (2004). Vitamin E protects against ethanol-induced cell loss and oxidative stress in the neonatal rat hippocampus. *International Journal of Developmental Neuroscience*, *22*(5-6), 363-377.

Mathis, M. P., Lavoie, M., Hadley, C., & Toomey, K. (1995). Birth certificates as a source for Fetal Alcohol Syndrome case ascertainment - Georgia, 1989-1992. *Morbidity and Mortality Weekly Report, 46*(47), 1118-1120.

Mattson, S. N., & Riley, E. P. (1995). Prenatal exposure to ethanol: What the images reveal. *Alcohol Health & Research World, 19*(4), 273-278.

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

May, P. A., & Gossage, J. P. (2001). Estimating the prevalence of fetal alcohol syndrome: A summary. *Alcohol Research & Health, 25,* 159-167.

May, P. A., & Hymbaugh, K. J. (1989). A macro-level Fetal Alcohol Syndrome prevention program for Native Americans and Alaska natives: Description and Evaluation. *Journal of Studies on Alcohol, 50,* 508-518.

May, P. A., Hymbaugh, K. J., Aase, J. M., & Samet, J. M. (1983). Epidemiology of Fetal Alcohol Syndrome among American Indians of the Southwest. *Social Biology, 30*(4), 374-387.

May, P.A., & Moran, J.R. (1995). Prevention of alcohol misuse: A review of health promotion efforts among American Indians. *American Journal of Health Promotion, 9*(4), 288-299.

McAlhany, R. E., West, J. R., & Miranda, R. C. (2000). Glial-derived neurotrophic (GDNF) prevents ethanol induced apoptosis and JUN Kinase phosphorylation. *Developmental Brain Research, 119*(2), 209-216.

McCarver, D. G., Thomasson, H. R., Martier, S. S., Sokol, R. J. and Li, T. K. (1997). Alcohol Dehydrogenase-2*3 Allele Protects Against Alcohol-Related Birth Defects Among Afriance Americans. *Journal of Pharmacology and Experimental Therapeutics, 283*(3),1095-1101.

McCrae, R. R. (1999). Age differences and changes in the use of coping mechanisms. *Journal of Gerontology, 44*(6), 161-169.

McWilliam, C. L., Stewart, M., Brown, J. B., McNair, S., Desai, K., Patterson, M. L., Del Maestro, N., & Pittman, B. J. (1997). Creating empowering meaning: An interactive process of promoting health with chronically ill older Canadians. *Health Promotion International, 12*(2), 111-123.

Miers, S. (1999). Fetal Alcohol Syndrome in South Australia and a report on the 1999 Prairie Province Conference on Fetal Alcohol Syndrome. http://users.chariot.net.all/~miers/fasrep4.PDF

Miller, J. (1983). *Coping with chronic illness: Overcoming powerlessness.* Philadelphia: F.A. Davis.

Miller, L., Tolliver, R., Druschel, C., et al. (2002). Fetal Alcohol Syndrome - Alaska, Arizona, Colorado, and New York, 1995-1997. *MMWR, 51*(20), 433-435.

Miller, L.A., Shaikh, R., Stanton, C., Montgomery, A., Rickard, R., Keefer, S., & Hoffman, R. (1995). Surveillance for fetal alcohol syndrome in Colorado. *Public Health Reports, 110*, 690-697.

Miller, R. R., Olson, B. M., Rorick, N., Wittingen, A. L., & Bullock, M. (2003). Embryonic exposure to exogenous tocopherol partially attenuates ethanol-induced changes in brain morphology and brain membrane fatty acid composition. *Nutritional Neuroscience, 6*(4), 201-212.

Miller, W. R., & C'DeBaca, J. (1995). What every mental health professional should know about alcohol. *Journal of Substance Abuse Treatment, 12*(5), 355-365.

Mills, J. L., & Graubard, B. I. (1987). Is moderate drinking during pregnancy associated with an increased risk for malformations? *Pediatrics, 80*, 309-314.

Minor, M. J., & Bernice, V. (1982). Prevention research on the tetratogenic effects of alcohol. *Preventive Medicine, 11,* 346-359.

Mitchell, J. J., Paiva, M., & Heaton, M. B. (1999a). The antioxidants Vitamin E and beta-carotene protect against ethanol-induced neurotoxicity in embryonic rat hippocampal cultures. *Alcohol, 17(*2), 163-168.

Mitchell, J. J., Paiva, M., & Heaton, M. B. (1999b). Vitamin E and beta-carotene protect against ethanol combined with ischemia in an embryonic rat hippocampal culture model of fetal ethanol syndrome. *Neuroscience Letters, 263*(2-3), 189-192.

Mitchell, J. J., Paiva, M., Blaine-Moore, D., Walker, D. W., & Heaton, M. B. (1998). A comparative study of ethanol, hypoglycemia, hypoxia and neurotrophic factor interactions with fetal rat hippocampal neurons: A multi-factor in vitro model for developmental ethanol effects. *Developmental Brain Research, 105*, 241-250.

Mitchell, K. T. (1999). Preventing fetal alcohol syndrome. *Journal of Pediatric Health Care, 13*(2), p. 87-89.

Moore, D. B., Madorsky, I., Paiva, M., & Heaton, M. B. (2004). Ethanol exposure alters neurotrophin receptor expression in the rat central nervous system: Effects of prenatal exposure. *Journal of Neurobiology, 60*(1), 101-113.

Moreland, N., La Grange, L., & Montoya, R. (2002). Impact of in utero exposure to ETOH on corpus callosum development and paw preference in rats: Protective effects of silymarin. *BMC Complementary and Alternative Medicine, 2*, 1-6.

Morrisette, P. J. (2001). Fetal alcohol syndrome: Parental experiences and the role of the family counselors. *The Qualitative Report, 6*(2). http://www.nova.edu/ssss/QR/QR6-2/morrisette.html

Morrow-Tlucak, M., Ernhart, C. B., Sokol, R. J., Martier, S., & Ager, J. (1989). Underreporting of alcohol use in pregnancy: Relationship to alcohol problem history. *Alcoholism: Clinical and Experimental Research*, *13*(3), 399-401.

Mullen, B., & Sulls, J. (1982). The effectiveness of attention and rejection as coping styles: A meta-analysis of temporal differences. *Journal of Psychosomatic Research, 26,* 43-49.

Murphy-Brennan, M., & Oei, T. P. (1999). Is there evidence to show that Fetal Alcohol Syndrome can be prevented? *Journal of Drug Education*, *29*(1), 5-24.

Neese, S., La Grange, L., Trujillo, E., & Romero, D. (2004). The effects of ethanol and silymarin treatment during gestation on spatial working memory. *BMC Complementary and Alternative Medicine, 4,* 1-8.

Nissen, S. J., & Newman, W. P. (1992). Factors influencing reintegration to normal living after amputation. *Archives of Physical Medicine and Rehabilitation, 73*(6), 548-551.

Noonan, J. A. (1976). Congenital heart disease in the fetal alcohol syndrome. *American Journal of Cardiology, 37,* 160.

Northern and Central Alberta Perinatal Outreach Program. (2003). *Alberta perinatal data 2001* [Data file]. Edmonton, AB: Author.

O'Brian, M. (1995) *A critical mess.* London and New York: Routeldge.

O'Connor, M. J., & Whaley, S. E. (2003). Alcohol use in pregnant low-income women. *Journal of Studies on Alcohol, 64,* 773-783.

Ockene, J. K., Ma Y., Zapka, J. G., Pbert, L. A., Goins, K. V., & Stoddard, A. M. (2002). Spontaneous Cessation of smoking and alcohol use among low income pregnant women. *American Journal of Preventive Medicine, 23,* 150-159.

Oklahoma State Department of Health – OSDH (1991). *Alcohol consumption levels of Oklahoma mothers before and during pregnancy,* Author.

O'Leary, C. (2002). *Fetal alcohol syndrome: A literature review*. National Alcohol Strategy 2001-2003-4. Occasional Paper, Commonwealth of Australia.

O'Leary, C. M. (2004). Fetal alcohol syndrome: Diagnosis, epidemiology, and developmental outcomes. *Journal of Pediatric and Child Health, 40,* 2-7.

Olney, J. W. (2004). Fetal Ethanol Syndrome at the cellular level. *Addiction Biology, 9*(2), 137-149.

Olson, H. C., Streissguth, A. P., Sampson, P., Barr, H. M., & Bookstein, F. (1997). Association of prenatal alcohol exposure with behavioral and learning problems in early adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 1187-1188.

Olsen, J., Frische, G., Poulsen, A. O., & Kirchheiner, H. (1989). Changing smoking, drinking and eating behavior among pregnant women in Denmark. *Scandinavian Journal of Social Medicine, 17,* 277-280.

Overholser, J. C. (1990). Fetal alcohol syndrome: A review of the disorder. *Journal of Contemporary Psychotherapy, 20*(3), 163-176.

Padmanabhan, R., & Shafiullah, M. (2004). Effect of maternal diabetes and ethanol interactions on embryo development in the mouse. *Molecular & Cellular Biochemistry, 261*(1-2), 43-56.

Palinkas, L. A., Atkins, C. J., Miller, C., & Diane, F. (1996). Social skills training for drug prevention in high-risk female adolescents. *Preventive Medicine, 25,* 692-701.

Palomo, T., Archer, T., Beninger, R .J., & Kostrzewa, R. M. (2002). Neurodevelopmental liabilities of substance abuse. *Neurotoxicity Research, 4*(4), 267-279.

Parker-Langley, L. (2002). Alcohol prevention programs among American Indians: Research findings and issues. In P. D. Mail (Ed.), *Alcohol use among American Indians and Alaska Natives: Multiple perspectives on a complex problem.* Bethesda, MD: U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, National Institute on Alcohol Abuse and Alcoholism

Parry, C. D. H. (2000). Alcohol problems in developing countries: Challenges for the new millennium. *Suchtmed, 2*(4), 216-220.

Peak, K. & Del Papa, F. S. (1993). Criminal justice enters the womb: Enforcing the "right" to be born drug-free. *Journal of Criminal Justice, 21,* 245-263.

Pearlin, L., & Schooler, C. (1978). The structure of coping. *Journal of Health and Social Behaivor, 19*(1), 2-21.

Peng, Y., Yang, P-H., Ng, S. S. M., Wong, O. G., Liu, J., He, M-L., Kung, H-F., & Lin, M. C. M. (2004). A critical role of Pax6 in ethanol-induced fetal microcephaly. *Neurobiology of Disease, 16,* 370-376.

Penninx, B. W. J. H, Van Tilburg, T., Deeg, D. J. H., & Kriegsman, D. M. W. (1997). Direct and buffer effects of social support and personal coping resources in individuals with arthritis. *Social Science and Medicine, 44*(3), 393-402.

Perham-Hester, K. A., & Gessner, B. D. (1997). Correlates of drinking during the third trimester of pregnancy in Alaska. *Maternal and Child Health Journal, 1*(3), 165-172.

Perry, C., Williams, C. L., Veblen-Mortenson, S., Toomey, T. L., Komro, K., Anstine, P. S., McGovern, P., Finnegan, J. R., Forster, J. L., Wagenaar, A., & Wolfson, M. (1996). Project Northland: Outcomes of a communitywide alcohol use prevention program during early adolescence. *American Journal of Public Health, 86*(9), 956-965.

Pfeiffer, J., Majewski, F., Fischbach, H., Bierich, J. R., & Volk, B. (1979). Alcohol embryo- and fetopathy. *Journal of Neurological Sciences, 41,* 125-137.

Pittman, D. J., Staudenmeier, W. J., Jr, & Kaplan, A. (1991). Alcohol and other drugs: the response of the political and medical institutions. *British Journal of Addiction, 86,* 967-975.

Plaisier, K. J. (1989). Fetal alcohol syndrome prevention in American Indian communities of Michigan's upper peninsula. *American Indian and Alaska Native Mental Health Research, 3*(1), 16-33.

Plant, M. L. (1984). Alcohol consumption during pregnancy: baseline data from a Scottish prospective study. *British Journal of Addiction, 79*(2), 207-214.

Poole, N. (2000). Evaluation report of the Sheway Project for high-risk pregnant and parenting women. British Columbia Centre of Excellence for Women's Health, Sheway Project, Vancouver, BC. Retrieved 8/26/2004 from http://www.bccewh.bc.ca/PDFs/shewayreport.pdf.

Program: Minnesota Media Campaign to promote alcohol-free pregnancy. (1996). *Health Education Quarterly, 23,* 418-419.

Railton, C. J. (2003). What do mothers of FAS children have in common (a critical review of the study Characteristics of mothers who have children with fetal alcohol syndrom or some characteristics of fetal alcohol syndrom). *Journal of FAS International, 1,* e18.

Randall, C. L., Anton, R. F., & Becker, H. C. (1987). Ethanol, pregnancy, and prostaglandins. *Alcoholism: Clinical and Experimental Research, 11*, 32-36.

Reid, C., Edwards, J., Wang, M., Manybeads, Y., Mike, L., Martinez, N., La Grange, L., & Reyes, E. (1999). Prevention by a silymarin/phospholipid compound of ethanol-induced social learning deficits in rats. *Planta Medica, 65*, 421-424.

Revenson, T. A., Wollman, C. A., & Felton, B. J. (1983). Social supports as stress buffers for adult cancer patients. *Psychosomatic Medicine, 45*, 321-331.

Rhodes, J. E., Gingiss, P. L., & Smith, P. B. (1994). Risk and Protective Factors For Alcohol Use Among Pregnant African-American, Hispanic, and White Adolescents: The Influence of Peers, Sexual Partners, Family Members, and Mentors. Addictive Behaviors, 19(5), 555-564.

Riley, E. P., Barron, S., Melcer, T, & Gonzalez, D. (1993). Alterations in activity following alcohol administration during the third trimester equivalent in P and NP rats. *Alcoholism: Clinical & Experimental Research, 17*(6), 1240-6.

Riley, E. P., McGee, C. L., & Sowell, E. R. (2004). Teratogenic effects of alcohol: A decade of brain imaging. *American Journal of Medical Genetics, 127C(*1), 35-41.

Ris, H. W. (1988). Fetal Alcohol Syndrome: Legislation urgently needed. *Journal of Public Health Policy, 9*, 556-558.

Robertson, G., & Nanson, J. (2000). *Best practices: Fetal alcohol syndrome? Fetal alcohol Effects and the effects of other substance use during pregnancy*. Health Canada.

Robinson, G. C., Conry, J. L., & Conry, R. F. (1987). Clinical profile and prevalence of Fetal Alcohol Syndrome in an isolated community in British Columbia. *Canadian Medical Association Journal, 137*(3), 203-207.

Rostand, A., Kaminski, M., Lelong, N., Denaene, P., Delestret, I., Klein-Bertrand, C., Querleu, D., & Crepin, G. (1990). Alcohol use in pregnancy, craniofacial features, and fetal growth. *Journal of Epidemiology and Community Health, 44*, 302-306

Roth, S., & Cohen, L. J. (1986). Approach, avoidance and coping with stress. *American Psychologist, 41*, 813-819.

Rothschild, M. L. (1999). Carrots, sticks, and promises: A conceptual framework for the management of public health and social issue behaviors. *Journal of Marketing, 63*(10), 24-37.

Rotter, J., (1996). Generalised expectancies for internal and external control of reinforcement. *Psychological Monographs, 80*, 609.

Rummelhart, D. E., & Norman, D. A. (1981). Analogical processes in learning. In Anderson, J. R. (ed.), *Cognitive Skills and Their Acquisition*. Hillsdale, NJ: Lawrence Erlbaum, pp. 335-359.

Russell, M. (1991). Clinical implications of recent research on the fetal alcohol syndrome. *Bulletin of the New York Academy of Medicine, 67*(3), 207-222**.**

Russell, M., Martier, S. S., & Sokol, R. J. (1994). Screening for pregnancy risk-drinking. *Alcohol Clinical and Experimental Research, 18*(5), 1156-61.

Russell, M., Martier, S. S., Sokol, R. J., Mudar, P., Jacobson, S., & Jacobson, J. (1996). Detecting risk drinking during pregnancy: a comparison of four screening questionnaires. *American Journal of Public Health, 86*(10), 1435-1439.

Sampson, P. D., Streissguth, A. P., Bookstein, F. L., Little, R. E., Clarren, S. K., Dehaene, P., Hanson, J. W., & Graham Jr., J. M. (1997). Incidence of Fetal Alcohol Syndrome and prevalence of Alcohol-Related Neurodevelopmental Disorder. *Teratology*, 56, 317-326.

Sari, Y., & Zhou, F. C. (2004). Prenatal alcohol exposure causes long-term serotonin deficit in mice. *Alcoholism: Clinical & Experimental Research, 28*(6), 941-948.

Scott, D. W., Oberst, M. T., & Dropkin, M. J. (1980). A stress-coping model. *ANS Advanced Nursing Science, 3*(1), 9-23.

Serdula, M., Williamson, S. F., Kendrick, J. S., Anda, R. F.. & Byers, T. (1991). Trends in alcohol consumption by pregnant woman. *Journal of the American Medical Association, 265,* 876-879.

Seyoum, G. G., & Persaud, T. V. N. (1991). Can methionine and zinc prevent the embryopathic effects of alcohol? *Medical Hypotheses, 34*, 153-156.

Sharpe, T. T., Alexander, M., Hutcherson, J., Floyd, R.L., Brimacombe, M., Levine, R., Mengel, M., & Stuber, M. (2004). Physician and allied health professionals' training and Fetal Alcohol Syndrome. *Journal of Women's Health, 13*(2), 133-139.

Sherbourne, C. D., Meredith, L. S., Rogers, W., & Ware, J. E. J. (1992). Social support and stressful life events: Age differences in their effects on health-related quality of life among the chronically ill. *Quality of Life Research, 1,* 235-246.

Shostak, M., & Brown, L. B. (1995). American Indians' knowledge about Fetal Alcohol Syndrome: An exploratory study. *American Indian Culture and Research Journal, 19*(1), 39-63.

Shumaker, S. A., & Brownell, A. (1984). Toward a theory of social support: Closing conceptual gaps. *Journal of Social Issues, 40,* 11-36.

Siler-Khodr, T. M., Yang, Y., Grayson, M. H., Henderson, G. I., Lee, M., Schenker, S. (2000). Effect of ethanol on thromboxane and prostacyclin production in the human placenta. *Ethanol, 21,* 169-180.

Simmel, G. (1950). The Sociology of George Simmel. New York Press.

Single, E. (2002). Alcohol and Youth: Time for Effective Action. Canadian Journal of Public Health, 93(3), 169-172.

Smith, D. W. (1980). Alcohol effects on the fetus. *Progress in Clinical and Biological Research, 36*, 73-82.

Solomon, R., & Peterson, M. (1994). Successful aging – How to help your patients cope with change. *Geriatrics, 49*(4), 41-47.

Sokol, R. J. (1981). Alcohol and abnormal outcomes of pregnancy. CMA Journal, 125, 143-148.

Sokol, R., Martier, S., & Ager, J. W. (1989). The T-ACE questions: Practical prenatal detection of risk-drinking. *American Journal of Obstetrics and Gynecology, 160,* 863-871.

Sokol, R. J., Ager, J., Martier, S., Debanne, S., Ernhart, C., Kuzma, J., & Miller, S. I. (1986). Significant determinants of susceptibility to ethanol teratogenicity. *Annals of the New York Academy of Sciences, 477,* 87-101.

Sokol, R. J., Miller, S. I., & Reed, G. (1980.) Alcohol abuse during pregnancy: An epidemiologic study. *Alcoholism: Clinical and Experimental Research, 4*(2), 135-145.

Southern Alberta Perinatal Outreach Program. (2003). *Alberta perinatal data 2001* [Data file]. Calgary, AB: Author.

Sowell, E. R., Jernigan, T. L., Mattson, S. N., Riley, E. P., Sobel, D. F., & Jones, K. L. (1996). Abnormal development of the cerebellar vermis in children prenatally exposed to alcohol: Size and reduction in lobules I-V. *Alcohol: Clinical and Experimental Research, 20*, 31-34.

Spinney, L. (1999). New peptides prevent brain damage. *Molecular Medicine Today, 5*.

Spong, C. Y., Abebe, D. T., Gozes, I., Brenneman, D. E., & Hill, J. M. (2001). Prevention of fetal demise and growth restriction in a mouse model of Fetal Ethanol Syndrome. *The Journal of Pharmacology and Experimental Therapeutics, 297,* 774-779.

Spong, C. Y., Auth, J., Vink, J., Goodwin, K., Abebe, D. T., Hill, J. M., & Brenneman, D. E. (2002). Vasoactive intestinal peptide mRNA and immunoreactivity are decreased in Fetal Ethanol Syndrome model. *Regulatory Peptides, 108*(2-3), 143-147.

Statistics Canada. (2002a). *Age (122) and sex (3) for population, for Canada, provinces, territories, censusmetropolitan areas and census agglomerations, 2001 census—100% data* [On-line database tabulation]. Retrieved June 25, 2004, from http://www12.statcan.ca/english/census01/products/standard/themes/RetrieveProductTable.cfm?Temporal=2001&PID=55437&METH=1&APATH=3&PTYPE=55430&THEME=37&FREE=0&AID=0&FOCUS=0&VID=0&GC=99&GK=NA&SC=1&CPP=99&SR=1&RL=0&RPP=9999&D1=0&D2=0&D3=0&D4=0&D5=0&D6=0&GID=431633

Statistics Canada. (2002b). *Canadian community health survey (CCHS), Cycle 1.1*. Available from http://www.statcan.ca/english/concepts/health/index.htm

Steptoe, S., & Wardle, J. (1996). The European health and behaviour survey: The development of an international study in health psychology. *Psychology and Health, 11*, 49-73.

Stoler, J. M., Huntington, K. S., Peterson, C. M., Peterson, K. P., Daniel, P., Aboagye, K. K., Lieberman, E., Ryan, L., & Holmes, L. B. (1998). The prenatal detection of significant alcohol exposure with maternal blood markers. *Journal of Pediatrics, 133*(3), 346-352.

Stoler, J. M., & Holmes, L. B. (2004). Recognition of facial features of Fetal Alcohol Syndrome in the newborn. *American Journal of Medical Genetics, 127*, 21-27.

Stoler, J. M., Ryan, L. M. & Holmes, L. B. (2002) Alcohol dehydrogenase 2 genotypes, maternal alcohol use, and infant outcome. *J Pediatr, 141*, 780-785.

Stratton, K., Howe, C., & Battaglia, F. (Eds.). (1996). *Fetal Alcohol Syndrome diagnosis, epidemiology, prevention, and treatment.* Washington, DC: National Academy Press.

Streissguth, A. P. (1994). A long-term perspective of FAS. *Alcohol Health & Research World, 18*.

Streissguth, A.P., & Dehaene, P. (1993). Fetal alcohol syndrome in twins of alcoholic mothers: Concordance of diagnosis and IQ. *American Journal of Medical Genetics, 47*, 857-861.

Streissguth, A. P. (1997). *Fetal alcohol syndrome: A guide for families and communities.* Baltimore, MD: Paul Brooks Publishing Company.

Streissguth, A. P. (1998). *Fetal alcohol syndrome: A guide for families and communities.* Baltimore, MD: Paul H. Brooks.

Streissguth, A. P., Barr, H. M., Bookstein, F. L., Sampson, P. D., & Olson, H. C. (1999). The long-term neurocognitive consequences of prenatal ethanol exposure: A 14 year study. *Psychological Science, 10*(3), 186-190.

Streissguth, A. P., Bookstein, F. L., Barr, H. M., Press, S., & Sampson, P. D. (1998). A fetal alcohol scale. *Alcoholism: Clinical and Experimental Research*, *22*(2), 325-333.

Streissguth, A. P., Bookstein, F. L., Sampson, P. D., & Barr, M. H. (1993). *The enduring effects of prenatal alcohol exposure on child development: Birth through 7Years, A partial least squares solution*. Ann Arbor, MI: University of Michigan Press.

Streissguth, A. P., Kogan, J., Bookstein, F. L., & Barr, H. (1996*). Understanding the occurrence of secondary disabilities in clients with Fetal Alcohol Syndrome and Fetal Alcohol Effects*. Seattle, WA: University of Washington Press.

Streissguth, A. P., Landesman-Dwyer, S., Martin, J. C., & Smith, D. W. (1980). Teratogenetic effects of alcohol in humans and laboratory animals. *Science; 209*, 353-361**.**

Substance Abuse and Mental Health Services Administration (SAMHSA). *SAMHSA Model Programs.* Retrieved Early 2004 from the World Wide Web: http://modelprograms.samhsa.gov/

Substance Abuse and Mental Health Services Administration (SAMHSA), Center for reproductive law and policy. (1996). Punishing women for the behavior during pregnancy: an approach that undermines women's health and children's interests. Retrieved July 18, 2004 from http://www.lindesmith.org/library/womrepro.cfm?printpage=1

Sugisawa, H., Liang, J., & Liu, X. (1994). Social networks, social support, and mortality among older people in Japan. *Journals of Gerontology, 49*(1), S3-S13.

Svikis, D. S., Golden, A. S., Huggins, G. R., Pickens, R. W., McCaul, M. E., Velez, M. L., Rosendale, T. C., Brooner, R. K., Gazaway, P. M., Stitzer, M. L., & Ball, C. E. (1997). Cost-effectiveness of treatment for drug-abusing pregnant women. *Drug and Alcohol Dependence, 45,* 105-113.

Tajuddin, N. F., & Druse, M. J. (2001). A persistent deficit of serotonin neurons in the offspring of ethanol-fed dams: Protective effects of maternal ipsapirone treatment. *Developmental Brain Research, 129,* 181-188.

Tanaka, H., Inomata, K., & Arima, M. (1983). Zinc supplementation in ethanol-treated pregnant rats increases the metabolic activity in the fetal hippocampus. *Brain & Development, 5/6,* 549-554.

Testa, M., & Leonard, K. E. (1995). Social influences on drinking during pregnancy. *Psychology of Addictive Behaviors, 9*(4), 258-268.

Thomas, J. D., & Riley, E. P. (1998). Fetal Ethanol Syndrome: Does ethanol withdrawal play a role? *Ethanol Health & Research World, 22*(1), 47-53.

Thornbury, K. M. (1982). Coping implications for health practioners. *Patient Counsellin in Health Education, 4*(1), 3-9.

Vaudry, D., Rouselle, C., Basille, M., Falluel-Morel, A., Pamantung, T. F., Fontaine, M., Fournier, A., Vaudry, H., & Gonzalez, B. J. (2002). Pituitary adenylate cyclase-activating polypeptide protects rat cerebellar granule neurons against ethanol-induced apoptotic cell death. *PNAS, 99*(9), 6398-6403.

Veiel, H .O., & Baumann, U. (1992). *The meaning and measurement of social support*. New York: Hemisphere.

Verbrugge, L .M. (1990). Disability. *Rheumatic Diseases Clinics of North America, 16*(3), 741-761.

Viljoen, D. L., Carr, L. G., Foroud, T. M., Brooke, L., Ramsay, M., & Li, T. K. (2001). Ethanol dehydrogenase-2*2 allele is associated with decreased prevalence of Fetal Ethanol Syndrome in the mixed-ancestry population of the Western Cape Province, South Africa. *Alcoholism: Clinical and Experimental Research, 25*(12), 1719-1722.

Viljoen, D., Croxford, J., Gossage, P., Kodituwakku, P.W. and May, P.A. (2002) Characteristics of Mothers of Children with Fetal Alcohol Syndrome in the Western Cape Province of South Africa: A Case Control Study. *J. Stud. Alcohol, 63*, 6-17.

Viljoen, D., Craig, P., Hymbaugh, K., Boyle, C., & Blount, S. (2003). Fetal Alcohol Syndrome - South Africa, 2001. *Morbidity and Mortality Weekly Report, 52*(28), 660-662.

Vinson, D. C., Galliher, J.M., Reidinger, C., & Kappus, J.A. (2004). Comfortably engaging: Which approach to alcohol screening should we use? *Annuls of Family Medicine, 2*(5), 398-404.

Walpole, I., Zubrick, S., & Pontre, J. (1990). Is there a fetal effect with low to moderate alcohol use before or during pregnancy? *Journal of Epidemiology and Community Health, 44*(4), p. 297-301.

Warner, R. H. & Rosett, H. L. (1975). Effects of drinking on offspring. *J. Stud. Alcohol, 36*, 1395.

Waterson, E. J., & Murray-Lyon, I. M. (1990). Preventing alcohol related birth damage: A review. *Social Science and Medicine, 30,* 349-360.

Weisman, A. (1979). *Coping and cancer.* New York: McGraw-Hill.

Weisman, A., & Worden, W. (1977). The existential plight in cancer: Significance of the first 100 days. *International Journal of Psychiatry and Medicine, 7,* 1-15.

Whitfield, J.B. (2001). Gama glutamyl transferase. *Critical Reviews in Clinical Laboratory Sciences, 38*(4), 263-355.

Wiemann, C. M., & Berenson, A. B. (1998). Factors associated with recent and discontinued alcohol use by pregnant adolescents. *Journal of Adolescent Health, 22,* 417-423.

Williams, R .J., Odaibo, F. S., & McGee, J. M. (1999). Incidence of fetal alcohol syndrome in northeastern Manitoba. *Canadian Journal of Public Health, 90*(3), 192-194.

Williams, R. J.**,** & Gloster, S. P. (1999). Knowledge of fetal alcohol syndrome in northern Manitoba Cree. *Journal of Studies on Alcohol, 60,* 833-837.

Wilson-Jones., M., & Bass, W. T. (2003) Follow up of the high-risk infant fetal alcohol syndrome. *Journal of Neonatal Nursing, 22*(3), 63-70.

Witte, K. (1992). Putting the fear back into fear appeals: Reconciling the literature. *Communication Monographs, 59,* 329-349.

World Health Organization. (1998). *Life in the 21st century: A vision for all.* Geneva: WHO

World Health Organization. (2004). *Global status report: Alcohol policy.* Geneva, Switzerland. Author.

Wrong, D. (2003). The influences of sociological ideas on American culture. In H. Gans, (Ed.), *Sociology in America* (19-30). Newbury Park, CA: Sage Publications.

Yang, Q., Witkiewicz, B. B., Olney, R. S., Liu, Y., Davis, M., Khoury, M. J., Correa, A., & Erickson, D. (2001). A Case-Control Study of Maternal Alcohol Consumption and Intrauterine Growth Retardation. *Annals of Epidemiology, 11,* 497-503.

Yukon Liquor Commission. (2002). *Social responsibility.* Retrieved November 18, 2004 from the World Wide Web: http://www.ylc.yk.ca/socialresp.htm.

Zell, J. A., Montague, J. R., Lopez, T. F., & Mudd, L. M. (1999). Inhibition of ethanol neurotoxicity by treatment with growth factors and estrogen. *McGill Journal of Medicine, 5,* 13-24.

Zeller, S. (1998). Fetal abuse laws gain favor. *National Journal, 30*(30), 1758.

Zhand, E., & Klein, D. (1997). The needs of pregnant and parenting American Indian women at risk for problem alcohol and drug use. *American Indian Culture and Research Journal, 21*(3), 119-143.

Zhou, F. C., Sari, Y., Powrozek, T. A., & Spong, C.Y. (2004). A neuroprotective peptide antagonizes fetal ethanol exposure-compromised brain growth. *Journal of Molecular Neuroscience, 24(2),* 189-200.

Appendix A
FASD Data Extraction Form

Add/Edit FASD Data TEMP

Name:

Complete Citation:

Aims/Objectives:

Conclusions:

Type of article:

- Editorial ☐
- Experiment ☐
- Commentary ☐
- Review ☐
- Program Description ☐
- Policy ☐
- Other text

Description of independent variable:

Primary:

Secondary:	
Tertiary:	
FASD/FAE Prevention:	○ Yes ○ No
	Reviewer's Decision:
Include paper in supporting chapter?	○ Yes ○ No
	Which chapter? (include page numbers in box provided)
Incidence and prevalence: Most common symptoms, effect on physical/behavioral/cognitive:	
Preventative Strategies:	
Prevention Policy:	
Definitions/theories/indicies/general:	
Differences between FASD, FAS, FAE, ARND, ARBD:	
Differences among cultures (Aboriginals or others):	
Measurement of FASD/FAS/FAE/ARND/ARBD:	
Identification of Risk:	
Protective Factors:	
Implementation of Strategies:	
Evaluation Strategies:	
Other:	
Other Text:	
	Reasons for rejection?
Not relevant?	☐

Reason Other	
	Outcome Measures:
Knowledge:	
Attitude:	
Behaviour:	
Other:	
	Research Methods:
Quantitative:	
Survey (epidemiological)	☐
Experimental	☐
Randomized Controlled Trial	☐
Quasi Experiment	☐
Controlled Observational	☐

Qualitative:

Phenomenology ☐

Ethnography ☐

Grounded Theory ☐

Case Study ☐

Biography ☐

Observation ☐

Interview ☐

Triangulation: ☐

Data

Researchers ☐

Methods ☐

Samples ☐

Quality Measure: ○1 ○2 ○3

Study Methods (include tools and instruments):

Sample: ○Selective ○Representative

N=

Country:

Culture:

Inclusion/Exclusion Criteria:

What information was collected?

Population:

Environment:

Individual Characteristics:	
When was info collected?	
How was info collected?	
	Data Analysis/Study Results:
What methods were used?	
Results:	
	Models of Best Practice:
Type of Prevention Model:	

Description for survey/literature review type of articles:	

Other Comments/summary

Other Comments: (Mention key recommendations/ implications discussed by the authors here)	

Add