IS MATERNAL

ALCOHOL USE DURING PREGNANCY A

RISK FACTOR FOR SUDDEN INFANT DEATH SYNDROME IN MANITOBA?

By

Larry J. Burd

A Thesis
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A Thesis/Practicum submitted to the Faculty of Graduate Studies of the University of Manitoba in partial fulfillment of the requirements for the degree of

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ABSTRACT

OBJECTIVE: To determine if maternal alcohol use during pregnancy was a risk factor for Sudden Infant Death Syndrome (SIDS) in Manitoba. METHOD: A retrospective case-control methodology. The case definition for the study was: (a) Infant deaths occurring between 1986 - 1990, (b) reviewed by the Manitoba Pediatric Death Review Committe and by the Manitoba Medical Examiner, and (c) classified as SIDS were included. Two groups were compared: 104 SIDS cases and 104 living controls, matched by year of death, hospital of birth, race and maternal smoking status during pregnancy. A third unmatched comparison group of 105 non-SIDS infant deaths was also studied. Data related to the independent variable of prenatal alcohol exposure was routinely collected by nurses during the hospital stay and was recorded on the Manitoba Maternal Nursing Database. RESULTS: Univariate comparisions of cases to matched living controls did not identify alcohol as a SIDS risk factor; (OR 0.91; 95% CI 0.50 to 1.67; McNemar chi-square .09; $p < .764$). Logistic regression modeling to control for confounding produced a three variable model: unmarrried mother (OR = 3.08); birthweight (OR = 0.17); and mothers age (OR = 0.89). The model parameters were: $\chi^2 = 35.81$; 3 df; $p < .001$. Alcohol exposure in a sub-sample of Aboriginals only (n = 42) with matched controls was also not significant (OR = 1.50; 95% CI 0.53 to 4.21). A logistic regression model for this group included only one variable significant at the .05 level, that of unmarried mother (OR =

6.00). Model parameters were:$\chi^2 = 7.93$; $p < .005$. A similar analysis a sub-sample of Whites only (n= 62) was also not significant for alcohol exposure (OR = 0.69; 95% CI 0.32 to 1.48); the logistic regression model for whites included two variables; not breastfeeding OR 7.65; birthweight (OR = 1.00). The model parameters were:$\chi^2 = 14.00$; $p < .001$. CONCLUSIONS: Prenatal alcohol exposure was not a risk factor for SIDS in this study. However, additional studies which can more closely examine dose-response relationships with prenatal alcohol exposure are warranted, since high dose exposure, as well as exposure during differrent periods of pregnancy, could not be excuded as SIDS risk factors by this study.

CHAPTER I

INTRODUCTION

The health status of a population can be assessed in several ways. Two common strategies include life expectancy and rates of infant mortality (Healthy People 2000, U.S. Government Printing Office, 1992). During 1985, 38% of infant deaths in Canada occurred during the first 24 hours of life, 17% between the first and seventh days of life, 10% in the period seven through 28 days, 27% in the period one through six months, and 8% of the deaths occurred after six months (Canadian Institute of Child Health, 1990). After the first month of life, sudden infant death syndrome (SIDS) was the leading cause of death in Canadian infants with a rate of 8.43 deaths per 10,000 live births (Canadian Institute of Child Health, 1990).

SIDS - The Leading Cause of Infant Mortality After 29 Days of Age

SIDS is the cause specific category for nearly half of the deaths occurring during the post neonatal period (29 to 365 days after birth) in Australia, Canada, England, Wales, Sweden and New Zealand (Mitchell, 1990). Kleinman and Kiely (1990) reported that while SIDS was the leading cause of post-neonatal mortality (PNM) for 1986, the rate varied two fold from 92.9 per 100,000 live births in Canada to 195 per 100,000 for England and Wales. The costs for medical care are low since most victims of SIDS are found dead and medical intervention is consequently limited.

The most widely used measure of preventability is the years of potential life lost (YPLL). In the U.S., YPLL due to SIDS ranked eighth among all causes in 1989. As comparative measures, congenital anomalies accounted for 660,346 YPLL and prematurity for 487,749 YPLL in 1989 or 3.9% of total all cause YPLL (MMWR, 1992), while for the same year, SIDS was the cause of 363,393 YPLL or 2.9% of total all cause YPLL (MMWR, 1992).

A series of conferences on definitional issues of SIDS have taken place since the first in 1969, with the most recent in 1992 (Willinger et al., 1991). In the 1969 conference the consensus definition of SIDS was: "the sudden death of any infant or young child which is unexpected by history and in which a thorough post-mortem examination fails to demonstrate an adequate cause for death" (Willinger, et al., 1991). This was the first of several conferences and publications which placed increased reliance on autopsies and the opinion of pathologists in the diagnosis of SIDS (Bass, et al., 1986; Bass, 1989; Smialek, et al., 1988; Valdes-Dapena, 1967). The principal contribution of pathologists to the diagnosis of SIDS has been to identify approximately 15% of cases of suspected SIDS as having other causes. Metabolic disease and filicide have been identified as two of the most common alternative causes of death as opposed to a SIDS diagnosis (Gilbert, et al., 1993).

The current consensus definition of SIDS is: "the sudden death of an infant under one year of age which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history" (Willinger, et. al., 1991). These deaths are nearly always at home or at the babysitter, occasionally while with the mother in a grocery store or in the doctor's

office or in an infant car seat. The death must be unexplained. The usual history is one where a previously healthy infant dies, and there is no known cause for the death. A diagnosis of SIDS is excluded in children who die suddenly with severe birth defects or metabolic disorders which are reported to be life-threatening or lethal.

Problem Statement

While the infant mortality rate (IMR) has declined in Canada and the rest of the developed nations in the past century, rates of SIDS have not declined as rapidly. After the first month of age, SIDS is the largest cause of infant deaths in Canada and in Manitoba. Since SIDS has a rather non-specific definition, that is, the sudden unexplained death of an infant, the search for a single underlying cause for this condition has not been successful. A lack of an agreed upon pathophysiologic basis for SIDS has resulted in an increasing focus on the identification of risk factors for SIDS. Epidemiologic research has been used to identify most of the currently known risk factors for SIDS. However, these risk factors have rarely been dramatic in magnitude and have often been associated with poverty, smoking and other general factors which are associated with poor health status for persons of all ages (Moffatt, et. al., 1988; Taylor & Sanderson, 1995).

In the literature, studies of alcohol as a risk factor for SIDS have received very limited attention. Alcohol may be important as a focus of study, since alcohol is a teratogen and has been suggested as a potential risk factor (DiNicola, 1985). While maternal alcohol use has been studied in several different racial populations (Scragg, et. al., 1993; Kandall & Gaines, 1991), similar studies have not been completed in other populations where SIDS rates are increased. The aboriginal people of Canada are an

example of this situation. In Canadian aboriginal infants, the rate of SIDS is 3.6 per 1,000 live births as compared to the SIDS rate in the non-aboriginal population of Canada of 1.1 per 1,000 live births (Canadian Institute of Child Health, 1990). In the province of Manitoba, treaty status Aboriginals comprise about 4% of the population. In 1990 Manitoba reported that 9.4% of children in the providence were Status Natives or classified as non-status Natives (Manitoba Pediatric Death Review Committee, 1990). In Manitoba 39% of the SIDS deaths are Status Native or non-status Native. Studies of maternal alcohol use as a risk factor for SIDS in the aboriginal populations of Manitoba, Canada have not been completed despite elevated SIDS rates in these populations.

Purpose

The purpose of this study was to examine maternal use of alcohol and to explore its potential role as a risk factor for SIDS. More specifically, the purpose of this study was also to determine if maternal alcohol use has a role in the elevated rates of SIDS among the aboriginal people of Manitoba. A case-control design was utilized to review the birth records of the cases and living controls. Cases and living controls were matched on race and smoking. Data on pregnancy and other maternal variables were obtained from the mothers' prenatal records. The province of Manitoba routinely collects data on maternal alcohol use as a part of the prenatal and newborn record. This study was also conducted to provide additional data on risk factors for SIDS in the Province of Manitoba which may be useful in a variety of Public Health activities.

CHAPTER II

REVIEW OF THE LITERATURE ON SUDDEN INFANT DEATH SYNDROME

The literature review will be targeted to the following topic areas: (a) historical trends in IMR; (b) distribution of infant deaths in the first year of life; (c) prevalence of IMR and postneonatal mortality; (d) prevalence of SIDS; (e) definitional issues affecting SIDS rates; (f) SIDS risk factors; (g) race as a risk factor; (h) classification systems using race as a variable; (i) prevalence of SIDS in aboriginals; (j) studies of SIDS risk factors in Canadian aboriginal populations; (k) infant mortality and maternal alcohol ingestion during pregnancy; (l) fetal alcohol syndrome; (m) maternal alcohol use during pregnancy; (n) alcohol as a SIDS risk factor; (o) alcohol as a SIDS risk factor - hypothesized mechanisms of action; and (p) a model of SIDS risk factors. The review of the literature will proceed according to the above sequence.

Historical Trends in IMR

Overall, IMR has declined worldwide for the past century (Pharoah & Morris, 1979). At the beginning of the twentieth century in the U.S., concerns were raised about working conditions, the condition of the milk supply, parental education, breast feeding, diarrhea, and enteritis as risk factors for infant deaths (American Association for Study and Prevention of Infant Mortality, 1911). In addition, concerns were raised about housing conditions, ventilation, overcrowding, and lack of appropriate prevention efforts

for diarrhea and pneumonia. Programs of public education to improve the outcome for live born infants were recommended (American Association for Study and Prevention of Infant Mortality, 1911).

During this time period, diarrhea and enteritis were by far the most important causes of death, being responsible for 29% of infant deaths. About 15% of deaths were attributable to premature births, and approximately 10% were due to birth defects. It was further noted that nearly half of the deaths from diarrhea and enteritis occurred in July and August. The dramatic changes in cause specific rates for infant mortality in North America can be observed by examining deaths in Canada. In 1921, 1300 children in Canada died from diarrhea, but by 1985 only seven deaths were reported due to the same cause (Canadian Institute of Child Health, 1990).

Reductions in IMR in the first half of the twentieth century were concentrated in the postneonatal period (Kleinman, et al., 1990). Since 1970 a rapid decline in neonatal mortality rates has led to an increase in the portion of infant deaths occurring during the postneonatal period. In the U.S. for the year 1987, deaths during the postneonatal period accounted for 36% of total IMR (Kleinman et al., 1990). In a cohort of 2,982 Canadian infants born in 1985, half of the infant deaths occurred during the first week of life (Canadian Institute of Child Health 1990). A very useful strategy for portraying IMR has been to compare rates during the neonatal and postneonatal period with total IMR as rates per thousand or rates per hundred thousand.

Distribution of Infant Deaths in the First Year of Life

For purposes of convenience, the first year of life of an infant is divided into three periods: (a) perinatal period, from 28 weeks gestation to seven days after birth; (b) neonatal period, birth through 28 days; and (c) postneonatal period, covering the period from 29 days to one year. Total infant mortality rates (IMR) are calculated by developing rates based on the number of live births in a calendar year (the denominator) and the identification of the number of deaths that occurred during that calendar year (the numerator), even though some children who die during the calendar year may have been born in another calendar year. Total IMR is then presented as the number of deaths for that calendar year over the number of live births occurring in that calendar year.

When IMR is calculated, the rates are considered incidence rates, since once death has occurred, the infant is no longer at risk for the outcome. The calculation of accurate IMR is primarily dependent upon a comprehensive system of capturing all births in a population or geographic area, and also upon the ability of the capture system to identify infant deaths. At the present time, two extremes for this situation are represented by the highly refined and elaborate public health surveillance systems present in developed nations as contrasted with a lack of governmental attention to this data in the poorest countries of the developing world.

Incidence of Infant Mortality and Postneonatal Mortality

Rates of PNM vary widely across countries (Mitchell, 1990). For example, during the period 1950 to 1985, PNM rates increased in New Zealand and Sweden and decreased in Australia, Canada, England and Wales. A comparison of PNM rates across countries

for the year 1986 ranged from 1.8 per 1,000 in Finland to 7.2 for Costa Rica (Kleinman, et al., 1990). Canada ranked seventh with a PNM rate of 2.8. The U.S. had an all races PNM rate that ranked seventeenth. White infants born in the U.S. were eleventh with a rate 3.1. However, black infants in the U.S. had a PNM rate of 6.3 for a rank of thirty-fifth, above only Bulgaria and Costa Rica (Kleinman, et al., 1990).

Since PNM rates are sensitive to changes in a range of social factors, wide variation in PNM rates would be expected not only across different countries but also within countries across the spectrum of different social groups in a country. SIDS was the leading cause of PNM with rates varying from 92.9 per 100,000 live births in Canada, to 195.4 per 100,000 in England, to 215.1 per 100,000 in the United States among African American infants (Kleinman, et al., 1990). In the age group 1-5 months, SIDS accounts for nearly half of the deaths (Mitchell, 1990), with over 80% of the total SIDS deaths occurring in this age group. Congenital anomalies were the second leading cause of PNM, causing from 15% to 26% of all PNM deaths. However, only 11% of U.S. African American infants died from congenital anomalies (Kleinman, et al., 1990).

Infant mortality rates are even more informative when presented as cause-specific rates for each reporting period. For the 2,982 Canadian children who died during 1985, birth defects were the leading cause-specific category of neonatal infant mortality, while SIDS was the most frequent cause-specific group contributing to PNM (Canadian Institute of Child Health 1990). While neonatal mortality rates are useful as indirect measures of the quality of obstetric and neonatal services, PNM rates provide a measure of social factors and, to a lesser degree, access to child health services in a population

(Mitchell, 1990). PNM is widely considered to be related to a variety of environmental factors which influence health, the most important of which is socioeconomic status (Arntzen, et al.,1995; Stockwell, et al., 1988; Mitchell, 1990).

Incidence Rates of SIDS

SIDS constitutes a higher percentage of post-neonatal deaths among American Indians (20 percent), than in white or black populations in the United States (14 percent each) (U.S. Department of Health and Human Services, 1986). In the U.S., Asians had the lowest rates of SIDS at 0.5 deaths per 1,000, and American Indians/Alaskan Natives the highest at 5.3 per 1,000 among Alaskan natives (U.S. Department of Health and Human Services, 1986). In American Indians, PNM comprised the majority of the deaths in the first year of life, while in other ethnic groups neonatal deaths predominated (U.S. Department of Health and Human Services, 1986).

Definitional Issues Affecting SIDS Rates.

In contrast to the definitional problems surrounding many outcome variables, death is a variable that can be widely agreed on. While a few exceptions exist, these are largely the result of modern technology or legal disputes, and rarely have a substantial influence on IMR.

The current definition of SIDS presents several problems. Taylor and Emery (1990) reported that only 17% of the deaths diagnosed as SIDS in Great Britain were completely unexplained. Dehan (1988) found only 7 of 45 SIDS deaths were unexplained. In contrast, deaths attributable to SIDS in Hong Kong are so rare as to be nearly

nonexistent. In the Netherlands, only half the infants with SIDS have had autopsies and in France only a third.

Recent publications have suggested that the term Sudden Unexplained Infant Death be substituted for SIDS so that as the diagnosis is refined and etiologic subgroups are identified, SIDS rates can remain comparable over time (Gilbert, et al., 1993). Indeed, for some time, more than the delineation of the syndrome of SIDS has been questioned. The whole concept of SIDS has been suggested to be "such a diagnostic dustbin". Emery (1989) suggested that the use of SIDS as a diagnosis is seductive for five reasons; (a) it enables doctors to tell parents that the child has died of natural causes which were not preventable, (b) it excuses all concerned with any defect in care, diagnosis or treatment, (c) pathologists welcome the diagnosis since the less they find, the more certain they can be of the diagnosis, (d) health authorities have no basis for prevention, and finally, (e) the diagnosis has facilitated the development of parent support groups and the raising of money for research.

Role of the Autopsy and Death Scene Investigation

The most recent definition expands the requirements for a diagnosis of SIDS to include a death scene investigation (Willinger, et al., 1991). The primary motivation for this inclusion of the death scene appears to be a series of papers by Bass and Smialek (Bass, et al., 1986; Bass, 1989; Smialek, et al., 1977; Smialek, et al., 1988; Meadow, 1990). Smialek (1988) has suggested that the diagnosis of SIDS depends on the analysis of information from two primary sources: the death scene investigation and the autopsy (Smialek, et al., 1988). A brief review of two papers by Smialek detailing death scene

investigations in 475 cases of infant deaths in Wayne County, Detroit may cast some doubt on the importance of these data sources in the diagnosis of SIDS. Of these 475 infants, 212 (45%) died of SIDS; 170 (38%) died of causes other than SIDS, 62 (11%) were accidental deaths, 14 (3%) were attributed to neglect or abuse, and 10 (less than 2%) were of undetermined cause. In addition, 16 infants were found to have died from accidental bed deaths, usually strangulation or suffocation.

Nowhere in these papers was the unique or essential contribution of the death scene investigation to the appropriate classification of the deaths described. Of the 16 bed deaths discussed, nearly all 16 of them would appear to have been overtly obvious to parents or ambulance personnel. For example, Case 1 described a five-month-old girl who was found unresponsive lying on a living room couch with her face covered by a plastic sheet; Case 10 was a seven-month-old infant who managed to get out of the crib because of a defective side rail and drowned in the bathroom; and Case 15 was a seven-month-old infant who crawled out of his crib and fell three stories to his death on the pavement below.

The role of death scene investigation in the classification of accidental deaths is also unclear since the majority of these would have been due to motor vehicle accidents with the children dying from massive trauma. Neglect or abuse constituted 3% of the infant deaths in this group. The autopsy and death scene investigation would appear to have been essential in the legal disposition of these cases of homocide, but many cases of neglect and abuse are apparent to emergency room personnel when an infant is brought in dead or dies in the emergency room.

Meadows (1990) reviewed 24 infant deaths due to suffocation and reported that the clinical course of the death and a confession by the mothers were the primary factors in determining the cause of death. In the absence of a confession by the mother, videotapes of a mother suffocating a child or eyewitnesses were essential. In all cases, the autopsies were unable to definitively determine a cause of death.

Regional variation in SIDS rates attributable to differing autopsy rates and wide variation in the prevalence rates for SIDS risk factors will explain some but not all of the variation in regional or national SIDS rates (Helweg-Larson, et al., 1992). The variation in SIDS rates accounted for by these differences are distributed across variations in postmortem protocols, the interpretation of the clinical course of the deaths, and the range of autopsy information available.

Changes in SIDS Rates Due to Diagnostic Transfer

Hoffmann, et al. (1992) have suggested that for SIDS rates to be interpreted appropriately they must be considered within the context of overall IMR, and more importantly, PNM rates. This is especially important, he stated, since more and more cases of what would have been termed classic SIDS deaths five or ten years ago are now being identified as "possible" or "probable" SIDS. As a result, the prevalence rates for SIDS in many areas are decreasing but the prevalence rates for sudden, unexplained postneonatal deaths may not be changing at a similar rate. Friede, et al. (1988) also cautioned that due to improvements in neonatal care, some infant deaths are now being postponed into the postneonatal period and that this influence should be considered when calculating IMR rates.

SIDS Risk Factors

In 1892, Templeman reported a study of 258 infants whom he described as having died of suffocation (Templeman, 1892). In his description of these 258 infants (most of whom would now be classified as having died of SIDS), Templeman noted a strong association with alcohol use, low socioeconomic status, and parents with lower levels of education. He also noted that peak death rates occurred between October and March and that infants were at greatest risk between one and six months of age. Ninety years later, Guntheroth reported the 10 most significant facts about the epidemiology of SIDS were: (a) infrequency of SIDS in the first month of life and after 6 months; (b) more common occurrence in winter; (c) more common occurrence in poor and non-white populations; (d) the mildness of symptoms that most victims had prior to death; (e) more common occurrence in unwed mothers, younger mothers, multiparous mothers with shorter interpregnancy intervals, cigarette smoking mothers, and mothers who utilized health care facilities less and later in pregnancy; (f) more common occurrence in premature and small-for-gestational-age infants, with most having slower than average growth after birth; (g) the frequency of unobserved deaths; (h) frequency of bottle feeding vs breast feeding; (i) familial recurrence of 1% to 2%; and (j) occurrence of SIDS which parallels the general rate of infant mortality (1982; 1989).

Smoking

A very durable finding over time in the SIDS literature has been the presence of smoking as a risk factor. One of the earliest case-control studies was done in Ontario which reported an odds ratio of 2.5 for infants of smoking mothers (Steele, et al., 1966).

Since then, numerous studies have implicated maternal cigarette smoking during pregnancy as a SIDS risk factor (Haglund, 1993; Malloy, et al., 1992; Mitchell, et al., 1993; Scragg, et al., 1993; Illing & Kaiserman, 1995). In a study of SIDS in Missouri, smoking was found to be a risk factor for SIDS, enhanced by a dose response relationship (Malloy, et al., 1992).

In a study of 15,285 infant deaths, which included 649 cases of SIDS, only low maternal education and smoking were found to be risk factors (Taylor & Sanderson, 1995). However, after controlling for smoking, low maternal education no longer emerged as a risk factor for SIDS. This finding was important since the sample size was sufficiently large to allow the authors to compare infants dying from SIDS with other infant deaths. In this study, the population attributable risk for SIDS from maternal smoking was found to be 30%. In Canada, the total smoking attributable mortality for SIDS has been reported at 69 infant deaths each year, and the smoking attributable YPLL before age 65 from SIDS was reported as 4,619 for the year 1991 (Illing & Kaiserman, 1995). Perhaps the largest and most comprehensive study of SIDS done to date was completed by the National Institute of Child Health and Human Development (Hoffman, et al. 1988). However, this study entitled "The SIDS Cooperative Epidemiologic Study," did not identify maternal smoking as a SIDS risk factor.

Exposure to Passive Smoking and SIDS Risk

In 1976, Bergman, et al. suggested that exposure to passive tobacco may be a risk factor for SIDS. Schoendorf and Kieley (1992) have recently provided additional evidence that exposure to passive tobacco does increase the risk of SIDS. Their data

suggested a doubling of risk for passive smoking, increasing to an odds ratio of 3 for both maternal smoking during pregnancy and for exposure to passive cigarette smoking. Mitchell, et al. (1993) reported that infants of mothers who smoked during pregnancy had a 4.09 greater risk of death than infants of mothers who did not smoke during pregnancy. The odds ratio of SIDS death was 2.4 if the father or other household members smoked, although smoking by the father increased the risk only if the mother smoked. Maternal smoking was also associated with an increased risk of SIDS, even if the mother never smoked in the house (Mitchell, et al., 1995). In a recent study of passive smoking, the odds ratios for SIDS were significantly increased by passive smoking among the infants' mothers, while the fathers' smoking resulted in an odds ratio of 3.46, and smoking by other "live in" adults resulted in increased risk of SIDS OR 2.18 (Klonoff-Cohen, et al., 1995). The authors of this study were able to control for birthweight, sleeping position, prenatal care, breastfeeding and maternal smoking status during pregnancy. These authors also found that the risk of SIDS increased with increasing numbers of cigarettes smoked. Lastly, in this study breastfeeding was found to be protective for SIDS in nonsmokers' infants but not in infants of smokers, where the odds ratio was increased by 1.38.

Social and Economic SIDS Risk Factors

In 1980, U.S. babies born to the least educated mothers were 2.3 times as likely to die as babies born to white, college graduates. The mortality risk ratio for babies born to black mothers with low education (below 12th grade) was 1.9, when compared to college-educated white mothers (Houge & Hargraves, 1993). In contrast, in Sweden only a 40% excess mortality rate among Swedish infants born to women with less than 10 years of

education, and a 20% excess mortality rate was found among women with 10 to 11 years of education as compared to women who had higher educational levels.

In Sweden, the only PNM cause-specific entity associated with significant social differences was SIDS. Nordstrom, et al. (1993) found a crude odds ratio for SIDS of 2.6 for mothers with less than 10 years of education and 1.9 for mothers with 10 to 11 years of education when compared with mothers with 15 years or more education.

Social-Cultural Influences

A review of the existing science base on SIDS is remarkable by the presence of two phenomena. The first is the very large number of case control studies using birth and death certificates to compare known risk factors for infants dying of SIDS. The second has been the inability of researchers to consistently identify a specific risk factor across time that is supported by a variety of studies (McKenna, et al., 1990). A study by Mitchell, et al. (1993) which suggested that bed-sharing was a risk factor for SIDS in the Maori population demonstrates the problem of separating biologic risk from risk due to ethnic or cultural influences.

McKenna, et al. (1990) noted that the earliest data on SIDS cases trace the origin of the disorder back to very early times when the deaths of children now thought due to SIDS was attributed to overlying (Savitt, 1979). A retrospective review of these case descriptions suggests that many of these children likely died of SIDS. A few technical comments about overlying may be useful. In most cases, no evidence of overlying will be present at autopsy. This is quite characteristic of a number of other possible etiologies for SIDS, including hyperthermia, hypothermia, intentional suffocation and the presence of

most drugs if the traditional autopsy is not extended to include toxicology (Harry Wilson, pediatric pathologist, Children's Hospital, Denver, personal communication). Overlying is usually considered as a possible cause of the infants' death when other etiologies have been excluded, and a history of bed sharing with an adult is available.

In one of the few papers from the medical anthropological literature on SIDS, McKenna, et al., (1986; 1990) made an interesting argument that the risk of SIDS, is in fact increased by not co-sleeping. They offered the hypothesis that throughout the evolution of humans, those infants who stayed in close proximity to their parents had an increased chance of survival. The authors based this hypothesis on the relatively long period of physical dependence exhibited by human children, coupled with the suggestion that infants who sleep alone lost the external sensory stimulation that may stabilize breathing during periods of neurological immaturity and change. McKenna, et al. reviewed a number of studies addressing the relationship of synchronous breathing and arousal patterns for parents and co-sleeping infants and then offered limited experimental evidence in support of their hypothesis that long periods of isolated sleeping are, in fact, important risk factors for SIDS.

The attribution of an infant death to overlying is usually made by persons distant from the scene, and most often without a death scene investigation. In fact, a death scene investigation would have to reconstruct the events at the time of death. An accurate reconstruction of these events would be very difficult to carry out, since overlying is believed to occur when the parents are asleep. These deaths can also occur when infants are in bed with adults who are intoxicated. While it is obvious that overlying can occur

and can result in death, it is very difficult in many circumstances to determine whether or not the infant died from overlying, or was already dead and was unable to signal the person overlying him by signs of a struggle (Mckenna, et al., 1990).

Berry and Paxton (1971) provided a survey of 90 societies and concluded that co-sleeping is practiced, or may be practiced in 71. However, data on SIDS rates were not available from these societies. Five studies do provide data to support the McKenna hypothesis described in the preceding paragraph (Mckenna 1986; Mckenna &Mosko 1990). Two of the studies were from Hong Kong where Davies (1984) reported that the incidence rate of SIDS was about 100 times lower than would be expected, occurring at a rate of .036 per thousand live births. This is even more interesting since he noted that the frequency of breast feeding in this population is quite low with only 24% of infants being breast fed at birth, and 9% at two months. In other studies, low rates of breast feeding have been thought to be an important risk factor for SIDS (Hoffman, et al., 1988).

Davies (1984) also noted that infants in Hong Kong live in very overcrowded conditions, with very high rates of respiratory infection, both of which have been implicated as possible causal risk factors for SIDS in a variety of other studies. He reported that co-sleeping is frequent but did not provide any rates on prevalence. In Hong Kong, parents virtually never raise the question of when a baby can be put into his own room. The low rates of SIDS in Hong Kong have been confirmed by a subsequent prevalence study (Lee, et al., 1989). Very low SIDS rates have also been reported in Japan, where co-sleeping is also common place (Tasaki, et al., 1988). In contrast,

Mitchell's study of the Maori (1990) suggested that bed-sharing and smoking were risk factors for SIDS in this population.

Gantley, et al. (1993) utilized a qualitative social anthropological approach to explore the quantitative and epidemiological evidence of ethnic and national variations in the incidence rate of SIDS. They extended the observations of Balarjan, et al. (1989) who identified significantly lower SIDS rates among babies born in England to mothers from Africa, Asia, India, Pakistan, or Bangladesh when compared with babies born to mothers from the United Kingdom or Ireland. The rates were highest among Afro-Caribbeans in Great Britain. Gantley, et al. (1993) noted that Bangladeshi infants had SIDS rates approximately half those of the Caucasian rates in Great Britain, and rates in Asians living in Birmingham, England were also low.

Interestingly, they noted that the low PNM rates in this group were not achieved by high rates of neonatal mortality. They reported that mothers with the lower SIDS rates lived in poorer socio-economic conditions, in crowded housing, and were young mothers with many children, all of which have been considered to be SIDS risk factors (Hoffman, et al., 1988). The Bangladeshi mothers, for example, had twice as many children as the comparable Caucasian group, however, Bangladeshi women were reported to smoke at low levels. The most interesting finding of this study was the practice of close sleeping of infants and parents. These infant care practices differed from the sleeping patterns practiced by the Welsh and English parents, where infants were placed on separate cots in their parents' rooms for a period of two or three months, and then were encouraged at the time of the peak age for SIDS, to get used to sleeping alone. Bangladeshi mothers also

seemed to have a lower prevalence of two other identified risk factors for SIDS, that is, the babies were typically put to bed in the supine position, and the mothers tended to monitor their infants closely for overheating.

While co-sleeping was not a risk factor in Gantley, et al.'s study, the concept serves to demonstrate the close relationship between biologic, cultural, and ethnic influences which may be associated with increased risk for infant death. In the following section, race as a risk factor for SIDS is reviewed. It is recognized that the variable race may include biologic, cultural and socioeconomic factors which interact to produce the variance measured by the variable race (Williams, et al., 1994).

Race as a SIDS Risk Factor

The rates of PNM due to SIDS in New Zealand were found to be 3.6 per 1000 in the non-Maori population and 7.4 per 1000 live births in the Maori population (Mitchell, et al., 1993). This finding suggested that race alone may be an important risk factor for SIDS. When Mitchell, et al. (1933) compared the prevalence of known individual risk factors for the Maori and non-Maori controls, the distributions of risk in the two groups was similar. However, the prevalence of several risk factors were much higher in Maori infants. The addition of multivariate analysis allowed Mitchell, et al. (1993) to control for the increased prevalence of these risk factors, and suggested that infants of Maori race had an increased risk ratio for SIDS of 1.37. However, the 95% confidence intervals ranged from 0.95 to 2.01, yielding insufficient evidence to confirm race as a risk factor after controlling for social and economic risk factors.

In the U.S., Canada, New Zealand and Australia, PNM has been found to be elevated in certain ethnic or racial groups. For example, in Canada the most frequent cause specific category of PNM is SIDS, which occurs in native infants three times more frequently than in non-natives (Moffatt, et al., 1988). A similar rate of disparity has been found in American Indian and African American infants in the U.S. when compared to SIDS rates among Caucasian infants in the U.S. (Bergman, et al., 1972; Peterson, 1984; Oyen, et al., 1990).

Classification Systems Using Race as a Variable

Birth and death certificates are the principal source of data used to calculate IMR. Several studies have been completed in the U.S. evaluating the extent of agreement between racial coding on birth and death certificates. In 1991, Nakamura, et al. reviewed deaths on the Warm Springs Indian Reservation in Oregon from 1940 to 1990. They found that while a dramatic decline in infant mortality had occurred, the IMR in the 1980s was five times the national average with nearly all the excess IMR attributable to much higher rates of SIDS. They noted that these rates are likely to underestimate infant mortality among the American Indian population in the U.S. due to problems with misclassification of race on death certificates.

Hahn and his colleagues (1992; 1992) reviewed inconsistencies in coding of race and ethnicity between birth and death in U.S. infants. They noted that inconsistencies in the coding of race is low for whites (error rate of 1.2%), higher for African Americans, (4.3%), and greatest for other races (43.2%). Most infants that were misclassified at death (87.3%) were classified as white. Misclassification occurred 46.9% of the time for

American Indian infants. After correction of these errors, the IMR for the period 1983 through 1985 for American Indians rose from 9.8 to 12.3 per 1000. If the new Center for Health Statistics algorithm (MMWR, 1992) were used which assigns the infant's mother's race to the infant at both birth and death, it is estimated that the IMR would increase to 14.4 per 1,000 live births. This would result in a 60% increase in IMR in this population.

In a ten year period, Reed and McBroom (1992) found 14,893 infants born in Montana who were classified as Indian on the birth record. During that same ten-year period, they found 210 infant deaths where race was coded as American Indian on the death record. The IMR for American Indian infants during this period was 14.1, however, in 12.5% of the cases where the dead infants were classified as Indian on the birth record, the infants' race had been misclassified (typically as white) on the death certificate. This served to substantially understate Indian IMR which, when corrected, rose to 15.6 per 1000, an increase of 10.6%. This finding was not an anomaly of a brief period but a pattern which was consistent over time .

Previous work by Frost and Shy (1980) indicated that the time of death during the first year of life is an important source of misclassification. They found that if death occurred during the first six days of life, the rate of misclassification was much higher than for infants who died after the first week of life. Reed, et al. (1992) also examined this problem, dividing the first year of life into three time periods: (a) the first 24 hours, (b) from day 2 through 28, and (c) after day 28 to day 365 (the post-neonatal period). They found that 17% of infants dying during the first 24 hours of life, were misclassified. For the period from the 2nd to the 28th day of life, misclassification dropped to 4% and then

again increased during the post-neonatal period to 16%. In some countries, such as Canada, data on racial group membership is not collected on either birth or death certificates. In Manitoba data on race was available from the Medical Examiner's Office (MEO) on deaths they had investigated.

Race would be a useful risk factor if the identification of an infant's race could be used to identify a risk attributable to race. However, Moffatt, et al. (1988) cautioned that while racial differences in SIDS rates are evident, social factors probably account for much of the differences in rates between groups.

Incidence of SIDS in Aboriginal People of Canada

In 1983, birth defects was ranked first as a cause of aboriginal infant deaths in Canada, with a rate of 4.2 per 1,000 live births. Birth defects was followed by sudden, unexplained deaths at 3.6 per 1,000 live births (Canadian Institute of Child Health, 1990). A comparison of Aboriginal infant mortality rates to total Canadian infant mortality rates has shown an excess rate of infant mortality among aboriginals (Canadian Institute of Child Health, 1990). Post-neonatal mortality rates among aboriginal infants are four times those of the overall Canadian population (Canadian Institute of Child Health, 1990). Among all Canadians in 1982, sudden, unexplained deaths occurred at a rate of 1.1 per 1,000 live births while in the native population, sudden unexplained deaths occurred at a rate of 3.6 per 1,000 live births in 1983 (Canadian Institute of Child Health, 1990).

In some areas this discrepancy in rates may be even more pronounced. In the Sioux Lookout Zone of Northwestern Ontario, SIDS accounted for 22 of 127 infant deaths (17.3%), and for 18 of 76 deaths (23.6%) in the post-neonatal period (Young,

1983). In Canada, PMR is considered to be sensitive to social, environmental, and socioeconomic factors (Canadian Institute of Child Health 1990; Moffatt, et al., 1988).

Incidence Rates of SIDS

Table 1 is a summary table of reported incidence rates for SIDS by race in the U.S. and Canada. These studies which span an 18 year period from 1972 to 1990, suggest large variations in SIDS rates between northern native and caucasian populations in the U.S. and Canada, with American Indians in the southwest having the lowest reported SIDS rates in the U.S. (Bulterys, 1990; Torrez, 1990). Kaplan, et al. (1984) found that the SIDS rate for American Indians and caucasians in Oklahoma was 2.32 compared to 1.8 for caucasians alone, suggesting a relative risk of 1.29 for Indian infants dying of SIDS through the years 1975 to 1981. In one study, native infants from Alberta dying of SIDS accounted for 13 percent of the SIDS deaths in that Province, while natives represented only 2 percent of the population (Wilson, 1990). The mean SIDS rates in the southern U.S. for the years 1984-1986 was 1.36 for American Indians from a population of 31,527 and 1.61 for whites from a population of 241,711. This yielded a native - white risk ratio of 0.8, with a 95 percent confidence interval of 0.6-1.2 (Bulterys, 1990).

During the same period, the SIDS rate for Indians living in all five of the northern Indian Health Service areas was 4.6 as compared to 2.1 for whites (Bulterys, 1990). American Indians living in North and South Dakota, Nebraska, and Iowa have the highest SIDS rates of any Indian Health Service area in the U.S. (Indian Health Service, 1990). Canadian natives living on reserves in Quebec, Ontario, Manitoba, Saskatchewan, and Alberta had a standardized mortality ratio of 3.6 for the years 1976 to 1983 (Morrison, et al., 1986).

Table 1

The Incidence Rates of SIDS Per 1,000 from Studies of Caucasians and Native Populations in the U.S. and Canada. (Source: Burd, et al., 1994).

Study	Location and Year	Rate per 1,000 live births: Caucasian	Rate per 1,000 live births: Indians
UNITED STATES			
	Northern United States		
Bergman et al.	Washington 1965-1970	2.04	8.03
Adams et al.	Alaska 1976-1980	2.14	6.28
Harrison	Alaska	-	2.17
Oyen et al.	North and South Dakota 1977-1984	1.50	5.70
Burd et al.	North Dakota 1977-1979	1.6	4.4
Hayward et al.	Wisconsin 1978-1987	1.4	6.7
Lum et al.	Alaska 1960-1980	-	1.7
Fleshman et al.	Alaska 1970-1975	-	2.7
	Southwest United States		
Bulterys	1984-1986		
	Albuquerque	1.75	0.47
	Navajo	1.45	1.22
	Phoenix	1.41	2.05
	Tucson	1.41	1.26

Table 1 (contnued)

The Incidence Rates of SIDS Per 1,000 from Studies of Caucasians and Native Populations in the U.S. and Canada. (Source: Burd, et al., 1994).

OTHER			
Black and Minority Health in U.S.	USA	-	5.3
Kraus et al.	California 1968	1.32	5.93
Kaplin et al.	Oklahoma 1975-1981	-	2.32
Henry et al.	North Carolina 1979-1982	1.33	2.88
Vandandingham et al.	Alaska, Arizona Montana, New Mexico North Dakota, South Dakota 1980	0.66	2.33
Blok	North Carolina 1972-1974	1.23	6.56
CANADA			
Young	Sioux Lookout Ontario	-	6.3
Moffatt et al.	Manitoba 1978-1984	1.21	3.74
Canadian Institute of Child Health	Canada 1982 & 1983	1.1	3.6

(1) Rates based on 1 death
(2) Assuming 350 births per year over 10 year period.

Studies of SIDS Risk Factors in Aboriginal Canadian Populations

A recent literature review of published data described risk factors for SIDS in Aboriginal populations (Burd, et al., 1994). In this review, risk factors for SIDS were divided into six categories: (a) maternal and paternal risk factors; (b) neonatal and newborn factors; (c) socioeconomic status; (d) familial clustering - genetic findings; (e) environmental factors; and (f) age of death, month of birth and seasonality. The following subsection of the literature review will follow these categories in sequence.

Maternal and Paternal Risk Factors

Lower levels of education for both mothers and fathers have been found to be significant risk factors for SIDS among native populations (Moffatt, et al., 1988). The relative risk for death from SIDS associated with this variable ranged from 1.7 for caucasians to 4.6 for Canadian natives. In North Dakota, significantly more American Indian fathers and mothers had less than a twelfth grade education ($p < .0003$) compared to caucasians (Burd, et al., 1994). Data for North and South Dakota combined found that for maternal education levels of 1 through 8 years, the relative risk for SIDS death was 3.7 for American Indians and 4.6 for caucasians (Burd, et al., 1994). For mothers with 9 through 12 years of education, the relative risk has been reported as 2.3 for American Indians, and 1.7 for caucasians (Oyen, et al., 1990). Maternal education and the trimester that prenatal care was begun were significantly related to SIDS among both natives and caucasians (Oyen, et al., 1990). In Canada 8 of 16 mothers of native SIDS infants had only an elementary school education (Moffatt, et al., 1988).

Parental age has also been reported as a risk factor for SIDS. American Indian fathers in North Dakota were younger than caucasian fathers ($p < .002$), and American Indian mothers were younger than Caucasian mothers ($p < .001$) (Burd, et al., 1994). When maternal age was under 20 years, the relative risk for American Indians to die from SIDS ranged from 1.38 to 1.4, and for caucasians from 1.5 to 3.4 (Oyen, 1990; Adams, 1985). In Canada, fourteen of forty-four mothers (32 percent) who had infants who died of SIDS were under 20 years of age (Moffatt et al., 1988). If maternal age was less than 20 years, it was found that the relative risk for SIDS was 3.4 for natives and 1.4 for caucasians. For mothers 20 to 24 years of age, the relative risk was found to be 1.0 for natives, and 1.5 for caucasians (Oyen, 1990). The trimester that prenatal care began accounted for a substantial part of the association between maternal education and SIDS rates among natives, but made little difference in the association between maternal education and SIDS in caucasians (Oyen, 1990).

The relative risk for late or no prenatal care ranged from 1.6 to 2.8 for natives, and 1.7 to 4.5 for caucasians in North and South Dakota. The later prenatal care was started the greater the relative risk for SIDS, with this association being particularly strong for caucasians (Oyen, 1990). The number of prenatal visits was also found to be a significant risk factor for SIDS. In North Dakota there was a significant difference in the number of prenatal visits between American Indians and caucasians, with native women having an average of 6.89 visits as compared to 8.93 for caucasian women ($p < .003$) (Oyen, 1990; Burd, et al., 1994). If a mother had 0 through 5 prenatal visits, the relative risk was 1.8 for American Indians and 2.3 for Caucasians (Oyen, 1990). For 6 through 10

prenatal care visits, the relative risk was 1.0 for Indians and 1.6 for Caucasians (Oyen, 1990). These findings were similar to those of Burd, et al. who found that American Indians initiated prenatal care significantly later than caucasians ($p < .014$) (1994). In Manitoba, it was reported that 24 of 34 native mothers (71 percent) had delayed prenatal care, however, only 2 had no prenatal care (Moffatt, 1988).

Bulterys investigated the role of chronic fetal hypoxia and maternal smoking in mothers of native infants dying of SIDS (Bulterys, et al., 1990). After adjustments for maternal age, infants born to mothers who smoked 10 or more cigarettes per day with hematocrits of less than 30 percent were at a much higher risk of SIDS (odds ratio of 4.0) than infants of mothers who did not fall into these categories (Bulterys, 1990; Bulterys 1993; Bulterys, et al., 1989). Li and Daling found that the risk from maternal smoking was no longer significant after controlling for race, birthweight, maternal age, and parity (1988).

Marital status has also been associated with SIDS, with a reported relative risk for unmarried native women of 1.56 and 2.84 for unmarried caucasians (Moffatt, et al., 1988). Native women had significantly more pregnancies prior to age 21 than whites, ($p < .030$) and were more likely to be unmarried. Also, infants dying of SIDS tended to be higher in the birth order for natives than caucasians, ($p < .02$). This may have been due to the increased number of previous births in the younger American Indian families (Burd, et al., 1994).

Oyen, et al. (1990) reported the following native/caucasian comparison risk ratios for SIDS deaths: (a) low birthweight 3.8; (b) maternal age 3.7; (c) maternal education

3.1; (d) for the trimester that care began 3.0; and (e) both maternal education and the trimester that care began 2.5. Another study, which did not examine SIDS deaths, reported higher rates of pregnancy complications among Sioux Indians, when compared to a population of caucasians in South Dakota (Peterson, et al., 1984). For example, this study reported that one out of 20 pregnant patients ingested alcohol at a level putting them at risk for having an infant with Fetal Alcohol Syndrome, and 47 percent of the women smoked. These authors also noted that nonreservation Indians had difficulty in accessing prenatal care, and because of their very low incomes, were dependent on funding from the welfare system for prenatal care (Peterson, et al., 1984).

Neonatal Risk Factors

A review of records for the perinatal and neonatal period found no significant differences between the native and white populations in terms of other neonatal risk factors (Bergman, et al., 1972). Apgar scores at 5 minutes were found to be 8.2 for native newborns, and 8.6 for caucasians, a non-significant difference (Burd, et al., 1994). Other variables that have been examined include: jaundice, chronic illness, persistent respiratory infection, probable cerebral palsy, and apneic episodes; no statistically significant differences were found between native and caucasian infants in North Dakota based on these variables.

Almost half of the infants who died of SIDS did have an upper respiratory infection in the two-week period prior to death (Burd, et al., 1994). It was also found that twelve percent of the mothers of SIDS infants in North Dakota had Caesarean-sections, however no significant differences were noted between native and caucasian women on

this variable (Burd, et al., 1994). In Manitoba, six of thirty mothers who had infants die of SIDS had had urinary tract infections during pregnancy (Moffatt et al., 1988). Feeding differences (breast or bottle fed) between ethnic groups have not been observed (Bergman, 1972).

Male gender, low birthweight, and the infant's age at death have been reported as having a strong association with SIDS among whites but these associations were weak or absent in the native population (Oyen, 1990). In Alaska, the peak probability of death occurred between 30 and 75 days of life for both population groups, and for the native population a second, smaller peak occurred between 120 and 134 days of age (Adams, 1985).

The male to female ratio for SIDS among American Indians in the Dakotas was reported as 1.0 to 0.9 and for Caucasians 1.8 to 1.1 (Oyen, 1990). In Wisconsin, the male to female ratios for SIDS deaths among Caucasians was 1.6 to 1 and for American Indians was 1.0 to 1.0 (Hayward, et al., 1990).

Among American Indians in the Dakotas, the relative risk for low birthweight (<2,500 grams) was reported at 1.9 and for Caucasians, 5.2. For low birthweight, the relative risk for SIDS was higher for Caucasians (5.32) than for Indians (4.32), and tests for statistical interactions between race and birthweight were not significant (Oyen, 1990). American Indians with birthweights under 2,500 grams were determined to have a relative risk of dying from SIDS of 4.23; while for Caucasians the risk was 5.32 (Oyen, 1990). Adams also noted that low birthweight alone explains less than 19 percent of post-neonatal deaths among the Indian population (1985). In the Manitoba study, the mean

birthweight was 3360 grams; only 3 of 41 babies weighed less than 2500 grams at birth; and only one had significant birth asphyxia (Moffatt, et al., 1988). Of the SIDS deaths in Manitoba, 9 percent of the deaths among natives occurred in infants weighing less than 2500 grams, as compared to 5 percent of the deaths among Caucasians.

The rates of both SIDS and apnea were reported as being more than twice as common in a native SIDS group than in a caucasian group (Davis, et al., 1988). However, apnea was considered to have a minimal effect on SIDS rates due to the very small number (less than 2 percent) of infants with apnea who later died of SIDS (Davis, et al., 1988).

Socioeconomic Factors

Adams observed that the overall socioeconomic status (SES) among the native population dying from SIDS is so low that many of these factors may have lost their discriminatory power (1985). When compared with caucasian families with a SIDS death, significantly more native families had no telephones available to them ($p < .01$) (Burd, et al., 1994). Many children who have died of SIDS have been reported to come from families with low socioeconomic status, and to not have received adequate health care (Bergman, et al., 1972). Moffatt, et al. have suggested that low SES factors were among the most important risk factors among Native SIDS deaths in Manitoba (1988).

Among infants dying in Winnipeg, Manitoba who were primarily non-native, there was a strong relationship between SIDS and economic levels (Moffatt, et al., 1988). There were 25 SIDS cases in the two lowest income groups among 15,174 infants at risk, while there was only one SIDS case in 4,116 infants at risk in the highest income level

(Moffatt,et al., 1988). These authors also reported that the SIDS rates for even the poorest non-native groups in Winnipeg were not as high as the rates among natives. At the time their study was conducted, native families had running water and waste disposal in less than 20 percent of their houses, unemployment ranged from 40 to 80 percent, and their education levels were lower. In a Wisconsin study of SIDS, 23 percent of the American Indians were found to be living below the federal definition of poverty, as compared to only six percent of the caucasian population (Hayward & Alessia, 1990).

Familial Clustering - Genetic Findings

Bergman, et al (1972) did not find differences between the family histories of American Indians and caucasians with a SIDS death. However, a 2 percent recurrence risk of SIDS in the families of SIDS infants was found in North Dakota, with 2 families having 3 SIDS deaths (Burd, et al., 1994). The small samples in these studies did not allow for significance testing for ethnic differences.

Environmental Risk Factors

One study that examined sleeping positions found no differences between races (Bergman, et al., 1972). Blok (1978) examined geographical distribution throughout the state of North Carolina by race and found that higher concentrations of non-white populations (American Indian and black) were associated with higher SIDS rates. There were five counties where the non-white SIDS rate was in excess of 10 per thousand; however, due to the small number of live births, this finding must be interpreted with caution. In the same study, the urban SIDS rates in North Carolina were higher for nonwhites for every city size, as compared to Adams' (1985) study which reported that

rural housing contributed a relative risk of 0.87 for SIDS deaths among American Indians and 1.36 for Caucasians.

Age of Death/Month of Birth/Seasonality

In the Dakotas the highest risk of SIDS was in American Indian infants born between August and February (Oyen, et al., 1990), while in Alaska the peak probability for death from SIDS was found to occur between 30 and 75 days of life. Among Alaskan Natives, a second smaller peak was also found to occur between 120 and 134 days of life (Adams, 1985). With the exceptions of SIDS occurring between 60 and 89 days of age, or after the first 120 days of life, native female infants were found to have a greater risk of SIDS than males (Adams, 1985). In contrast, Adams' study reported the reverse situation for Alaskan whites, and also observed a similar pattern of differences between native and white infants among the post-neonatal deaths in Alaska from other causes as well (1985).

In Oyen's study (1990), American Indian infants died of SIDS at a slightly younger age than whites; 36 percent of the Indian infants died by the end of their second month of life, but only 23 percent of caucasian deaths had occurred by the end of the eighth week.

In Adams work (1985), season of birth was not found to be a significant factor for either native or non-native infants. When season of death was examined (using spring as the reference season), the seasonal risk ratios for Indian infants were: summer (0.76), fall (0.78), and winter (0.79). For whites, the risk ratios were: summer (0.82), fall (1.04), and winter (1.22).

Autopsy Data

When comparisons of American Indian and white infants were made over a 10 year period in North Dakota, no differences between organ weights for heart, lung, brain or thymus were found when compared to normative values (Burd, et al., 1994).

The results of this review supported a 2 to 4 fold increase in SIDS rates found in aboriginal populations (Burd, et al., 1994). The risk factors described for SIDS do not appear to differ from the risk factors for most causes of post-neonatal mortality in these populations. While a few risk factors do differentiate Canadian aboriginals from caucasian populations, these primarily appear to be indicators of low socioeconomic status (Moffatt et al., 1988). This suggests that other variables, that are as yet unknown, could be important risk factors for SIDS.

The scope of SIDS, and the future implications of the infant mortality rate among aboriginal infants becomes more even more evident when the size and birth rate of the aboriginal population are examined. The birth rate for aboriginal women is twice that of Canada as a whole, with the highest birth rates found in Alberta, Manitoba, Northwest Territories, and Saskatchewan (Canadian Institute of Child Health, 1990). During the period 1982 through 1985, the birthrate for non-native Manitoba women was slightly more than 15 per thousand as compared to 32 per 1000 for native women (Canadian Institute of Child Health, 1990).

Infant Mortality and Maternal Alcohol Ingestion During Pregnancy

Since the first complete description of Fetal Alcohol Syndrome (FAS) by Jones and Smith (1973), alcohol has been recognized as an important etiologic factor for

childhood mortality and morbidity in the U.S. and Canada (Robinson, et al., 1987; Bray & Anderson, 1989; Abel, 1982; Abel, 1984; Abel, 1988). In his book on FAS, Abel reported on an early study of alcohol as a risk factor for infant mortality (1990). He cited the work of Sullivan (1899) who studied 120 female alcoholics who had been in a Liverpool prison. Sullivan studied the 600 children who were born to these alcoholic women, and found that 56% of their infants were either stillborn or died prior to 2 years of age. The infant mortality rate for the children of these women was more than 200 times the infant mortality rate of a control population who were blood relatives of the alcoholic women and who were married to sober husbands (cited in Abel, 1990).

In a study of FAS in West Germany, 2 children with FAS from an original sample of 72 had died at follow-up 4 years later (Spohr & Steinhausen, 1987). Olegard, et al. (1979) retrospectively studied all 52 children (25 girls and 27 boys) of 15 women who or had abused alcohol during pregnancy. They observed that in this population, the neonatal mortality was 5.8%, and infant mortality was 7.7%. In this group, one infant died of SIDS at three months of age. This suggested a death rate of 1.9% due to SIDS in a population of children of maternal alcoholics. These authors also observed that perinatal and infant mortality rates were increased 7 to 10-fold in this population, and found that 15% of the cerebral palsy in this population occurred in infants born to women who had used alcohol during pregnancy (Olegard, et al., 1979).

A 10-year follow-up study of children with FAS found that 18% of children had died, and that more than 27% of their mothers had died within ten years of the birth of the child diagnosed with FAS (Streissguth, et al., 1985; Streissguth, et al., 1991). While the

numbers of children in this study were small, the study does suggest that maternal alcohol use may be a risk factor for infant death. Prenatal exposure to alcohol has also been reported to increase the risk of spontaneous abortions and stillbirth. The risk of spontaneous abortions was found to be associated with a two to four-fold increase in women who consumed more than two ounces of absolute alcohol per week (Pietrantoni & Knuppel, 1991). In addition, these authors reported that excess rates of neural tube defects have been seen in populations of heavy drinkers.

Fetal Alcohol Syndrome

FAS has been identified as the most common identifiable cause of mental retardation and neurologic deficit in the United States and Western Europe, with prevalence rates exceeding Downs Syndrome (Trisomy 21) and Fragile X Syndrome (Abel & Sokol 1986; Abel & Sokol, 1987). It has been estimated that between six and eleven thousand children are born each year in the U.S. who have major or minor physical birth defects caused by prenatal alcohol exposure (Abel, 1982; Abel, 1984; Abel, 1988; Abel, 1990; Abel & Sokol, 1986; Abel & Sokol, 1987). Smart (1990) has also suggested that about 400 children with FAS would be expected to be born each year in Canada.

Previous prevalence estimates of FAS have varied 126 fold and ranged from 1.5 per 1,000 live births in the U.S., to 25 per 1,000 live births among chronically alcoholic women (Abel, 1982; Abel, 1984; Abel, 1988; Abel, 1990; Abel & Sokol, 1986; Abel & Sokol, 1987). Rates of FAS as high as 46.0 per 1,000 live births have been reported in some American Indian communities (Bray & Anderson, 1989; May, et al., 1983; Sandor, et al., 1981), which is about 20 times higher than the estimates of FAS in the general U.S.

population (Abel, 1982; Abel, 1984; Abel, 1988; Abel, 1990; Abel & Sokol, 1986; Abel & Sokol, 1987). Other research in the U.S. has suggested that FAS is much more common (up to 23 times) in the American Indian population compared with the caucasian population (Abel, 1988; May, et al., 1983).

In Canada, the highest reported rate of FAS and Fetal Alcohol effects in the world was based on a study of aboriginal children in British Columbia (Robinson, 1987). In this study, FAS rates as high as 190 per 1,000 in this community were estimated. Other reports have suggested elevated rates of FAS and Fetal Alcohol Effects in Canada of 46 per 1,000 live births in the Yukon, and 25 per 1,000 for children in Northwest British Columbia (Bray & Anderson, 1989). Sandor, et al. (1981) conducted a survey in two hospitals in Vancouver, British Columbia and found 69 aboriginal children and seven caucasian children with FAS, a 9.9 to 1 ratio. The elevated prevalence of FAS in aboriginal communities in Canada may present public health implications for the province of Manitoba. Manitoba has an aboriginal population of over 50,000 who also have one of the highest birth rates of any ethnic group, therefore, the potential exists for elevated FAS rates in this province as well.

Table 2 presents the results of a review of available FAS/FAE prevalence studies in 1994 from Burd and Moffatt, 1994.

Data from this review indicated that the rate of FAS among aboriginal peoples in the United States and Canada may be greatly increased as compared to caucasian rates. The ethnic differences between prevalence rates presented in table 2 are consistent across

Table 2. Summary data from the 8 studies of epidemiology of FAS in Indian or Native populations ages 0-18 years in U.S. and Canada up to November 1991. For Wong 1983, Winslow-Hill 1991, Duimstra, et al. 1993, and Bergeson, et al. 1993, denominator population is births but cases are identified over a several year period. N = information not available.

Study	Criteria	Total FAS & FAE in Study Population	Percent affected	Age Range	FAS		FAE		Rate per 1,000 Births	
					M	F	M	F	Prevalence	Incidence
May et al. * **	Developed for study by expert panel	115/243	47.3	0-14	41	35	26	13	FAS mean 2.0	FAS range ** 1.9 to 18.3 mean 2.8
Robinson et al.	RSA criteria	22/116	22.4	0-18	13	9	NA	NA	FAS 190/1,000	NA
Asante et al.	RSA criteria	166/391 Native children had FAS	Native 42.5%	0-16	113	63	NA	NA	Native Yukon FAS/FAE 46/1,000 NW British Columbia Native 25/1,000	NA
Christensen	NA	NA	NA	NA	NA	NA	NA	NA	From 1-1981 to 5-1986 FAS 5.1/1,000 FAE 1.7/1,000 6-1986 to 12-1988 FAS 2.7/1,000 FAE 1.7/1,000	NA
Chávez et al.	ICD-9	NA	NA	Newborns	NA	NA	NA	NA	NS	2.93
Burd et al. ***	RSA criteria	NA	NA	0-18	18	9	NA	NA	FAS 3.1 per 1,000	NA
Wong	ICD-9	NA	NA	Births	54	44	NA	NA	NS	6.6

Winslow-Hill	Rate includes FAS/FAE combined	NA	NA	NA	NA	NA	NA	NA	FAS/FAE 1987 0.53 1988 0.97 1989 2.73	NA
Duimstra et al. 1993	RSA criteria	4/24	17	under 2 years	NA	NA	NA	NA	Not stated.	Actual FAS 3.9%, best estimate > 8.5
Bergeson et al. 1993	RSA criteria	83/348	27	0-19	46	37	NA	NA	2.1 for 1978-1991	NA

* Adjusted by proportion of each cultural population in total area (May et al. 1983).

** Birth incidence (May et al. 1983).

*** For 34 children gender data was not available.

the studies and, even with different methods of ascertainment, the high rates among native peoples are evident.

Of great concern is a reported 25% recurrence risk for FAS in subsequent births (Abel, 1988; Abel & Sokol, 1986; Abel & Sokol, 1987; May, et al., 1983). The familial recurrence risk for FAS when compared to the general population risk is increased 350 times in the younger siblings of a child with FAS (Abel, 1988). This is presumably due to increased alcohol consumption as alcoholism worsens over time.

Maternal Alcohol Use During Pregnancy

The numbers of women of child-bearing age who drink alcohol is alarming. In the U.S. over 400,000 babies are born annually with exposure to opiates, stimulants, and illicit drugs (Kandall & Gaines, 1991). Of the U.S. population of 56 million women of child-bearing age, 34 million women drink alcohol, and 18 million smoke (Kandall & Gaines, 1991). It was reported that in the United States, 64% of non-pregnant women smoke and drink, and that among American women who smoke, 41% continue to drink throughout pregnancy. It has also been estimated that 65% of fetuses are exposed to alcohol prenatally (Serdula, et al., 1991).

In 1989, 72% of Canadian women classified themselves as having used alcohol in the last 12 months, with an average of consumption of 2.0 drinks per week (Kaplan, et al., 1991). In Manitoba during the year 1989, 73.6% of females reported drinking in the past 12 months, consuming an average 1.7 drinks per week (Kaplan, et al., 1991).

Alcohol as a SIDS Risk Factor

While several risk factors for increased SIDS rates in native populations have been examined, the identification of individual risk factors is far from complete (Canadian Institute of Child Health, 1990; Vanlandingham, et al., 1988). Several studies have investigated the role of maternal alcohol ingestion as a risk factor for SIDS in caucasians and African Americans (Hoffman, 1988; Southall, et al., 1987 ; Scragg, et al., 1993). These studies did not identify alcohol as a risk factor for SIDS in either whites or blacks. However, no published study has been identified that has examined alcohol as a risk factor for SIDS in any aboriginal populations where alcohol use rates during pregnancy are increased 5 to 10 fold (as indicated by FAS rates) (Bray & Anderson, 1989; May, et al., 1983; Sandor, et al., 1981).

Although not as yet systematically studied and reported, there are indications to support a hypothesis that alcohol may play an important role as a risk factor for SIDS. Havlicek, et al. (1977) reported one case study in which a Canadian infant who died of SIDS had a mother who was identified as a chronic alcoholic. Church, et al. (1986) reported in an abstract that an increased rate of SIDS was found in siblings of children with FAS.

Several investigators have hypothesized a relationship between maternal alcohol use and death from SIDS. In 1985, a pediatrician with Indian Health Service in the U.S. suggested that alcohol may be a risk factor for SIDS among American Indians (DiNicola, 1985). A review of charts of American Indian infants who had died in Alaska suggested that alcohol use after pregnancy may impair the caretakers of infants, and thus put the

infants at risk for deaths that may be misclassified as SIDS. These reports raise the possibility of a relationship between SIDS and maternal alcohol use during pregnancy; however, they are case reports only and consequently cannot suggest a systematic relationship with a defined magnitude of risk.

Alcohol as a SIDS Risk Factor - Hypothesized Mechanisms of Action

Risk factors for further study can be identified in several ways. One is by examining potential risk factors which have biological potential to cause or contribute to the outcome variable of interest. The potential for alcohol to increase morbidity and mortality in native populations is examined in this following section. A second important criteria in the selection of risk factors for study is an increased rate of exposure to a specified risk factor in a population where outcome event is elevated. The exposure of Native infants to alcohol has been established as being increased, and alcohol is known to have a range of effects which increases both mortality and morbidity in exposed populations.

It can be argued that alcohol abuse involves multiple mechanisms as a potential risk factor for infant death. One potential mechanism may be modification of the biological characteristics of the fetus during pregnancy, e.g., alteration of the respiratory drive centers in the brain stem which may predispose the infant to succumb to risk factors occurring later in the infant's life. A second hypothesis may be to suggest that low levels of alcohol transferred to infants after birth, through breast-feeding, alters the infants response to illness, especially respiratory infections. This mechanism may be sufficient to

cause a respiratory induced death in an infant who would otherwise not be vulnerable to the infection in the absence of alcohol.

A third mechanism, also post-birth, may be that the impairment of caretakers through alcohol ingestion substantially increases the risk of death in infants who are susceptible. Support for this mechanism has been suggested in the study results reported by Bass and Hass (1991). In a population of infants who had been classified as succumbing to SIDS, it was found that on the day of the infants' deaths, 30% of the caretakers were impaired by alcohol.

Alteration of Respiratory Drive

The exact pathophysiological mechanisms through which exposure to alcohol may increase the risk of SIDS is unknown. Southall, et al. (1987) examined electrocardiograms and abdominal breathing movements of the siblings of 78 infants who died of SIDS; in all, 170 control infants and 301 siblings of infants who died from SIDS were examined. It was found in this study, that the mothers of the siblings of the SIDS cases not only smoked more often during pregnancy ($p<0.01$), but also consumed alcohol more frequently during pregnancy ($p<0.05$). In this study siblings tended to have more short apneic pauses and higher respiratory rates than controls; however, these differences did not reach the statistical significance at $p<0.05$ (Southall, et al., 1987).

Since most infants die during periods of sleep, the work of Steinschneider is important (1972). He found that normal infants at two to three months of age tend to have apneic periods during sleep, and that these episodes are prolonged during periods of upper respiratory infection. Upper respiratory infection is a common finding in infants

succumbing to SIDS. However, it is as yet unknown if upper respiratory infection would interact with the normal developmental patterns of apneic episodes occurring during the period of risk from two to four months of age (Steinschneider, 1972).

Both structural and functional defecits in the brain have been found after prenatal alcohol exposure (West, 1986; West, 1993). West (1986) summarized the three studies evaluating the functional effects of alcohol on fetal respiration in humans. Two studies demonstrated that 40 ml of beverage alcohol (vodka) supressed fetal breathing movements (Fox et al 1978 and Lewis and Boylan 1978). A subsquent study found total supression of fetal breathing activity for three and one-half hours by a 0.25 gram dose of ethanol per kilogram of maternal body weight (Mcleod et al 1983). While these effects were transient the studies do demonstrate the potent effects of ethanol exposure for even for a brief time period. West noted that many women consume alcohol over long periods of pregnancy and that the long terms effects of alcohol on fetal development were unknown (West 1986). While not specific to alcohol exposure, research on alteration of respiratory centers during pregnancy in infants dying of SIDS has been extended by (Kinney et al., 1992). They presented a review of the neuropathology of SIDS and suggested that abnormalities in the brain stem and specifically those regions that control respiration and sleep cycles are damaged in utero and may combine with a critical developmental period to result in SIDS. In a subsequent paper they have presented data to suggest that abnormalities in these critical regions are present in many infants dying of SIDS (Kinney et al., 1995). While evidence of specific evidence of damage to these centers by prenatal alcohol exposure is lacking, maternal alcohol use during pregnancy has been demonstrated to cause a wide

range of damage throughout the central nervous system (West et al., 1994; Bonthius and West, 1991). Kinney et al. (1995) also suggests that there may not be a single cause for SIDS but that several factors factors may interact to greatly enhance the risk for SIDS especially during critical periods of development.

Post-Birth Exposures: Transfer through Breastfeeding/ Impaired Caregivers .

Maternal alcohol ingestion could also be a risk factor for infants who are exposed after birth. Alcohol crosses freely into breast milk, reaching levels that are nearly equivalent with maternal serum levels (Anonymous, 1983). It may be hypothesized that some infants may be exposed to levels of alcohol sufficient to interact with the development of apnea and/or upper respiratory symptoms that trigger a sequence of apneic episodes, followed by decreased respiratory drive, and leading to a fatal episode of hypoxia. It is further proposed that continued alcohol use after birth can result in the impairment of caretakers.

Smoking: A Potential Confounder

Smoking during pregnancy is an important risk factor for SIDS, and may represent a confounder for alcohol as a risk factor. Several studies have examined smoking as a risk factor for SIDS, with risk ratios ranging from 1.6 to 4.1 (Kraus & Borhami, 1972; Drews, et al., 1990). In populations of women who drink, smoking is common, and it has been found that drinking and smoking during pregnancy are also common (Strychar, et al., 1990; Serdula, et al., 1991; Kesby, et al., 1991; Heller, et al., 1981; Waterson, et al., 1990). In one study that examined smoking rates in a population of 162 women from

different racial groups in the Northwest Territories, it was reported that 88 of 154 women (57%) smoked during their pregnancy (Godel, et al., 1992).

Bulterys (1990) reported that the increased rates of smoking may explain part of the increased rate of SIDS among northern American Indians and Alaska Natives in the U.S. However, the nature of this relationship is not established, especially given Li and Daling's study (1991) which demonstrated that the relationship between SIDS and maternal smoking was not significant after controlling for race and birthweight in Indian infants from Washington state.

Alcohol Use and Smoking Prior to and During Pregnancy

Alcohol use prior to and during pregnancy may have been overlooked as a risk factor since the close relationship between alcohol use and smoking, may have confounded the detrimental effects of alcohol. While the rates of both smoking and drinking decline during pregnancy, smoking rates have been found to decline only about one-half as much as drinking during pregnancy (Kesby, et al., 1991). This relationship has been reported in pregnant women (Bonati and Fellin 1991; Bolumar et al., 1994). Bonati and Fellin (1991) reported that when pregnancy was confirmed 12% of women stopped smoking and 6% stopped drinking. In the Bolumar study 60% of women smoked and 72% drank. Forty-eight percent of the smokers quit during pregnancy and 37% of the drinkers quit drinking during pregnancy (Bolumar et al., 1994).

In a study of 128 primigravida women in British Columbia, 75% reported having consumed alcohol before they knew they were pregnant and 39% smoked (Kaplan, et al., 1991). In another group of women who found out they were pregnant, 82% reduced their

alcohol consumption, and smokers reduced their cigarette smoking by an average of 52% (Strychar, et al., 1990). In this study, 16% had drunk heavily (more than 4 drinks on one occasion and more than 45 drinks in the month prior to becoming pregnant) before they knew they were pregnant (Strychar, et al., 1990). In a 1993 survey of alcohol use in Canada 74.4% of Canadians reported drinking alcohol in the past year (Single et al., 1995). In this survey 68.4% of women identified themselves as current drinkers. Women reported that they drank an average 2-3 drinks per week. In this sample 33.1% of women consumed 5 or more drinks on one occasion and 8.4 percent of women were categorized as heavy alcohol users. Unmarried women used more alcohol than married women. The Prairie provinces had the lowest levels of alcohol consumption 3.5 drinks per week compared with Quebec at 4.5 and Ontario at 4.4 drinks per week. A study of alcohol use among White, Inuvik, Mixed Race and Eskimo women in the Inuvik Zone of the Northwest Territories indicated that 50 of 145 women (34%) drank during their pregnancy (Godel, et al., 1992). Due to the increased rates of smoking in Native women compared to Caucasians the potential for confounding may be increased in Native populations.

Summary

The absence of studies examining the role of alcohol as a risk factor in contributing to the 2 to 4 fold increase in SIDS rates in aboriginal populations represents a major gap in the science base related to risk factors for SIDS (Canadian Institute of Child Health, 1990; Vanlandingham, et al., 1988). The variable of alcohol begs a systematic inquiry into its potential role as a risk factor for SIDS, and may yield useful information towards the

reduction of post-neonatal mortality in both aboriginal and caucasian communities. It is suggested here, that in trying to establish the possible role of alcohol in SIDS, multiple risk factors must be studied for their potential relationships/interactions with alcohol.

Towards this end, a multifactorial model of risk factors for SIDS is presented in Figure 1. Further study is required to answer the question: Can maternal alcohol use during pregnancy increase the risk for SIDS? The study presented here represents an effort to address this question.

A Model of Risk Factors for SIDS

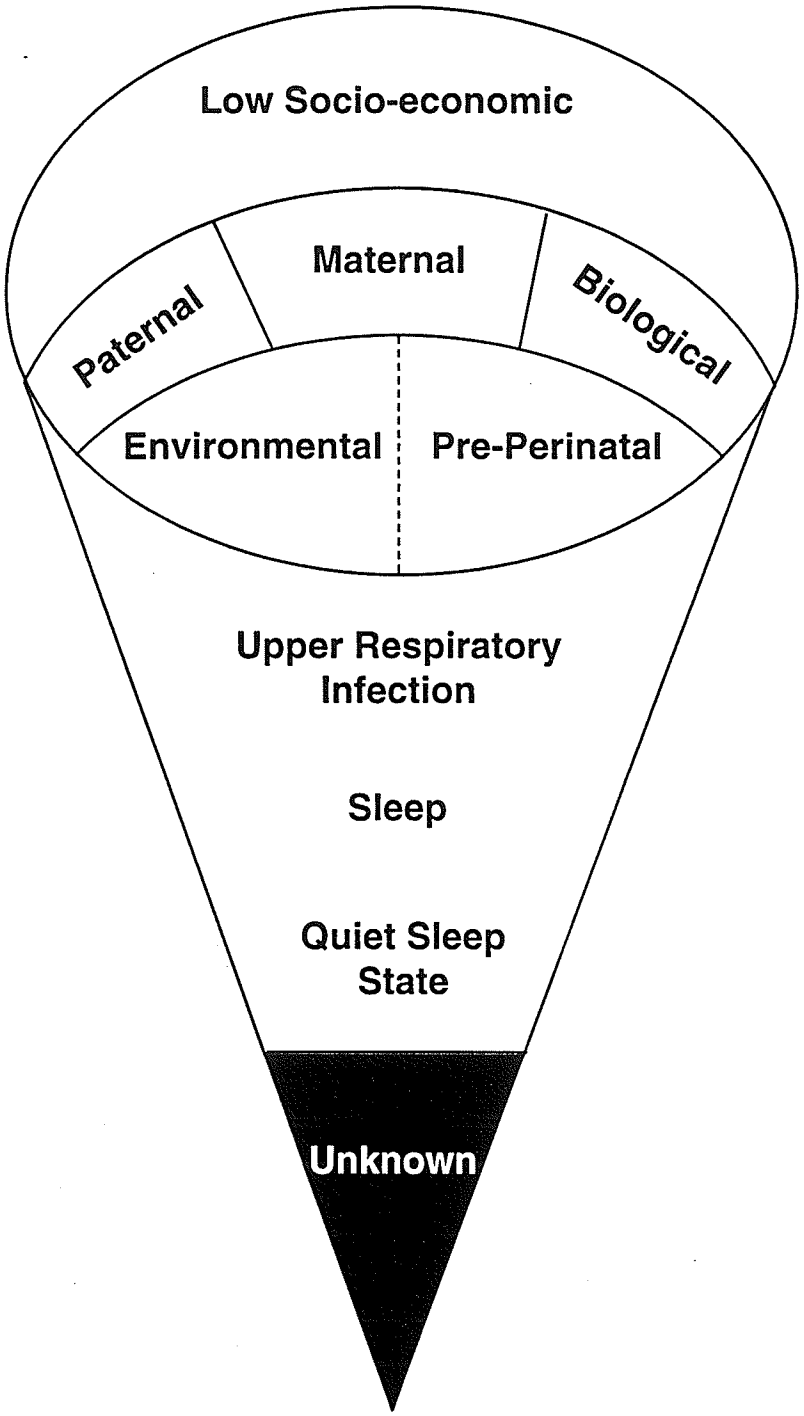
The model presented in Figure 1 was developed to demonstrate that many infants have any given risk factor and that no one risk factor is essential for SIDS to occur. The tip of the funnel indicates that very little is known about the mechanism of death in these infants. This model was not meant to be exhaustive and primarily utilized risk factors relevant to this study.

Given the role of SIDS as a cause specific category of infant death, innovative approaches need to be utilized to develop prevention strategies to reduce the incidence of SIDS. One strategy is the identification of risk factors that are amenable to prevention efforts. Several significant advances have been made in identifying risk factors for SIDS (Guntheroth, 1982; Guntheroth, 1989; Guntheroth and Spiers, 1990 and 1992). Sleeping position has been identified as a risk factor for SIDS and may have potential as a focus for prevention efforts (Fleming et al., 1990). The prone sleeping position is associated with a relative risk of between 3 and 5 compared to a supine sleeping position (Fleming, 1990).

Figure 1

Risk Factors for SIDS

A model depicting the interaction of SIDS risk factors. In this model many infants are exposed to 1 or more risk factors for SIDS. The only common factor in SIDS appears to be death during sleep. The unknown portion of the funnel denotes our lack of knowledge about the essential risk factors which nearly always result in SIDS.



Other risk factors for SIDS which may be useful in prevention are the type of and amount of clothing that the child was wearing at the time of death and either a lack of or early cessation of breast feeding (Guntheroth, 1982; Guntheroth, 1989). Other consistent risk factors are lack of prenatal care, short interpregnancy interval, maternal smoking, lower rates of maternal education, and low birthweight (Guntheroth, 1982; Guntheroth, 1989; Hoffman, 1988).

In order to control for the possible confounding effects of smoking on alcohol, cases and controls were matched on the variable of smoking. The matching utilized 2 groups: smokers and non-smokers during pregnancy. A more discrete match was not possible due to the limitations of data collected in medical charts. This approach removed smoking as a possible confounder of the association of maternal alcohol use of SIDS.

CHAPTER III

METHODOLOGY

Design

A case control study design was chosen to determine if maternal alcohol use is a risk factor for SIDS among the offspring of women who have used alcohol during pregnancy. Existing medical records were utilized to do two separate sets of comparisons: (a) comparison of cases (infants who had died of SIDS) to a control group of living infants (infants currently alive, who had not died of SIDS); and (b) comparison of cases (infants who had died of SIDS) to a control group of dead infants (infants who had died of causes other than SIDS). The cases and controls were matched on the following variables: (a) hospital of birth, (b) year of birth, (c) race, and (d) maternal smoking status during pregnancy.

Hypotheses

There were three null hypotheses to be tested in this study of infants who have died of SIDS:

1. Infants dying from SIDS will not be more likely (.05) to have fetal alcohol exposure as determined by maternal drinking history than living infant controls matched for age, hospital of birth, race and maternal smoking status.

2. White infants dying from SIDS will not be more likely (.05) to have fetal alcohol exposure as determined by maternal drinking history than living infant controls matched for age, hospital of birth, race and maternal smoking status.

3. Aboriginal infants dying from SIDS will not be more likely (.05) to have fetal alcohol exposure as determined by maternal drinking history than living infant controls matched for age, hospital of birth, race and maternal smoking status.

4. Infants who have been exposed to maternal alcohol use during pregnancy will not have a statistically significant increase in risk for SIDS ($p<.05$) when compared to dead (non-SIDS) infant controls.

If alcohol emerged as a significant risk factor in groups one through three the specificity of alcohol as a risk factor would be examined with hypothesis four.

Subject Selection

The Pediatric Death Review Committee (PDRC) functions under the auspices of the Manitoba College of Physicians and Surgeons and currently reviews all childhood deaths in Manitoba. At the time this study was undertaken, the PDRC was comprised of one general practitioner, seven pediatricians and a pediatric pathologist who review autopsy results. In Manitoba, all childhood deaths are reported to the Medical Examiner's Office (MEO) by law. Each death is reviewed both by the MEO and the PDRC. The PDRC reviews the information on infants who die, and uses criteria to classify deaths as SIDS or non-SIDS deaths.

During the period of time covered by this study, the criteria used by the PDRC and the MEO were from Guntheroth (1982; 1989). The criteria for SIDS were established in

1969 as the sudden or unexpected death of an infant who has seemed well; the death remains unexplained after performance of an adequate autopsy (Bergman, 1972). This was the definition of SIDS suggested in the 1969 Consensus Conference (Willinger, et al., 1991).

During the period of time covered by this study, the MEO assigned a cause of death after reviewing all available information, including the results of the PDRC. In this study, the cases were infant deaths classified as SIDS by either of these sources. The average number of deaths from SIDS in Manitoba is about 20 per year (Moffatt, et al., 1988).

Selection of Cases, Controls and Non-SIDS Deaths

A list of SIDS cases for the years 1986 through 1990 in the Province of Manitoba were requested from the MEO of Manitoba and the PDRC. A list of childhood deaths was obtained from the Manitoba Division of Vital Records. All three lists were then reviewed to ensure that all cases in this study met the inclusion criteria.

From the PDRC, 295 infant deaths were identified. The data from the PDRC for each individual case review was obtained, with the exception of data from 1986 which had been discarded and was not available. A similar process was used to review the lists from the MEO. The MEO, during the period 1986 through 1990, classified the SIDS deaths independently. As a result there were cases where disagreement regarding a diagnosis of SIDS existed between the PDRC and the MEO. In the data entry form, three columns were used for this data, one indicating status as SIDS or non-SIDS death for the PDRC, one for the MEO, and third, if both agreed.

The three data sources produced a list of 324 deaths. From this list, 29 deaths were excluded for not meeting case inclusion criteria, such as, the infant was not born in Manitoba (no records would be available), if the child was not a Manitoba resident, or if an infant was under 14 days or over 365 days of age. The three lists were then consolidated to avoid duplication, and a single final list was produced. This produced a final list of 282 infants. The exact number of deaths differs since some non-Manitoba births are included.

Inclusion Criteria: Cases

Cases for this study were included based on the following criteria: (a) infants whose deaths had been reviewed by the Manitoba PDRC or the MEO; (b) the deaths had been classified as SIDS by the Manitoba PDRC and/or the MEO; (c) the infants were born in Manitoba and were Manitoba residents at the time of their deaths; (d) the infants had died between 14 and 365 days after birth; and (e) the infant deaths occurred during the years 1986 to 1990.

Exclusion Criteria: Cases

Children who died within the first two weeks of life, or after 365 days of age were excluded as cases. The overall incidence of SIDS in the first month of life is about 5% of all SIDS deaths, and far less than half of these deaths would be expected to occur during the first 14 days of life. Infants who were not Manitoba residents at the time of their death were excluded. Infants whose medical records did not include information on maternal smoking or alcohol use were also necessarily excluded.

Inclusion Criteria: Living Controls

For each case, one living control was selected from the birth records of the same hospital as the SIDS case. As stated above, the controls were matched on hospital of birth, year of birth, race, and exposure status to maternal smoking during pregnancy. Hospital birth records were utilized to select living controls. Infants chosen as controls in this study were those of the same race, who were born closest to the time immediately after the birth of the case infant. If the medical chart for a control was unavailable, another control was selected in the same manner, selecting the next appropriate infant from the birth registry of the hospital. Only controls with smoking and alcohol information were included. This matching plan was selected to reduce the potential confounding from smoking.

Dead Infant Controls (non-SIDS deaths)

A second comparison group consisted of unmatched infants whose deaths occurred after 2 weeks of age and before 365 days of age, but whose deaths were not classified as SIDS. The inclusion of these non-SIDS deaths was used to determine if maternal alcohol use during pregnancy increased the risk for infant death in general, or if the risk is specific to SIDS. These infants were included if they were not classified as SIDS by the PDRC or the MEO. These infants also needed to meet all of the criteria for cases, with the exception of not being classified as SIDS.

Exclusion Criteria: Controls

Infants used as controls were excluded if they could not be assigned to an ethnic group. Controls whose charts did not contain the risk factor information about alcohol use from Appendix 2 were also necessarily excluded.

Confidentiality

The records used for data collection have been kept completely confidential. In order to maintain this trust, all cases and controls were assigned a 3 digit ID number. Only the ID number was used for data stored in the computer file and during data analysis. The records are stored in a locked room.

Matching Decisions

Controlling for **race** through matching was considered to be important since previous studies had demonstrated differential distributions of SIDS risk factors in different populations. **Race** had also been used as a matching variable in previous studies to reduce the variance associated with: (a) socioeconomic status (Moffatt, et al., 1988; Hayward, et al., 1990; Burd, et al., 1994), (b) cultural differences (McKenna, 1990), and (c) differing rates of alcohol use (Bray & Anderson, 1989; May, et al., 1983; Robinson, 1987; Burd & Moffatt, 1994).

It was also considered important to control for maternal **smoking** status as reported in previous studies, since varied rates of smoking among people of different races was also considered a potential source of confounding in this study (Malloy, et al., 1992; Schoendorf & Kieley, 1992). Matching on **year of birth** in the present study was used to reduce the variance associated with changes in medical care over the five year period of

the subjects' births and deaths. **Hospital of birth** was matched to reduce the potential variance both in medical care (rural versus urban), and to make the subjects and controls as comparable as possible on socioeconomic status. The variables which were matched with cases when selecting controls could not be analyzed as risk factors. This results in some loss of information and the potential to introduce some additional confounding. This decision was carefully weighed and thought to be a useful plan at this stage of research on SIDS in this population.

Assignment of Race

All children who are reported to the PDRC and MEO are carefully investigated for a variety of factors that may have influenced the manner of their death, and which may be useful for future prevention efforts. In addition, all children are classified by race by the MEO in order to determine if death rates, or causes of deaths, differ between races in Manitoba. For the MEO, this classification is based on physical examination of the body and on identification of treaty status. This method of classification was therefore used to determine the race of the SIDS cases in this study. Treaty numbers from the medical records were also used in the determination of race for the living controls. The potential of some error in assignment of race could occur and would effect the identification of race specific risk factors. However, no other data on race was available.

Sources of Data for Risk Factors

All data related to risk factors being investigated in this study were excerpted from the following record sources: (a) infants' medical records, (b) MEO records, (c) PDRC charts, and (d) medical records of the infants' mothers. The two primary sources of data

within the medical charts was the Maternal Nursing Database (see Appendix 2) and the Antepartum Risk Pregnancy Scoring Form (see Appendix 3). The Maternal Nursing Database is completed by the nursing staff at the hospital of birth.

Exposure Status

One area of concern was that the primary independent study variable, **alcohol**, may not adequately discriminate between the exposed and unexposed groups due to measurement bias (Sitthi-amorn & Poshyachinda, 1993). In this scenario, confounding could occur due to incorrect categorization of women on exposure status. Mothers of cases may have had very limited exposure, for example, women who drank 3 or 4 drinks in the first month of pregnancy may have reported that they used alcohol, although it would not have been an ongoing pattern. The dose would have been too low to cause the SIDS effect.

In a similar way, controls could have had the same, or slightly higher amounts of exposure to alcohol, but their mothers may have reported that they did not drink during pregnancy. For example, the level at which some mothers had consumed alcohol may have been, in their opinion, equivalent to not "really" drinking. In this scenario the measurement characteristics of the variable would not be sensitive enough to classify the exposure status of either the cases and controls, and a type II error could have been

introduced. In order to examine these possibilities, a pilot study to assess the reliability of the data sources (completed prior to the data collection phase) will be reviewed.

Pilot Study Regarding Reliability of the Data Sources

The data on alcohol use during pregnancy for this study was collected from maternal medical records. In order to verify the accuracy of the information on alcohol use reporting a pilot study was completed. The purpose of this pilot study was to determine the reliability of women's self-reports of alcohol use during pregnancy.

The application to do the pilot study was reviewed and approved by the University of Manitoba Research Committee and by the Health Sciences Center Ethics Committee.

The data collection form and the consent form were then given approval by the following: Health Sciences Center Research Committee and Ethics Committee, nursing staff at the Women's Center, nursing staff and Institutional Review Board and Ethics Committee at St. Boniface Hospital, and Director of Nursing at the Prenatal Clinic located at the Health Sciences Center.

Three sites were selected: The Women's Hospital at the Health Sciences Center, the Outpatient Prenatal Clinic at Women's Hospital, and the Obstetrics Department at St. Boniface Hospital. In a one month period, five separate visits were made to these sites to collect data. The methodology of subject selection varied slightly by site. At the Prenatal Clinic at the Woman's Hospital each individual woman was asked by the nursing staff if they were willing to participate, and over the course of two one-half day visits two women did participate from this site. This represented less than 10% of the women who actually attended the Prenatal Clinic during that period of time.

On the two obstetric wings of the Women's Hospital, each Director of Nursing would review the charts to identify those women who were not appropriate candidates for this study (for example, women who heavily medicated, women who had emotional problems severe enough to warrant their exclusion, or women who had delivered so recently that the staff felt it would be advisable not to disturb them). These women were excluded from the study.

From the remaining patient list, the interviewer then would go to the individual rooms, describe the study briefly, and invite the women to participate. Participation rates for those women who could be contacted were very high, in excess of 90%. However, due to the mobility of this population (women visiting family, going outside to smoke, etc.) it was not possible to contact all potential subjects. Two-thirds of the women on the final list approved by the Director of Nursing in the two wings were eventually contacted. After consent was obtained, these women were interviewed, or alternatively, if they wished, the form was given to them to read. Where they had questions, a brief discussion session was utilized to formalize their responses.

At the St. Boniface Hospital, the Director of Nursing accompanied the researcher to each individual room and women were asked if they would like to participate in the study. Again in this setting, the response rate was very high and this system of subject selection proved to be most efficient from the standpoint of the interviewer. At all three sites the women were interviewed, and their medical charts were then reviewed.

Results of the Pilot Study

Forty-one women participated in the pilot study to assess reliability and validity of the data source. Of these 45 women, data from the chart was available for 38. While information from the interview was available as to quantity of alcohol use, comparable information was not available in the chart, where alcohol use is nearly always listed as either a "yes" or "no" variable. Of the women interviewed, 12 of 41 (26%) reported using alcohol during pregnancy. However, when the charts were reviewed, 7 women were identified by the data in their medical chart to have used alcohol during pregnancy. None of the three women with the missing maternal data bases reported using alcohol at the interview.

Five women had inconsistent reports about alcohol use during pregnancy. The inconsistency appeared to be primarily related to varied interpretation of having consumed a minimal amount of alcohol very early in pregnancy. For example, one woman noted "I had three drinks at my birthday party a week before I found out I was pregnant." Two other women reported drinking modest amounts at other functions early in pregnancy. However, no woman was identified who drank substantial amounts of alcohol and was subsequently misclassified. All women who reported substantial amounts of alcohol use in the early part or throughout pregnancy had consistent reports at both times.

Outcome of Pilot Study

Three possible outcomes of the pilot study were analyzed: (a) cases where the interview had identified women who reported drinking activity, but who were not recorded as alcohol users in the chart (n = 5); (b) cases where the chart identified women

who were drinking but who were not identified at interview (n = 0); and (c) cases where the interview and chart shared agreement on the women's drinking status (n = 33). For outcome one a Kappa was calculated and was .74. For outcome three a correlation coefficient was computed and was 0.699.

Conclusions Regarding Reliability and Validity of Data Source

Due to the substantial agreement (kappa of .74 and a correlation cofficient of 0.699) found between the interview and the chart data sources, the reliability and validity of the data concerning maternal alcohol use during pregnancy was considered to be adequate for use as the independent variable in this study. The interview approach appears to be effective in identifying women who had ingested "moderate" to "high" amounts of alcohol during pregnancy; however, the interview may not have been as sensitive to women's drinking very minimal amounts of alcohol early in pregnancy.

Sample Size and Power Calculations

Schlesselman (1982) noted that sample calculations for case-control studies depend on 4 values: (a) the relative frequency of exposure among controls in target population; (b) a hypothesized relative risk associated with exposure that would have sufficient biological or public health importance to warrant its detection; (c) the desired level of significance; and (d) the desired study power. Given these four values, he then provided the following formula for calculation for sample size: $n = 2_{\bar{p}\bar{q}} (Z_a + Z_B)^2/(P_1-P_o).^2$ The values for this study are $\alpha = .05$; $\beta = .10$; $Z_a = 1.96$; and $Z_B = 1.28$.

Following this formula calculations for the appropriate sample size for alcohol exposure $p_o = .60$ with a relative risk (odds ratio) of 2.0 are:

α = .05

β = .10

Z_a = 1.96

Z_b = 1.28

P_o = .6

$P_1 = P_o (R) / [1 + P_o (R-1)]$

$\bar{p} = \frac{1}{2} (P_o + P_1)$ $q = 1 - p$

$n = 2\bar{p}\bar{q} (Z_a + Z_b)^2$

OR = 2

P_1 = .6 (2) / [1 + .6 (2 - 1)] = 1.2 / (1 + .6) = 1.2/1.6

= .75

$\bar{p}$ = ½ (.6 + .75) = ½ (1.35) = .675

$\bar{q}$ = 1 - .675 = .325

n = 2 (.675) (.325) $(1.96 + 1.28)^2$ / $(.75 - .6)^2$

= 2 (.219375) (10.4976) / $(.15)^2$

= 4.605822 / .0225 = 204.7032 n=205 cases in each group

OR = 3

P_1 = .6 (3) / [1 + .6 (3 - 1)] = 1.8 / (1 + 1.2) = 1.8/2.2

= .81818

$\bar{p}$ = ½ (.6 + .81818) = ½ (1.41818) = .70909

$\bar{q}$ = 1 - .70909 = .290909

$$n = 2\,(.70909)\,(.290909)\,(1.96 + 1.28)^2 / (.81818 - .6)^2$$

$$= 2\,(.20628)\,(10.4976) / (.21818)^2$$

$$= 4.33091 / .04760 = 90.9292 \quad n=91 \text{ cases each group}$$

Procedure

Letters were written to the hospital of birth for each case describing the study to them and asking for consent to review the relevant medical records. Copies of the institutional review forms from the University of Manitoba were included. Thirteen hospitals required additional forms for their own internal research committee reviews, and the remaining hospitals utilized the Institutional Review Board from the University.

Lists of SIDS cases and dead controls were produced for each hospital. Fourteen hospitals were visited personally and 9 hospitals were asked to review the cases and provide the data to the investigator. In four hospitals, telephone interviews were conducted with the health records staff during the data extraction process.

Each hospital was asked to retrieve the records. Typically these were stored off site and a delay between the request for records and the availability of the records was common. When the records had been retrieved, the hospitals were visited. The charts were reviewed individually. The records were not retrieved or reviewed by status, either SIDS or dead controls, but were rather reviewed in aggregate to diminish any possibility of bias in the data extraction process. In most cases, the data was extracted from both an infant's chart and the corresponding mother's chart. In some situations, there was sufficient duplication of records so that only one chart required review.

At most hospitals, the first review of the names yielded several records which could not be identified. This was most commonly due to children being born to an unmarried mother, but who were registered under their father's name. This circumstance also arose in several other situations, such as when the mothers used hyphenated names, or had been divorced and changed their names. At the larger hospitals (Health Sciences and St. Boniface), the process of locating all available records took a substantial amount of time, and necessitated repeated visits to complete the process. In all, to complete data collection required 28 trips to hospitals in the Winnipeg area and surrounding communities

When the cases had been reviewed, the controls were then requested. Controls were retrieved using the established criteria for matching: year of birth, month of birth, race, and maternal smoking status. In the larger hospitals this could be done by a computerized search of their data bases. In the smaller hospitals this required hand searching hospital admission records. Where possible, six potential control subjects were requested for each SIDS case. In smaller hospitals, these were requested consecutively, which was substantially more time-consuming, since the several matching variables required large numbers of charts that had to be reviewed in order to identify appropriate controls.

Where information on alcohol use was not indicated, those cases and controls were necessarily deleted. Matching on race was at times quite complicated, and often several appropriate controls would be identified, only to find that information on race was not available. This was significant, in that the information related to race could not be found

in any of the expected data sources (i.e., prenatal care record, nursing data base, billing forms under treaty status, nor in the records of the admitting physician/nursing staff).

Data Retrieval Form

In order to improve the accuracy of the data extraction process, photocopies of the Maternal Nursing Data Base and the Antepartum Risk Indicator were used to record the data from the chart review. On the original data collection tool, it had been planned to include both mothers' names, as well as fathers' names. However, due to concerns about confidentiality, the names were deleted. A similar problem occurred with medical record numbers, and they were also deleted from the data collection process.

Availability of Data

Of the 117 SIDS cases that were identified from the search of the PDRC records, the MEO records, and the Department of Vital Records of Manitoba, 110 could be located. These 110 also contained sufficient information to be considered as cases for the study. Six SIDS cases then had to be excluded due to conflicting information on alcohol use. In these six cases, the Manitoba Maternal Nursing Database indicated that the mother did not drink during pregnancy, while the physician notes or the prenatal care record indicated that the mother had used alcohol during pregnancy.

Prior to the start of data collection, the reliability of the data on the Manitoba Maternal Nursing Database (MMND) form was assessed, and found to be appropriate for use in this study. In the planning phase of this investigation, the potential problem of conflicting data entries had been anticipated, and it was decided at that time that the MMND would be the principal data source for the study. The use of the nursing database

as the principal data source was particularly justified by the higher rates of systematic inquiry about alcohol use during pregnancy by nurses. The rates of completion of the alcohol use sections of the MMND were much higher as compared with the infrequent completion of the corresponding section of the prenatal care record by physicians. Completion of the prenatal care record section on alcohol use, or the mention of alcohol use in the labor and delivery record was rarely seen.

Two cases could not be located, even after repetitive searches of the hospital's data base. In an attempt to find these two cases, the two larger tertiary hospitals were searched to see if the children had been referred, and a second search of Vital Records was made to confirm that the hospital of birth was correct. Some potential cases also could not be established. In one situation, a physician had left the hospital and taken the medical records of patients he had seen with him. In other situations, the records simply could not be located; in one hospital the maternal nursing data base was not utilized, and data on alcohol use from the prenatal care record or physician records was not sufficient.

For the non-SIDS deaths, a similar process of data retrieval was used. Substantial numbers of these records were not identifiable since there was considerable confusion concerning where the birth records were; also, prenatal care records were frequently absent in this group due to emergency transfers of these patients. Of the list of 165 non-SIDS deaths, it was possible to identify records and complete the extraction of data for 105 infants.

Data Entry

Data was transferred from medical records to data extraction forms, then entered into the computer for analysis. During the process of transferring data from the data extraction form to the computer, every SIDS case was entered twice in order to be able to identify and correct any data entry errors. When accuracy of data for the SIDS cases was in question, records were retrieved again from the birth hospital for verification. Data for the other cases and for the controls (living and dead) were entered into the computer once. Data entry was completed on the Number Cruncher Statistical System program (NCSS), and then transferred into the Epi Info 5.1 program.

A peculiarity of data transfer between these programs is that on the 5.1 version of Epi Info, missing data was coded as 0 when transferred from NCSS. Therefore, technical assistance was obtained form the Department of Biomedical Communications at the University of North Dakota to correct this aberration. The data were then checked again for accuracy.

When the data was entered into the statistical program, each variable was summarized to identify any potential coding error. These were then corrected by either referring to the original data extraction sheet or, in some cases, retrieval of the original records from the birth hospital.

CHAPTER IV

RESULTS

Data Analysis

This study had three primary objectives: (a) to describe the total population of SIDS cases from Manitoba for the years 1986 - 1990, in order to facilitate comparison with other studies of SIDS and non-SIDS deaths; (b) to determine if maternal alcohol use is a risk factor for SIDS; and (c) to determine if alcohol use during pregnancy was a specific SIDS risk factor, or if maternal alcohol use during pregnancy was a general-not cause specific-risk factor for infant death. In this Chapter, the results of the data analysis will be presented in reference to these objectives.

Descriptive Analyses

Records were available on 104 infants who died of **SIDS** and who also met the inclusion criteria for cases. Table 3 presents a breakdown of **SIDS** deaths and unmatched **non-SIDS** deaths by race and **year of death**. Sixty-two (60%) of these infants who died from **SIDS** were classified as **white,** and 42 (40%) were **aboriginal**. In this study the category **white** was used to group all non-aboriginals, and in fact, six infants were included who were non-white and non-aboriginal. Aboriginal people constitute 7-10% of the population of the Province of Manitoba. Of the 104 non-SIDS deaths, 41 (39%) were **aboriginal**.

Table 3

Year of death for study subjects by group SIDS and Controls

	SIDS				non-SIDS deaths			
Year of Death	Total	White		Aboriginal	Total	White		Aboriginal
1986	17	10	(16)	7 (17)	22	12	(19)	10 (24)
1987	18	13	(21)	5 (12)	17	9	(14)	8 (20)
1988	20	13	(21)	7 (17)	23	15	(23)	8 (20)
1989	22	10	(16)	12 (29)	23	16	(25)	7 (17)
1990	27	16	(26)	11 (26)	20	12	(19)	8 (20)
Total	104	62		42	105	64		41

These **non-SIDS** deaths were not matched, but were included because they died in one of the study years, were of the appropriate age, and were classified as **non-SIDS** deaths.

Table 4 lists **birth hospitals** for the 313 infants included in the study. Nine controls for some infants from the smaller hospitals were not available and needed to be selected from births at the Health Sciences Center or St. Boniface.

Table 5 lists the prevalence of the study risk factors by the three groups: **SIDS** death, **controls,** and **non-SIDS deaths,** which are also subgrouped by race. The study risk factors were drawn from the NICHD study of SIDS (Hoffman, et al., 1988), and from the review of SIDS risk factors reported by Carrol and Loughlin (1993). These variables are grouped into three categories: (a) maternal risk factors, (b) pregnancy, labor and delivery risk factors, and (c) postpartum risk factors.

Table 6 presents the **season of birth** for the three study groups: **SIDS, control** infants, and **non-SIDS.** The variable **"season"** is defined using the criteria from the Manitoba PDRC classification system. Winter consists of the months of December, January and February; spring the months of March, April, May; summer the months of June, July, August; and fall the months of September, October, November. There were no differences in the **season of birth** for SIDS and non-SIDS deaths ($\chi^2 = 6.15$; $p<.104$), indicating that a seasonal difference for month of birth of the sample was not present. No significant differences for season of birth was found for non-SIDS versus living controls ($\chi^2 = 5.96$; $p<.113$), nor for a comparison of SIDS versus non-SIDS versus living controls ($\chi^2 = .427$; $p<.935$).

Table 4

Hospital of Birth for the 104 SIDS Cases, 104 Matched Controls and the 105 Non-SIDS Deaths

		Total	SIDS		Living Controls		non-SIDS Deaths	
			White	Aboriginal	White	Aboriginal	White	Aboriginal
		n	n	n	n	n	n	n
1	Health Sciences Center	96	13	12	15	11	32	13
2	St. Boniface	84	16	9	16	12	22	9
3	Thompson	28	1	8	1	8	--	10
4	Steinbach	1	--	--	--	--	1	--
5	Swan Lake	2	--	1	--	1	--	--
6	Grace	11	5	--	6	--	--	--
7	Victoria	21	9	1	8	1	2	--
8	The Pas	8	2	2	2	2	--	--
9	Glenboro	2	1	--	1	--	--	--
10	Misercordia	14	3	3	3	3	--	2
11	Winkler	0	--	--	--	--	--	--
12	Selkirk	2	1	--	1	--	--	--
13	Dauphin	1	--	--	--	--	1	--
14	Brandon	12	4	1	4	1	--	2
15	Portage	7	1	1	1	1	2	1
16	Morden	5	2	--	2	--	1	--
17	Swan River Valley	7	1	2	1	2	1	--
18	Winipegosis	0	--	--	--	--	--	--
19	Killarney	1	--	--	--	--	1	-

Table 4 (continued)

	SIDS Total n	Control Aboriginal n	Control White n	non-SIDS Aboriginal n	non-SIDS White n	Aboriginal n	White n
Souris	1	--	--	--	--	1	--
Carmen	2	1	--	1	--	--	--
Flin Flon	7	2	1	--	--	--	4
Gods Lake Narrows	1	--	1	--	--	--	--
TOTALS	313	62	42	62	42	41	64
		104		104		105	

Table 5

Prevalence (number and percent) of study risk factors in the 3 study groups (SIDS, Controls, non-SIDS deaths)

	SIDS				Living Controls				Dead non-SIDS			
	White		Aboriginal		White		Aboriginal		White		Aboriginal	
	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)
Maternal Risk Factors												
Mothers Age												
< 20	9	(15)	15	(36)	7	(11)	5	(12)	5	(8)	10	(24)
> 35	3	(5)	1	(2)	3	(5)	2	(5)	7	(11)	3	(7)
Parity > 2	16	(26)	15	(36)	7	(11)	18	(43)	16	(25)	17	(41)
Marital status - unmarried	24	(39)	31	(74)	15	(24)	19	(45)	12	(19)	28	(68)
Previous abortion												
< 20 wks gestation	7	(11)	6	(14)	3	(5)	1	(2)	6	(9)	2	(5)
Prenatal care												
starting after first trimester	31	(50)	24	(57)	29	(47)	21	(50)	25	(39)	21	(51)
< 5 prenatal visits	10	(16)	14	(33)	10	(16)	6	(14)	10	(16)	14	(34)
No prenatal class	31	(50)	26	(62)	38	(61)	29	(69)	31	(48)	26	(63)
Previous live births now dead	3	(5)	2	(5)	1	(2)	3	(7)	4	(6)	5	(12)
Pregnant < 21 yrs age	27	(44)	32	(76)	15	(24)	31	(74)	26	(41)	30	(73)
Smoking	41	(66)	34	(81)	41	(66)	34	(81)	26	(41)	27	(41)
Alcohol exposure	17	(27)	14	(33)	22	(35)	11	(26)	12	(19)	14	(33)
Diabetic	0	(--)	3	(7)	1	(2)	2	(5)	2	(3)	0	(--)
Thyroid disorder	0	(--)	0	(--)	0	(--)	0	(--)	0	(--)	0	(--)
Not a planned pregnancy	20	(32)	22	(52)	23	(37)	18	(43)	20	(31)	19	(46)

Table 5 (continued)

	SIDS				Living Controls				Dead non-SIDS			
	White		Aboriginal		White		Aboriginal		White		Aboriginal	
	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)
Pregnancy, Labor and Delivery Complications												
Labor and Delivery Complications	16	(26)	12	(29)	15	(24)	8	(19)	30	(47)	19	(46)
Gestation < 37 weeks	9	(15)	7	(17)	5	(8)	5	(12)	20	(31)	12	(29)
Birthweight < 2500 grams	8	(13)	3	(7)	2	(3)	1	(2)	30	(47)	10	(24)
Birthweight < 1000 grams	4	(6)	0	(--)	0	(--)	0	(--)	16	(25)	4	(10)
Apgar 1 min < 5	5	(8)	4	(10)	4	(6)	3	(7)	13	(20)	6	(15)
Apgar 5 min < 7	2	(3)	4	(10)	1	(2)	1	(2)	9	(14)	4	(10)
Infant Complications at Delivery	14	(23)	3	(7)	11	(18)	1	(2)	39	(61)	17	(41)
Multiple Birth	4	(6)	1	(2)	3	(5)	1	(2)	7	(11)	1	(2)
Antepartum high risk score*												
Reproductive history	29	(47)	26	(62)	36	(58)	23	(55)	36	(56)	25	(61)
Associated conditions	6	(10)	6	(14)	11	(18)	9	(21)	13	(20)	5	(12)
Present pregnancy	19	(31)	14	(33)	14	(23)	8	(19)	34	(53)	14	(34)
Total score > 0	40	(65)	33	(79)	41	(66)	31	(74)	47	(73)	30	(73)

Table 5 (concluded)

	SIDS				Living Control				Dead non-SIDS			
	White		Aboriginal		White		Aboriginal		White		Aboriginal	
	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)
Postpartum Risk Factors												
Postpartum depression	2	(3)	1	(2)	2	(3)	4	(10)	1	(2)	1	(2)
Did not breast-feed baby before leaving hospital	23	(37)	17	(40)	9	(15)	16	(38)	17	(27)	19	(46)
Contact with protective service	3	(5)	6	(14)	0	(--)	0	(--)	5	(8)	2	(5)

*See Appendix 2

Table 6

Season of Birth for the 331 Study Subjects Subgrouped by Race

	SIDS				Controls				Non-SIDS Deaths			
	White		Aboriginal		White		Aboriginal		White		Aboriginal	
	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)
Season of birth												
Winter (Dec - Feb)	14	(23)	11	(26)	13	(21)	11	(26)	22	(34)	11	(27)
Spring (Mar - May)	13	(21)	7	(17)	12	(19)	6	(14)	15	(23)	8	(20)
Summer (June - Aug)	18	(29)	8	(19)	16	(26)	14	(33)	16	(25)	15	(37)
Fall (Sept - Nov)	17	(27)	16	(38)	21	(34)	11	(26)	11	(17)	7	(17)
TOTAL	62		42		62		42		64		41	

	SIDS		Controls		Non-SIDS Deaths	
Season	n	(%)	n	(%)	n	(%)
Winter	25	(24)	24	(23)	33	(31)
Spring	20	(19)	18	(17)	23	(22)
Summer	26	(25)	30	(29)	31	(30)
Fall	33	(32)	32	(31)	18	(17)

Group	Chi Square	p≤
SIDS vs Non-SIDS Deaths; df=3	6.15	.104
SIDS vs Non-SIDS Deaths v Controls; df=4	.427	.935
Non-SIDS Deaths vs Controls; df=3	5.96	.113

Table 7 presents **season of death** comparisons between the SIDS and non-SIDS deaths grouped by race. No significant difference between SIDS deaths and non-SIDS deaths were found ($\chi^2 = 1.33$; $p<.721$).

Table 8 presents **age of death** in days comparisons for the SIDS group and the non-SIDS group. Based on the entire sample, it was found that the SIDS infants died 26.3 days earlier than the non-SIDS infants, a significant finding ($t = 2.12$; $p<.036$). When each of these groups were stratified by race, however, significant differences were not found for SIDS vs Controls by race: whites ($t = 1.14$; $p<.259$); aboriginals ($t = 1.80$; $p<.075$).

Table 9 presents the **gender** breakdown by group and by race. The male : female ratio was 1.3 : 1 for the SIDS cases; 1.5 : 1 for the matched controls; and 1.2 : 1 for the non-SIDS deaths. No significant differences at the .05 level were found for gender between: (a) SIDS versus controls ($\chi^2 = .494$; $p < .482$); (b) living controls versus non-SIDS deaths ($\chi^2 = .611$; $p < .434$); or (c) SIDS versus non-SIDS deaths ($\chi^2 = .006$; $p < .938$).

Oyen, et al. (1990) had reported in their study of SIDS in North and South Dakota, that male gender was not associated with SIDS in American Indian infants. However, they did report an increased risk (odds ratio 1.8) for males in the white group only. Hayward, et al. (1990) had also found a gender ratio of 1.6 : 1 for whites in their study of American Indian and white SIDS cases in Wisconsin.

Table 7

Season of Death for SIDS Cases and the Non-SIDS Deaths.

	SIDS				Non-SIDS Deaths			
	White		Aboriginal		White		Aboriginal	
	n	(%)	n	(%)	n	(%)	n	(%)
Season of death								
Winter	16	(26)	14	(33)	14	(22)	10	(24)
Spring	13	(21)	11	(26)	19	(30)	9	(14)
Summer	13	(21)	8	(19)	15	(23)	10	(24)
Fall	20	(32)	9	(21)	16	(25)	12	(19)
TOTAL	62		42		64		41	

Group	Chi Square	p ≤
SIDS v Non SIDS Deaths; df=3	1.335	.721

Table 8

Age of Death in Days of Study Subjects by Group (SIDS and Non-SIDS Deaths).

Group	Race		Total
	White	Aboriginal	
SIDS	n = 62	n = 42	n = 104
Mean	96.3	99.9	97.75
SD	62.3	70.3	65.30
Range	27-303	29-335	27-335
Non-SIDS	n = 64	n = 41	n = 105
Mean	124.8	123.1	124.12
SD	109.5	110.8	109.49
Range	14-363	15-352	14-363

Group	t-value	p ≤
SIDS v Non-SIDS Deaths	2.12	.036
Aboriginals Only	1.14	.259
Whites Only	1.80	.075

Table 9

Gender by Group (%)

Gender	SIDS n	(%)	White n	(%)	Aboriginal n	(%)	Living Controls n	(%)	White n	(%)	Aboriginal n	(%)	Non-SIDS Deaths n	(%)	White n	(%)	Aboriginal n	(%)
Females	46	(44)	27	(44)	19	(45)	41	(39)	25	(40)	16	(38)	47	(45)	33	(52)	14	(34)
Males	58	(56)	35	(56)	23	(55)	63	(61)	37	(60)	26	(62)	58	(55)	31	(48)	27	(66)
	104						104						105					

Group	Chi Square	p ≤
SIDS v Controls	.494	.482
Controls vs Non-SIDS	.611	.434
SIDS vs Non-SIDS	.006	.938

Significant T-tests Comparisons of Cases and Living Controls

Table 10 portrays the results of the matched analysis for the comparisons of the continuous study variables for SIDS and living controls (paired t-tests). Table 11 presents the odds ratios for the categorical variables using a McNemar chi-square for the matched analysis. Six continuous variables were significant at the $p<.05$ level: **birthweight** ($t = 4.60$; $p < .0001$) (SIDS cases were 338 grams smaller than the controls); **Apgar score at five minutes** ($t = -2.11$; $p < .01$) (average score of 8.7 for cases and 8.9 for controls); **mothers' age** ($t = 2.71$; $p < .01$) (mothers of SIDS infants were an average of 1.9 years younger than mothers of controls); **number of pregnancies before 21** ($t = -2.37$; $p < .02$) (1.2 average pregnancies for cases and 0.8 for controls); **present pregnancy risk score** ($t = 3.49$; $p < .01$) (average score of 1.0 for cases and 0.4 for controls); and **total risk score** ($t = 2.73$; $p < .01$) (SIDS = 2.3 and controls = 1.5).

Significant Odds Ratio Comparisons of Cases and Living Controls

Seven categorical variables were significant at the $p<.05$ level: **began prenatal care after first trimester** (OR = 2.00; $p < .049$); **previous abortion** (OR = 5.50; $p < .02$); **birthweight < 2500 grams** (OR = 3.67; $p < .04$); **mothers' age** (OR = 1.44; $p < .02$); **pregnancy before age 21** (OR = 2.17; $p < .03$); **present pregnant risk score** > 1 (OR = 3.13; $p < .01$) ; and **unmarried** (OR = 2.50; $p < .01$) (Table 11).

Table 10

All Cases and Controls (n=104)

Paired t-Test Comparisons of SIDS Cases and Living Control Pairs on Continuous Variables

Variable	Cases			Controls			Pair differences			t-Value	P
	n	Mean	SD	n	Mean	SD	n	diff	se		two-tailed
Month Prenatal Care was begun	87	4.3	2.0	99	4.0	1.7	85	.32	.26	1.22	.23
Birthweight	104	3122	634	104	3461	478	104	-338	73.5	-4.60	.0001
Apgar 1 Minute	102	7.5	1.9	104	7.8	1.6	102	-.36	.25	-1.43	.15
Apgar 5 Minutes	102	8.7	1.0	104	8.9	0.7	102	-.24	.11	-2.11	.04
Mothers Age	103	23.7	5.3	104	25.6	4.9	103	-1.87	.69	-2.71	.01
Number Previous Pregnancies	104	1.8	1.8	104	1.5	1.5	104	.35	.22	1.59	.11
Number Pregnancies before 21	104	1.2	1.7	104	0.8	1.0	104	.46	.19	2.37	.02
Number of Prenatal Visits	89	7.4	4.1	99	8.1	3.6	87	-.64	.58	-1.11	.27
Gestation in Weeks	74	37.7	3.0	70	38.6	2.2	57	-.59	.51	-1.16	.25
Reproductive History Score	91	1.0	1.2	103	0.9	1.1	91	.13	.17	.75	.46
Associated Conditions Score	91	0.2	0.6	103	0.2	0.5	91	-.02	-.08	-.25	.80
Present Pregnancy Risk Score	91	1.0	1.6	103	0.4	0.8	91	.63	.18	3.49	.01
Total Risk Score	95	2.3	2.5	104	1.5	1.6	95	.85	.31	2.73	.01

Table 11

All Cases and Controls (N=104)

Categorical Variables Comparing SIDS to Paired Living Controls

McNemar Chi-Square Values (Aboriginals Only)

Variable	Discordant pairs Case + Control -	Case - Control +	McNemar Chi-Square	Odds Ratio	95% Confidence Interval		P Two-tailed
Began prenatal care after first trimester	24	12	3.84	2.00	1.0	4.00	.049
Prenatal visits < 5	20	10	3.20	2.00	0.94	4.27	.07
Gestation < 37 weeks	10	7	.52	1.43	0.54	3.75	.46
Previous abortion	11	2	4.92	5.5	1.22	24.81	.02
No Prenatal classes	12	13	.04	.92	.42	2.02	.84
Did not breast feed	25	13	3.66	1.92	.98	3.76	.06
Gender (M=1 F=0)	21	26	.53	.81	.45	1.44	.47
Referred to protective services	-	-	-	-	-	-	-
Labor & delivery complications	22	17	.64	1.29	.69	2.44	.42
Birthweight < 2500 gr	11	3	3.98	3.67	1.02	3.14	.04
Birthweight < 1500	4	0	-	-	-	-	-
Apgar1 < 5	9	6	.59	1.5	.53	4.21	.44
Apgar5 < 7	6	2	1.81	3.0	.61	14.86	.18

Table 11 (continued)

104 Pairs

Variable	Discordant pairs Case + Case -	Discordant pairs - +	McNemar Chi-Square	Odds Ratio	95% Confidence Interval		P Two-tailed
Infant complications at delivery	14	9	1.07	1.56	.67	3.59	.30
Multiple Birth	5	4	.11	1.25	.34	4.66	.74
Mother's Age < 20	23	16	1.24	1.44	.76	2.72	.02
Parity > 2	23	16	1.24	1.44	.76	2.72	.26
Live births now dead	5	4	.11	1.25	.34	4.66	.74
Pregnant before age 21	26	12	4.91	2.17	1.09	4.29	.03
Alcohol	20	22	.09	.91	.50	1.67	.76
Diabetic	3	3	-	-	-		
Not Planned pregnancy	18	17	.03	1.06	.55	2.05	.87
Reproductive history score ≥ 1	27	22	.50	1.23	.70	2.16	.48
Associated Conditions	10	17	1.77	.59	.27	1.29	.18
Present pregnancy score ≥ 1	25	8	7.87	3.13	1.41	6.93	<.01
Total risk score ≥ 1	24	15	2.04	1.60	.84	3.05	.15
Post-partum depression	3	5	.48	.60	.14	2.51	.48
Unmarried	35	14	8.40	2.50	1.35	4.65	<.01

In a study with a large number of variables interactions are an important source of potential variance. In this study the three matching variables were examined for interaction. The three variables **race, smoking** and **alcohol** were combined with each study variable. Table 12 presents the significant interactions by matching variables that were significant at p = <.05. Three variables had significant interaction with **race.** They were **number of previous pregnancies before 21, mothers age,** and **breastfeeding.** One variable had a significant interaction with **Smoking, mothers age** and two variables interacted with **Alcohol, Labor and delivery complications** and **associated conditions.**

Logistic Regression Modeling Utilizing Cases and Living Controls

A combination of study variables were used to develop a regression model to predict risk factors for SIDS (Table 13). This model retained two variables variables that were significant in the model at the p<.05 level. The predictor model was comprised of: **present pregnancy risk score** (OR = 4.91) and **mothers age** (OR = .90). The model parameters were $\chi^2 = 16.86$; $p < .01$.

The regression model was then rerun including the interaction terms from table 12. Each term was entered individually and all were entered together. The model that emerged is a 3 variable and 1 interaction term model (Table 14). The model parameters were $\chi^2 = 43.7$; $p < .001$. The null hypothesis of the study, that the independent study variable **alcohol** would not increase the risk of infant death from SIDS, could not be rejected.

This 3 variable, 1 interaction term model was comprised of **unmarried** a risk factor which increases the risk of SIDS OR=2.95 when compared to a married mother

matched by race and smoking status. The second variable was **birthweight**. Each 100 gram increase in birthweight decreases the risk for SIDS by 16% (OR=0.84) when compared to control infants matched by race, maternal smoking status, who weigh 100 grams less. The third variable was **mothers age**. A one year difference in mothers age between cases and controls, matched by race and maternal smoking status reduces the risk of SIDS by 10% (OR=0.90). The model includes one interaction term comprised of **race** X **number of previous pregnancies**. In this model **aboriginal** women and **number of previous pregnancies** was not significant $p < .44$. The interaction term was significant for **white** mothers and **number of previous pregnancies**. In this circumstance the risk of SIDS increases (OR=2.18) when two **white** mothers who are matched on smoking status are compared the woman with one additional pregnancy has a doubling of risk for SIDS compared with a white woman of similar smoking status with one less pregnancy.

This model demonstrated that alcohol use during pregnancy in this data set is not a risk factor for SIDS. Furthermore, alcohol use was not found to be a risk factor in the univariate analysis after using matching to control or reduce variance in the data due to race, maternal smoking during pregnancy, year of birth, and hospital of birth. In the design phase of the study, these variables were considered to be potential confounders. The results of the data analysis indicated that alcohol use was not a risk factor for SIDs after controlling for confounding from other significant risk factors by logistic regression. Interactions for race, smoking and alcohol were also considered in the analysis.

Table 12

Table of Significant Interactions for Race X Variable,

Smoking X Variable and Alcohol X Variable

Variable	Interactions β White = 0 β	OR	Aboriginals = 1 β	OR	P=
Number of previous pregnancies	.52	1.69	-.07	.93	.005
Mothers Age	-.027	.97	-.198	.82	.028
Breastfeeding	1.67	5.33	-.10	.89	.022
	Smoking Not Exposed = 0 β	OR	Exposed = 1 β	OR	
Mothers Age	-.008	.99	-.148	.86	.028
	Alcohol Not Exposed = 0 β	OR	Exposed = 1 β	OR	
Labor and Delivery Complications	-.274	.760	.888	2.43	.038
Associated Conditions	-1.26	.284	.396	1.49	.027

Table 13

All Cases and Controls (n=104)

Results of Stepwise Conditional Logistic Regression Modeling of 1 to 1 Matched SIDS Cases and Their Living Control Pairs (104 pairs maximum) Includes all variables significant at .10 in univariate analysis

65 Pairs were Retained in the Model.

Variable	Beta Estimate	Standard Error Estimate	Chi-Square	Odds Ratio	95% Confidence Interval		Probability
Pres Preg Score	1.59	.542	8.61	4.91	1.70	14.2	0<.01
Mother's Age	-.11	.048	5.39	.90	.82	.98	.02

Model Chi-Square = 16.86
df = 2
p = <.01

Variables Entered

Began prenatal care after first trimester
Apgar 5 minute
Mother's Age
Pregnant before age 21
Prenatal visits less than 5
Total risk score
Previous Abortion
Alcohol
Birthweight
Unmarried
Did not breast feed
Present Pregnancy Score > 0
Birthweight < 2500
# of preg before 21
Mother age <20

Table 14

All Cases and Controls (n=104)

Results of Stepwise Conditional Logistic Regression Modeling of 1 to 1 Matched SIDS Cases and Their Living Controls

Pairs (104 pairs maximum) Includes all variables significant at .05 in univariate analysis

92 Pairs Were Retained in the Model

Variable	Beta Estimate	Standard Error Estimate	Chi-Square	Odds Ratio	95% Confidence Interval		Probability
Unmarried	1.08	0.42	6.62	2.95	1.30	6.74	.0101
Birthweight (100 grams)	-.169	0.50	11.59	0.84	.76	.93	.0007
Mother's Age	-0.109	.046	5.56	0.90	.82	.98	.018
Interaction							
Aboriginal x Number Previous Pregnancies	-0.17	0.22	0.60	0.84	0.55	1.30	.44
White x Number Previous Pregnancies	0.78	0.27	8.13	2.18	1.28	3.74	.004

Model Chi-Square = 43.7
df = 5
p = <.001

Variables Entered

Birthweight
Mother's Age
Previous Abortion
Pres Preg Score > 0
Total risk score
Alcohol
Unmarried
Pregnant before age 21
Btwt < 2500
Number pregnancies
Race x Number Pregnancies

Further Analysis by Race

While **alcohol** (the principal independent variable of interest in this study) was not found to be a risk factor for SIDS in the total population of SIDS deaths, it may be possible that alcohol use during pregnancy could be a risk factor, with differing levels of risk based upon race. This possibility is a concern since aboriginals have the highest rates of SIDS in Manitoba (Moffatt, et al., 1988). The differential distribution of SIDS risk in aboriginal versus white populations remains an area of concern. To further study any potential contribution of alcohol as a risk factor for SIDS, it was decided to conduct a separate analysis stratified by race.

Comparisons of Aboriginal Cases to Matched Living Controls

Continuous Variables

The univariate analysis of the study risk factors was stratified by race. Tables 15 and 16 present the results for the aboriginal cases and matched living controls. Table 15 presents the paired t-tests for the continuous variables which were significant at the $p<.05$ level. These were: **birthweight** ($t = -3.10$; $p<.01$) (cases weighed 330 grams less than controls on average); **mothers' age** ($t = -3.74$; $p<.01$) (mothers of SIDS infants were 3.6 years younger than mothers of controls); **present pregnancy risk score** ($t = 2.58$; $p<.01$) (score was 0.7 higher in SIDS cases) and **total risk score** ($t = 2.54$; $p<.02$) (score for SIDS cases was 1.3 higher than controls).

Table 15

Aboriginals Only (n=42)

Paired t-Test Comparisons of SIDS Cases and Living Control Pairs on Continuous Variables

Variable	Cases			Controls			Pair differences			t-Value	P
	n	Mean	SD	n	Mean	SD	n	diff	se		two-tailed
Month Prenatal Care was begun	33	5.0	2.2	38	4.3	1.6	32	.63	.50	1.24	.22
Birthweight	42	3233	537	42	3564	490	42	-330	106	-3.10	<.01
Apgar 1 Minute	42	7.5	1.9	42	7.8	1.8	42	-.33	.44	-.76	.45
Apgar 5 Minutes	42	8.5	1.2	42	8.9	0.7	42	-.38	.20	-1.89	.07
Mothers Age	42	21.9	4.7	42	25.5	5.1	42	-3.55	.94	-3.74	<.01
Number Previous Pregnancies	42	2.1	2.1	42	2.4	1.7	42	-2.62	.42	-.62	.53
Number Pregnancies before 21	42	1.6	1.6	42	1.4	1.1	42	-2.38	.31	.76	.45
Number of Prenatal Visits	34	5.9	4.4	38	7.9	3.7	33	-1.58	1.04	-1.51	.14
Gestation in Weeks	28	37.7	2.1	30	38.4	2.8	24	-.46	.87	-.53	.60
Reproductive History Score	37	1.3	1.4	42	1.1	1.3	37	.29	.31	.95	.35
Associated Conditions Score	37	0.3	0.8	42	0.2	0.5	37	.05	.15	.34	.73
Present Pregnancy Risk Score	37	1.0	1.7	42	0.3	0.7	37	.75	.29	2.58	.01
Total Risk Score	39	2.9	2.9	42	1.6	1.6	39	1.38	.55	2.54	.02

Table 16

Categorical Variables Comparing SIDS to Living Controls.

McNemar Chi-Square Values Not Corrected(Aboriginals Only)

Variable	Discordant pairs Case + / Case -	- / +	McNemar Chi-Square	Odds Ratio	95% Confidence Interval		P Two-tailed
Began prenatal care after first trimester	11	6	1.43	1.83	.68	4.96	.23
Prenatal visits < 5	12	5	2.71	2.40	.85	6.81	.10
Gestation < 37 weeks	5	4	.11	1.25	.34	4.66	.74
Previous abortion	6	1	2.75	6.00	.72	-49.84	.09
No Prenatal classes	6	4	.39	1.50	.42	5.31	.53
Did not breast feed	9	10	.05	.90	.37	2.21	.82
Gender (M=1 F=0)							
Referred to protective services	7	10	.52	.70	.27	-1.84	.47
Labor & delivery complications	9	5	1.11	1.80	.60	5.37	.29
Birthweight < 2500 gr	3	1	.91	3.00	.31	28.84	.34
Birthweight < 1500	-	-	-	-	-	-	-
Apgar1 < 5	4	3	.14	1.33	.30	5.96	.71
Apgar5 < 7	4	1	1.54	4.00	.45	-.35.79	.22
Infant complications at delivery	3	1	.91	3.00	.31	28.84	.34

Table 16 (continued)

Aboriginals Only

Variable	Discordant pairs Case + / Case -	Discordant pairs - / +	McNemar Chi-Square	Odds Ratio	95% Confidence Interval		P Two-tailed
Multiple Birth	1	1	.0	1.00	.06	15.99	1.00
Mother's Age < 20	12	2	5.50	6.00	1.34	26.81	.02
Parity > 2	9	11	.20	.82	.34	1.97	.66
Live births now dead	2	3	.20	.67	.11	3.99	.66
Pregnant before age 21	7	5	.33	1.40	.44	4.41	.57
Alcohol	9	6	.59	1.50	.53	4.21	.44
Diabetic	3	2	-	-	-	-	-
Not Planned pregnancy	11	6	1.43	1.83	.68	4.96	.23
Reproductive history score ≥ 1	13	7	1.74	1.86	.74	4.66	.19
Associated Conditions	4	7	.80	.57	.17	1.95	.37
Present pregnancy score ≥ 1	11	3	3.98	3.67	1.02	13.14	.046
Total risk score ≥ 1	8	4	1.54	.25	.03	2.24	.22
Post-partum depression	1	4	1.54	.25	.03	2.24	.22
Unmarried	17	5	5.79	3.40	1.25	9.22	.02

Categorical Variables

The categorical variables significant at p<.05 were: **mothers' age less than 20** (OR = 6.00); **unmarried (OR = 3.40)**; and **present pregnancy risk score** > 1 (OR = 3.67) (Table 16).

Regression Modeling

These variables and the other study variables were used to develop a regression model to identify SIDS risk factors that may be specific to the aboriginal population (Table 17). Again, the model retained only variables that were significant in the model at p<.05. The only significant variable specific to this model was **unmarried** mother (OR = 6.00). Again, **alcohol** did not emerge as a significant risk factor for SIDS in the aboriginal infants in this study, even after matched a stratified analysis of data for race, smoking, hospital of birth, and year of birth and while simultaneously controlling for the presence of confounding among the other variables. The model parameters were $\chi^2 = 7.93$; $p < .005$.

Comparisons of White Cases to Matched Living Controls

Continuous Variables

Tables 18 and 19 present the univariate analysis of risk factors for the white population only. As seen in Table 18, the continuous variables that were significant were: **birthweight** (t = -3.41; p<.001) (SIDS infants weighed on average 343 grams less than controls at birth); **number of previous pregnancies** (t = - 3.46; p<.001) (SIDS mothers

Table 17

Aboriginals Only (n=42)

Results of Stepwise Logistic Regression Equation for

1-1 Matched SIDS Cases and Their Living Controls

(42 Pairs Maximum)

24 Pairs Were Included in the Model.

Variable	Beta Estimate	Standard Error Estimate	Chi-Square	Odds Ratio	Probability
Unmarried	1.79	.764	5.50	6.00	.019

Model	Chi-Square	=	7.93
	df	=	1
	p	=	.005

= 1.6 and controls = 0.8); **number of pregnancies before age 21** (t = - 2.46; p<.01) (SIDS mothers = 0.9 and controls = 0.4); and **present pregnancy risk score** (t = -2.36; p <.02) (SIDS scores were 0.5 higher than controls).

Categorical Variables

The significant categorical variables (Table 19) were **non-breastfeeding** (OR = 5.33); **parity greater than 2** (OR = 2.80), and **pregnant before age 20** (OR = 2.71) and present pregnancy risk score ≥1 (OR = 2.80).

Regression Modeling

A regression model of SIDS using the derived categorical (RF) risk factors for the white group is presented in Table 20. The two model variable was **non-breastfeeding** (OR = 7.65 and birthweight (OR = 1.00). **Alcohol** was not a risk factor for SIDS in this population of white infants, even after using matching and simultaneous adjustment for confounding in the analysis. The second null hypothesis of the study, that alcohol as a risk factor would not differ between the two subgroups defined by race, could not be rejected.

Analysis Comparing Non-SIDS Deaths to Living Controls

The last area of planned analysis was to compare living controls to non-SIDS deaths to determine if **alcohol** may be a risk factor for death in the non-SIDS group, even though it was not found to be a risk factor in the comparison of SIDS and matched living controls.

Continuous Variables

Table 21 presents the results of comparisons between the 104 living controls and the 105 non-SIDS deaths for the continuous variables significant at the p<.05 level. These

Table 18

Whites Only (n=62)

Paired t-Test Comparisons of SIDS and Paired Living Controls pairs on Continuous Variables

Variable	Case n	Case Mean	Case SD	Control n	Control Mean	Control SD	Pair differences n	Pair differences diff	Pair differences se	t-Value	P< two-tailed
Month prenatal care was begun	54	3.9	1.6	61	3.8	1.7	53	.13	.28	.46	.64
Birthweight	62	3047	687	62	3391	461	62	-343	100	-3.41	.001
Apgar 1 minute	60	7.5	2.0	62	7.8	1.6	60	-.38	.30	-1.26	.21
Apgar 5 minutes	60	8.8	0.9	62	8.9	0.6	60	-.13	.12	-1.05	.29
Mothers Age	61	25.0	5.3	62	25.7	4.8	61	-.72	.95	-0.76	.45
Number previous pregnancies	62	1.6	1.5	62	0.87	1.1	62	.76	.21	3.46	.001
Number pregnancies before 21	62	0.98	1.7	62	0.37	0.8	62	.61	.25	2.46	.01
Number of prenatal visits	55	8.44	3.5	61	8.23	3.5	54	-.07	.68	-.10	.91
Gestation in weeks	46	37.7	3.5	40	38.8	1.7	33	-.69	.64	-1.09	.28
Reproductive history score	54	0.8	1.1	61	0.8	.9	54	.02	.20	.08	.92
Associated conditions score	54	0.1	0.5	61	0.2	0.5	54	-.07	.11	-.70	.48
Present pregnancy risk score	54	0.9	1.5	61	0.4	0.9	54	.55	.23	2.36	.02
Total risk score	56	1.9	2.1	62	1.5	1.6	56	.48	.36	1.32	.19

Table 19

Whites Only (n=62)

Categorical Variables Comparing SIDS to Living Controls.

McNemar Chi-Square Values Not Corrected for Continuity.

Variable	Discordant pairs Case + Case -	- +	McNemar Chi-Square	Odds Ratio	95% Confidence Interval		P Two-tailed
Began prenatal care after first trimester	13	6	2.45	2.17	.82	5.70	.12
Prenatal visits < 5	8	5	.68	1.60	52	4.89	.41
Gestation < 37 weeks	5	3	.49	1.67	.40	6.97	.48
Previous abortion	5	1	2.16	5.00	.58	42.8	.14
No Prenatal classes	6	9	.59	.67	.24	1.87	.44
Did not breast feed	16	3	7.08	5.33	1.55	18.30	<.01
Gender (M=1 F=0)	14	16	.13	.87	.43	1.79	.72
Referred to protective services	-	-	-	-	-	-	-
Labor & delivery complications	13	12	.04	1.08	.49	2.37	.84
Birthweight < 2500 gr	8	2	3.07	4.00	.85	18.84	.08
Birthweight < 1500	4	0	-	-	-	-	-
Apgar1 < 5	5	3	.49	1.67	.40	6.97	.48
Apgar5 < 7	2	1	.32	2.00	.18	22.06	.57
Infant complications at delivery	11	8	.47	1.38	.55	3.42	.49

Table 19 (continued)

Whites Only

Variable	Discordant pairs Case + / Case -	Discordant pairs - / +	McNemar Chi-Square	Odds Ratio	95% Confidence Interval		P Two-tailed
Multiple Birth	4	3	.14	1.33	.30	5.96	.71
Mother's Age < 20	7	5	3.33	1.40	.44	4.41	.57
Parity > 2	14	5	3.91	2.80	1.01	7.77	.048
Live births now dead	3	1	.91	3.00	.31	28.84	.34
Pregnant before age 21	19	7	5.10	2.71	1.14	6.46	.02
Alcohol	11	16	.92	.69	.32	1.48	.34
Diabetic	-	-	-	-	-	-	
Not Planned pregnancy	7	11	.87	.64	.25	1.64	.35
Reproductive history score ≥ 1	14	15	.03	.93	.45	1.93	.85
Associated Conditions	6	10	.98	.60	.22	1.65	.32
Present pregnancy score ≥ 1	14	5	3.91	2.80	1.01	7.77	.048
Total risk score ≥ 1	16	11	.92	1.46	.68	3.13	.34
Post-partum depression	2	1	.32	2.00	.18	22.06	.57
Unmarried	18	9	2.88	2.00	.90	4.45	.09

Table 20

Whites Only (n=62)

Results of Stepwise Logistic Regression Equation for

(62 Pairs Maximum)

41 Pairs Were Included in Analysis.

Variable	Beta Estimate	Standard Error Estimate	Chi-Square	Odds Ratio	Probability
Not breast feeding	2.04	.847	5.77	7.65	.02
Birthweight	-.001	.001	5.46	1.00	.02

Model Chi-Square = 14.00
df = 2
p = <.001

variables were: **birthweight** (t = 8.28; p < .001) (non-SIDS deaths weighed 878 grams less than the living controls); **Apgar at 1 minute** (t = 4.81; p < .0001) (non-SIDS deaths = 6.5 living controls = 7.8); **Apgar at 5 minutes** (t = 5.28; p < .0001) (non-SIDS deaths = 7.9 living controls = 8.9); **number of previous pregnancies** (t = - 2.20; p < .029) (non-SIDS deaths 2.0 living controls = 1.5); **number of pregnancies before 21** (t = -2.09; p = < .019) (non-SIDS deaths 1.2 living controls = 0.7); **number of prenatal visits** (t = 2.52; p <. 006) (non-SIDS deaths =6.6 living controls = 8.1); **gestation in weeks** (t = 4.39; p < .001), (non-SIDS deaths = 35.5 weeks and living controls = 38.6 weeks); **reproductive history score** (t = - 2.29; p < .011) (non-SIDS deaths = 1.4 and living controls = 0.9); **present pregnancy score** (t = - 5.69; p <. 001) (non-SIDS deaths = 1.7 and living controls = 0.4); and **total score** (t = - 5.44; p < .001) (non-SIDs deaths = 3.6 and living controls = 1.5).

Categorical Variables

The categorical variables significant at the .05 level (Table 22) were: **gestation < 37 weeks** (OR = 3.84); **referral to protective services** (OR = 1.59); **labor and delivery complications** (OR = 3.14); **birthweight < 2500 grams** (OR =6.25), **birthweight < 1500 grams** (OR = 2.24); **1 minute Apgar < 5** (OR = 3.17); **5 minute Apgar < 7** (OR = 7.45); **complications at delivery** (OR = 9.33); **present pregnancy risk score > 1** (OR = 3.93); and **total risk score** (OR = 2.14).

Regression Modeling

The model included three variables that were significant in the logistic predictor model at .05 for non-SIDS deaths (Table 23). These were: **birthweight < 2500 grams**

Table 21

t-Test Comparisons of Living Controls (n=104) and non-SIDS Deaths (n=105) on Continuous Variables

Variable	Living controls			Non-SIDS Deaths			t-Value	P
	n	Mean	SD	n	Mean	SD		
Month prenatal care was begun	99	3.9	1.6	81	4.21	1.9	-0.97	.165
Birthweight	104	3460	478	104	2582	969	8.28	<.001
Apgar 1 minute	104	7.8	1.6	102	6.5	2.3	4.81	<.001
Apgar 5 minutes	104	8.9	0.6	102	7.9	1.8	5.28	<.001
Mothers aAge	104	25.6	4.9	105	25.7	6.1	-0.18	.429
Number previous pregnancies	104	1.5	1.5	105	2.0	1.9	-2.20	.014
Number pregnancies before 21	104	0.7	1.1	105	1.2	1.8	-2.09	.019
Number of prenatal visits	99	8.1	3.6	88	6.8	3.8	2.52	.006
Gestation in weeks	70	38.6	2.2	82	35.9	5.2	4.39	<.001
Reproductive history score	103	.09	1.1	93	1.4	1.9	-2.28	.012
Associated conditions score	103	.02	0.5	93	0.3	0.7	-0.54	.296
Present pregnancy risk score	103	.04	0.8	93	1.7	2.2	-5.69	<.001
Total risk score	104	1.5	1.6	93	3.6	3.3	-5.44	<.001

Table 22

Categorical Variables Comparing Living Controls (n=104) and Non-SIDS Deaths (n=105).

All Chi-Square Values are Yates Corrected.

Variable	Odds Ratio	95% Confidence Interval	Chi-Square	P
Began prenatal visits after first trimester	1.29	0.71 to 2.32	0.71	.400
Prenatal visits < 5	1.95	0.96 to 3.96	3.42	.064
Gestation < 37 weeks	3.84	1.72 to 8.57	11.56	.001
Previous abortion	2.19	0.64 to 7.54	1.64	.201
Prenatal classes	0.08	0.46 to 1.72	0.13	.721
Did not breast feed	1.82	0.98 to 3.37	3.65	.056
Referred to protective services	1.59	1.29 to 1.97	3.88	.049
Labor & delivery complications	3.14	1.72 to 5.73	14.36	<.001
Birthweight < 2500 gr	21.04	6.25 to 70.87	40.13	<.001
Birthweight < 1500 gr	2.24	1.9 to 2.62	22.13	<.001
Apgar1 < 5	3.17	1.27 to 7.92	6.61	.010
Apgar5 < 7	7.45	1.64 to 33.91	8.93	.003
Infant complications at delivery	9.33	4.56 to 19.12	43.79	<.001
Multiple birth	2.08	0.61 to 7.15	1.42	.234
Mother's age < 20	1.28	0.57 to 2.88	0.35	.554

Table 22 (continued)

Variable	Odds Ratio	95% Confidence Interval	Chi-Square	P
Parity > 2	1.45	0.79 to 2.71	1.53	.216
Live births now dead	2.39	0.71 to 8.04	2.10	.147
Pregnant before age 21	1.54	0.89 to 2.66	2.35	.126
Alcohol	0.71	0.39 to 1.29	1.25	.263
Diabetic	0.65	0.11 to 3.99	0.22	.642
Unplanned pregnancy	1.44	0.76 to 2.74	1.26	.262
Reproductive history score ≥ 1	1.42	0.79 to 2.54	1.42	.233
Associated conditions	0.99	0.49 to 2.02	.000	.991
Present pregnancy score ≥ 1	3.93	2.11 to 7.32	19.48	<.001
Total risk score ≥ 1	2.14	1.08 to 4.23	4.90	.027
Post-partum depression	0.33	0.07 to 1.68	1.96	.162
Unmarried	1.31	0.74 to 2.31	0.85	.357

*Fishers exact 1-tailed - due to empty cell or smaller than predicted cell size.

Table 23

Results of Stepwise Logistic Regression Equation for Living Controls (n=104) and Non-SIDS Deaths (n=105).

The numbers of Living Controls in the analysis: cases (n=73) and Non-SIDS Deaths (n=44).

Variable	Beta Estimate	Standard Error Estimate	Chi-Square	Odds Ratio	Probability
INTERCEPT	-1.7463	0.4075	18.36	0.17	<.001
Birthweight < 2500	2.5454	1.0978	5.38	12.75	.020
Pregnant before 21	0.9976	0.4807	4.31	2.71	.038
Infantcomplications at delivery	1.5746	0.5475	8.27	4.83	.004

Model	Chi-Square	30.12
	df	3
	p=	.0001

(OR = 12.75); **pregnant before 21** (OR = 2.71) and **complications at labor** (OR = 4.83). The model parameters were $X^2 = 33.12$; $p<.0001$. Maternal alcohol use during pregnancy was not a risk factor for non-SIDS deaths in this sample.

Conclusions Regarding the Alcohol Variable

In this study, **alcohol** has not been found to be a risk factor for SIDS when SIDS cases were compared to the matched living controls.

CHAPTER V

DISCUSSION

Insufficient evidence has been found to reject the primary null hypothesis of this study which was that **alcohol** exposure during pregnancy would not be a risk factor for SIDS. The secondary null hypothesis of the study also could not be rejected, that is, the results of this study did not indicate that maternal alcohol use during pregnancy was a significant risk factor for SIDS in either aboriginal or white samples. In addition, alcohol was not found to be a risk factor in the non-SIDS infant death group in this study. When compared with living controls, the group of infants who died from non-SIDS causes were not found to be at increased risk of death related to maternal alcohol use during pregnancy.

The prevalence rates of exposure to alcohol in this study were: 29.8% among SIDS cases, 31.4% among the living controls, and 24.7% among the infant group which died from non-SIDS causes (Table 24). While no other studies have examined alcohol use as the primary independent study variable, several others have included alcohol use during pregnancy as a variable for SIDS. Prevalence of alcohol exposure was previously reported by Dwyer, et al. as 52% (OR = 0.92) (1995), and by Hoffman, et al. as 61% (1988 a; 1988 b). However, no previous studies found maternal alcohol use during pregnancy to be a risk factor for SIDS.

Table 24

Summary Data on Alcohol Exposure

Group		Exposed		OR	95% CI	p=
	n	n	%			
Cases	104	31	29.8	.91	0.50 to 1.67	.76
Controls		33	31.4			
Whites	62	17	27.4	0.69	0.32 to 1.48	.34
Controls		22	35.5			
Aboriginals	42	14	33.3	1.50	0.53 to 4.21	.44
Controls		11	26.2			

Outcomes in this Study

In a study in which the outcomes are negative findings, that is, in which the null hypotheses are not rejected, an area of concern that needs to be addressed is that of internal validity (Campbell & Stanley, 1963). In this section of this report the validity of the study findings will be discussed.

Sample Size

In this study, the first area of concern would be that of adequate sample size. It needs to be questioned if the sample of 104 SIDS cases did not have sufficient power to detect differences due to alcohol exposure. Inadaquate sample size could increase the chance of finding an effect in one sample and missing it in a subsequent sample. Table 25 presents the power calculations for the analyses that were completed to determine if alcohol exposure is a risk factor for SIDS. Adequate power to detect risk differences were present.

Sensitivity of the Variable **Alcohol**

The reliability of the independent study variable was found to be adequate in this pilot study. This section of the report will now address the sensitivity of the alcohol variable in this study. Since **alcohol** was not found to be a risk factor for all SIDS cases who were exposed, the cases were then stratified for analysis of cases with **high exposure** to their matched controls. For this unplanned analysis, cases were stratified by

Table 25

Power Calculations for the Subgroups Analysis to Determine

Power to Detect a Doubling of Risk for Exposure to Primary Independent Study Variable Alcohol

Group	Pairs	Risk	Zβ	Power
SIDS and Controls	104	3	2.2344	.98727
SIDS and Controls, Whites Only	62	3	1.254	.89501
SIDS and Controls, Aboriginals Only	42	3	0.6876	.75415

anecdotal data from notes taken during the data collection phase of the study which identified mothers of SIDS cases who were very heavy drinkers (n=10). Since the power of this comparison was low due to the very small sample size, the non-significant variables were also examined to determine if differences were present which would suggest that a type II error may have occurred.

High Dose Alcohol Exposure

In order to determine if differences in the prevalence of high alcohol exposure between the SIDS sample and living controls sample were compared. In the SIDS sample 10 cases had high alcohol exposure, in the living controls 2 cases of high alcohol exposure were found. The difference in the high alcohol exposure between the SIDS cases and living controls was significant (χ^2=4.33; p<.037). The relative risk for high alcohol exposure in the SIDS sample was 5.00 (95% CI 1.12 to 22.27).

The SIDS cases were then grouped into three groups: no exposure, exposed, and high exposure (Table 26). For this comparison, only continuous variables were used. The variables which demonstrated a dose-response relationship across the three exposure levels were: **birthweight**, **Apgar score 1 minute**, **Apgar score 5 minutes**, **number of previous pregnancies** and **gestation in weeks**. This suggests that variable **alcohol** could demonstrate sufficient sensitivity to differentiate among three levels of exposure in the SIDS sample. While alcohol exposure may have important biological implications, for example, decreased birthweight, alcohol exposure in this sample did not appear to increase the risk of SIDS. A one-way analysis of variance found that high alcohol exposure did

Table 26

Effects of differing levels of exposure to alcohol for SIDS cases.

The variables presented are those which demonstrated a dose-response relationship.

One Way Analysis of Variance

Variable	No Exposure			Exposed			High Exposure					
	n	Mean	SD	n	Mean	SD	n	Mean	SD	F	df	p
Birthweight	73	3182	575	21	3060	678	10	2812	885	1.64	2,101	20
Apgar 1 minute	72	7.7	1.8	21	7.3	1.9	9	6.3	2.7	2.12	2,99	.13
Apgar 5 minutes	72	8.8	1.0	21	8.7	0.9	9	8.2	1.1	1.23	2,99	36
Number Previous Pregnancies Before 21	73	1.8	1.8	21	1.9	1.6	10	2.1	1.7	.43	2,101	.65
Gestation in Weeks	50	38.4	2.3	16	37.3	3.8	8	34.6	3.4	6.33	2,71	.01

shorten the length of pregnancy by 3.8 weeks ($P < .05$) when compard to unexposed pregnancies.

Although this study did not identify alcohol as a risk factor for SIDS, it remains possible that maternal alcohol ingestion during pregnancy plays a role in SIDS. It is possible that this study failed to identify this relationship as a result of either a design flaw, or the presence of a confounder that was not considered and not controlled. An example of this is the possibility of a relationship between SIDS and very high levels of alcohol exposure. In fact, it may be reasonable to suspect that a woman could ingest alcohol at very high levels during pregnancy, and greatly increase the risk of a child's death in utero. However, it may be that this death would not be classified as SIDS, or that the event would not occur often enough to make this possibility a significant source of infant mortality.

External Validity Considerations

The last area of concern about the results of the study deal with the external validity of a study. Spector (1985) noted that the external validity of the study can be compromised under several conditions, two of which may be relevant to this study. The first threat is caused by use of a measurement tool that is not valid when applied to a larger population, for example, SIDS cases who died after the study period. The second threat can be caused by inappropriate sample selection. Although unlikely, in this project the SIDS cases used in the study may have differed in some important but unknown way from typical SIDS cases in Manitoba, or from other locations outside of Manitoba. The study sample was comprised of 104 of the SIDS cases identified by the PDRC and the

MEO in Manitoba. The possibility exits that the case definition used to classify SIDS cases from the all-cause population of infant deaths in Manitoba was applied selectively or that an inappropriate definition was used. However, the likelihood of either of these possibilities appears remote. The PDRC and the MEO used the commonly accepted definition of SIDS during the study period and the characteristics of Manitoba SIDS deaths appear to be similar to other populations in Canada (Moffatt, et al., 1988; Millar & Hill, 1993) and from other countries (Scragg, et al., 1993; Mitchell, et al., 1993; Kaplan, et al., 1984 and Hoffman et al., 1988).

Variables Recommended for Further Study

Table 27 summarizes the results of significant results that were found between SIDS cases and matched controls on the stratified analysis by **race**. The variables that were identified in this study, with two exceptions, are the same as those found to be risk factors for infant death and for SIDS in previous studies.

Table 28 summarizes risk factors comparing the SIDS cases with the living controls, and comparing the non-SIDS deaths with the living controls. This table

Table 27

Summary of Significant Results between SIDS and Matched Controls by Race

	t-tests	Odds Ratios	Logistic Regression
Aborigonal Only; n = 42 pairs	Birthweight, Mother's age,, Total risk score, Present pregnancy risk score, Apgar 5 minutes	Mother's age <20, Unmarried, Present pregnancy risk score	Unmarried
White Only: n = 62 Each	Birthweight, Number previous, pregnancies, Number of pregnancies before 21, Present pregnancy risk score	Parity >2, Pregnant before age 21, Did not breast feed	Did not breast feed

Table 28

Summary of Significant Risk Factors for SIDS vs Matched Living Controls and for Living Controls vs Non-SIDS Deaths.

	t-tests	Odds Ratios	Logistic Regression
SIDS and Controls:	Birthweight, Mothers age, Number, of pregnancies before 21, Total risk score, Present pregnancy risk score, Apgar 5 minutes, Previous Pregnancies, Gestation in weeks	Began prenatal care after first trimester, Mother's age <20, Pregnant before age 21, Present pregnancy score ≥1, Unmarried, Previous abortion	Mothers age, Present pregnancy risk score
Non-SIDS and Living Controls	Birthweight, Apgar 1 minute, Apgar 5 minutes, Number previous pregnancies, Number pregnancies before 21, Number of prenatal visits, Gestation in weeks, Reproductive history score, Number previous pregnancies, Total risk score	Gestation <37 weeks, Referred to protective services, Labor & delivery complications, Birthweight <2500 gr, Birthweight <1500 gr, complications Apgar <5, Apgar <7, Present pregnancy score, Reproductive history score ≥1	Birthweight <2500 gr Pregnant before age 21 Labor & delivery

identifies the risk factors which the two groups of infant deaths have in common, and those that were not shared. While considerable overlap was evident, the non-SIDS deaths had several unique risk factors that were not found in the comparison of the SIDS cases with the matched living controls. In general, the non-SIDS risk factors were typical for a sample of all cause infant deaths (SIDS excluded). These variables related to low birthweight, inadequate prenatal care, and complications at labor and delivery. The SIDS specific risk factors are more closely associated with adverse socioeconomic status, for example, evidenced by the numbers of young and/or unmarried mothers. Risk factors which would readily identify mothers at risk, or infants who are at risk for SIDS, were not found. This has been extensively discussed in Chapter 2 (literature review) above. Most recently, Arntzen, et al . (1995) reported in Norway, that much of the increased rate of postneonatal mortality in groups with lower socio-economic status is associated with higher SIDS rates.

Low socio-economic status has previously been found to be a risk factor for SIDS (Moffatt, et al., 1988; Krongrad, 1991). This study did not collect data on social or economic status. However, by comparing the maternal characteristics of the mothers of the SIDS cases and the matched living controls with data from a study of prenatal care in Winnipeg, it may be possible to estimate the SES status of the SIDS cases and controls. In 1994, Mustard and Roos published a study of prenatal care in Winnipeg, Manitoba. In their study, they presented the income quartiles for women from Winnipeg who had received prenatal care in 1987. Table 29 presents a comparison of the data from their study, with the maternal characteristics from this present study of Manitoba SIDS deaths

Table 29

Comparisons of Maternal Characteristics from Mustard and Roos 1994 to

Maternal Characteristics of SIDS and Matched Controls from 1986-1990

Variable	Mustard and Roos 1994			SIDS			Controls		
		Income Quartile							
	Total	Highest	Lowest	Total	Aboriginal	White	Total	Aboriginal	White
Gestation < 37 weeks	5.9	4.6	6.3	15.4	16.6	15.0	9.6	11.9	8.1
Aboriginal	4.4	0.2	13.4	40.1			40.1		
Unmarried	26.5	15.3	45.3	52.8	73.8	38.7	32.6	45.2	24.2
Smoked	27.6	15.3	38.1	72.0	81.0	66	72	81	66
Maternal Age									
<20	7.0	2.9	14.1	23.1	35.7	14.5	11.5	11.9	11.2
>35	9.0	13.9	6.5	3.8	2.4	4.8	4.8	4.7	4.8
Birthweight									
<1500	0.8	0.7	1.2	3.8	--	6.5	--	----	
<2500	3.9	2.8	5.0	10.5	7.1	12.9	2.8	2.4	2.8

for 1986 through 1990. Except for mothers over 35 years of age, the mothers of the SIDS cases would appear to be of lower socio-economic status than the group with incomes from the lowest income quartile in the Mustard and Roos (1994) study. This comparison would support earlier studies from Manitoba that low social and economic status are important risks for infant death generally, and for infant death specifically from SIDS (Moffatt, et al., 1988). The problem remains, however, that most infants whose deaths are classified as SIDS do not have a pattern of general or specific risk(s) which can be identified that will predict their death (Krongrad, 1991).

CHAPTER VI

POLICY IMPLICATIONS AND RECOMMENDATIONS

Historical Perspectives on Government Policy on SIDS

In 1986, Abraham Bergman published a book on the politics of SIDS in the U.S. entitled The "Discovery" of Sudden Infant Death Syndrome: Lessons in the Practice of Political Medicine. The book describes how government policy on SIDS in the U.S. was developed. Bergman also describes how influential political figures and parents of children who were victims of SIDS formed a coalition which brought SIDS to the attention of policy makers as a public health issue. This coalition eventually developed and passed legislation on SIDS leading to the joint resolution in the Senate and House of Representatives in 1972 on SIDS. Bergman then discussed how the rule making or regulation propagating phase of PL 93-270 was influenced. One emphasis was to expand funding for biomedical research in SIDS. The SIDS program which resulted from this effort still operates within the National Institute of Child Health and Development. The primary emphasis of the biomedical research was to identify features of SIDS that would be useful in the prevention of the event.

Elements of Prevention

An essential feature of an effective prevention effort is the identification and definition of the outcome event of interest (Halperin, et al., 1992). Death is a very reliable

event with an understandably high degree of agreement between observers. However, as previously stated, a number of problems with the current diagnostic criteria for SIDS do continue to cloud the development of an unambiguous definition of SIDS. A second essential feature of successful prevention efforts is the identification of those factors which predispose to the event under study, and which are amenable to change (Halperin, et al., 1992).

In the largest study of SIDS ever completed, the National Institute of Child Health and Development identified risk factors for SIDS which included maternal risk factors, for example, mothers' age <20 (O.R. = 3.3), as well as newborn risk factors, such as birthweight <1500 g. However, such risk factors were found in only 4.5% of the SIDS infants studied by Hoffman, et al. (1988). In general, risk factors with a relative risk of 3.0 or higher are very difficult to identify, and risk factors above 4 are seldom present in more than 10% of SIDS infants (Hoffman, et al. 1988). The study by Schoendorf, et al. (1992) demonstrated that most SIDS deaths occur in infants with very few risk factors, and in those infants who have high rates of risk factors, the diagnoses of SIDS is often questionable.

This represents a research paradox. As we continue to learn more about risk factors for SIDS this information is typically used to classify fewer infants as having died of SIDS. As a result of the study population changes over time making comparisons of race and risk factors problematic.

Identification of Infants at High Risk for SIDS

Two essential features of SIDS are the sudden and unexplained nature of infant deaths. As a result no known risk factor can be used to predict an infant who may die of SIDS (Haas, et al., 1993). Ironically and predictably, Haas, et al. (1993) noted that more risk factors appear to be present in infant deaths when the diagnosis of SIDS is doubtful, whereas in the typical SIDS death very few risk factors are identifiable. Nearly all risk factors that have been identified for SIDS have later been shown to be influenced by confounding. Often, subsequent research eventually refutes the role of potential risk factors that have been previously identified in a causal chain for SIDS. Very few risk factors are associated with even a doubling of SIDS rates. This makes the establishment of a prevention program problematic, in the absence of clearly defined risk factors.

A Model for SIDS Surveillance

Surveillance also serves as an important component of existing SIDS programs. Klaucke (1992) identified six components of effective surveillance: (a) the total number of cases indicating incidence and prevalence; (b) incidence of severity such as mortality rate and the case fatality ratio; (c) an index of lost productivity such as bed disability days, (d) an index of premature mortality, such as years of potential life lost (YPLL); (e) medical costs; and (f) preventability. In the U.S. there were 5,510 deaths classified as SIDS in 1980 and 5,476 cases were reported in 1988 (MMWR, 1991). SIDS was the second leading cause of infant mortality, accounting for 14.2% of all infant mortality in 1989.

In order to contribute to preventive efforts against SIDS, the adoption of a surveillance model for all infant deaths is also advocated. The model proposed here has

been adapted from a model of a public health surveillance system by Kalucke (1992) (See Figure 2). The public health model has been modified to make it applicable for use in surveillance of infant deaths, and specifically for deaths due to SIDS. The original model has also been amended to incorporate a community review of sentinel health events, which in the case of SIDS would be defined as infant deaths. The deaths are reviewed to determine if the death was preventable. The community review approach implies that a preventable infant death is a warning signal that the quality of medical care or social support may need to be improved (Seligman & Frazier, 1992).

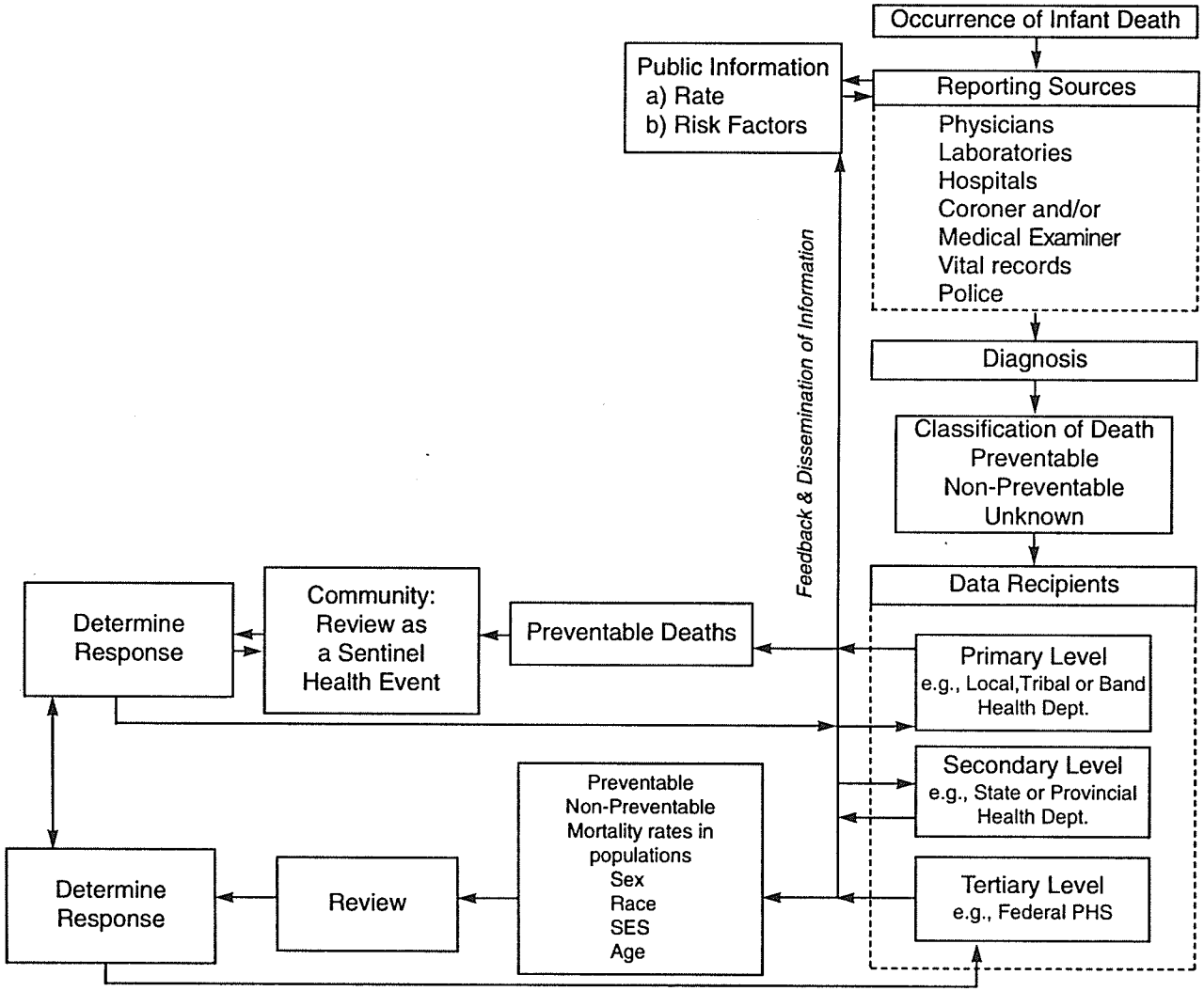
This is an especially important concept in SIDS, since deaths which occur during the post-neonatal period (from one month through one year) are thought to parallel socio-economic status and access to healthcare, while lower SES levels have long been associated with higher post-neonatal mortality rates (Pharoah, et al., 1979). Deaths during the post-neonatal period provide a useful opportunity for communities and institutions to review potential problem areas, and to ascertain if the identified conditions are modifiable.

The initial component of the surveillance model proposed here is the unexplained death of an infant. In Manitoba infant deaths must be reported to the MEO. The death may be reported by one of several sources: physicians, police departments, or emergency room personnel. After reporting, the deaths are reviewed by the MEO using a multidisciplinary committee including representatives from the MEO, Child Abuse Services, Police Department, Child and Family Services, a pathologist, and the Crown attorney. As necessary, other relevant experts are consulted. The next step in the model is the establishment of an accurate diagnosis, which in this situation is the diagnosis of a

Figure 2

A Public Health Surveillance System for Infant Mortality

modified from Halperin et al., 1992



case of SIDS. In Manitoba, the PDRC provides a process pathway for structured review and classification of infant deaths. Deaths would then be classified as preventable or non-preventable or don't know by the MEO and PDRC. The schema described by Honigfeld, et al. (1987) may prove useful in this classification. Data collected through this process would then be distributed to communties through local and regional health departments. To maximize utilization of data and comparisons across time, it is important to have a general category of sudden, unexplained infant deaths and a subgroup of children within that population who meet the current and changing criteria for Sudden Infant Death Syndrome. This would then allow development of a data set for comparison with longer term historic definitions of SIDS and would identify a population of children who meet current criteria for SIDS.

Systems Model for Prevention Efforts

The impact of this study resides in its potential of impacting future preventive programming priorities in the public health sector. The use of the information gathered from this study, as it relates to state-level planning of interventions for decreasing the prevalence of SIDS, can be framed within a systems model. From a conceptual framework perspective, systems function to process information into a planned outcome and can be described in terms of relationships among elements in reaching an objective (Arndt, 1980).

The data generated from this study can be conceptualized as an input element, connecting to the decision-making elements of the health care delivery system of Manitoba. The health care delivery system then generates decisions, policies, programs,

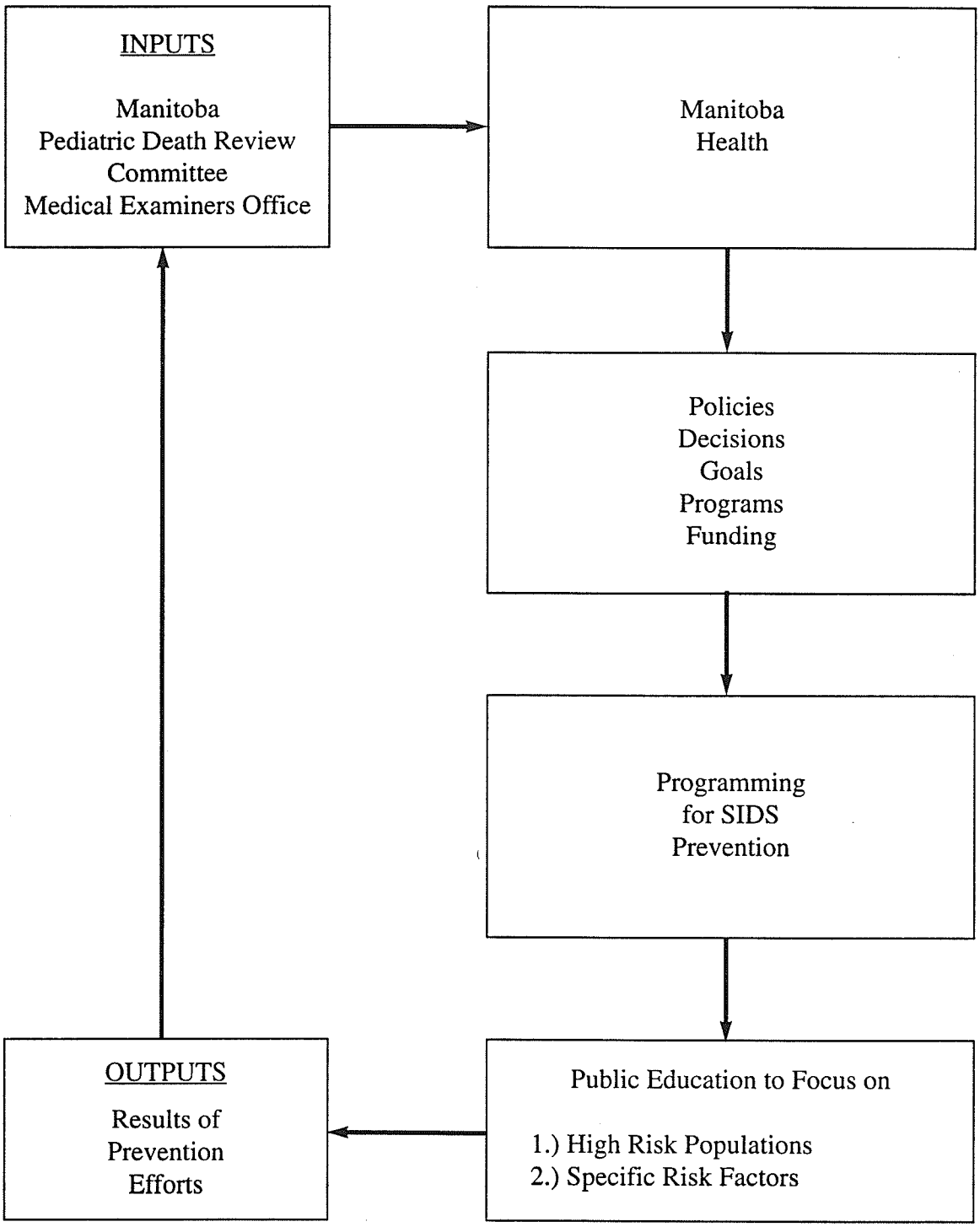
and funding. Outcomes generated by the programming element constitute "output", which again is transformed into further "input" returned into the system. An adapted schema of this process is presented in Figure 3 (Finney, et al., 1976).

Due to limited resources for prevention efforts in Manitoba it is important to identify risk factors that are amenable to implementation and that offer preventive potential. To this end, identified risk factors need to be prioritized to allow for a logical process of incorporation into the public health prevention system. Several factors have emerged in recent years as potential risk factors for SIDS (Moffatt, et al., 1988; Fleming, et al., 1990). Many of these appear to be risk factors with potential for inclusion into current prevention efforts.

Two examples of risk factors after which effective interventions are available and have been demonstrated to reduce risks of death are prone sleeping position and maternal smoking. In response to campaigns to reduce prone sleeping, rates of SIDS in several countries have fallen by as much as 70% (Dwyer et al., 1995). A reduction in rates of maternal smoking could also reduce the rates of SIDS. Bulterys et al. (1990) has provided data suggesting that 30% of all SIDS deaths in Native populations could be prevented by decreasing rates of maternal smoking during pregnancy.

Another useful example of this process would be to have health care providers discuss crib enviromental risk factors with the parents of all infants under their care. The use of a model crib and doll would be very helpful in illustrating appropriate infant care practices. A series of papers (Fleming, et al., 1990; Gilbert, et al., 1992; Kemp & Thach, 1991; Nelson et al., 1989) provide data which suggest t hat an effort such as this would

Figure 3
Theoretical Framework Adapted
from Arndt and Huckabay, 1980.



be useful in reducing the presence of SIDS risk factors. This effort could easily be undertaken as a routine part of both well child care or when infants are in for treatment of common illness. Other preventive measures that should be considered for inclusion into a preventive effort against SIDS are the four modifiable risk factors described by Mitchell, et al. (1992).

Another low cost but potentially important area for risk factor surveillance would be to add dose indicator information to the data currently being collected. One example of this strategy would be to add the 4 item T-ACE screening questions to the Maternal Nursing Database (Sokol et al, 1989). Two additional questions on the number of days per month women drink, and the average number of drinks per day would further enhance the utility of the alcohol data. Similar strategies for smoking would be important. The value of this information for Manitoba is enhanced by the nearly complete use of the Maternal Nursing Database in the province. It may be useful to consider adding these questions to birth certificates in areas where a system similar to the Maternal Nursing Database is not used.

System and Legislative Recommendations

Ongoing support for a death review team for all deaths under 18 years of age is important as well. The utility of child death review teams has been well established, with the particular strength of these teams being their ability to identify and classify infant deaths, and to provide a forum for multi-disciplinary and multi-agency discussion of child deaths (Durfee, et al., 1992). Funding for autopsies should also be provided to enhance the identification of modifiable risk factors for infant death.

While funding for autopsies is available in Manitoba, in many other areas adequate funding for autopsies is problematic. In these areas funding would be an important focus of efforts to improve identification of causal factors for infant death. Mandatory autopsies conducted by a pediatric pathologist using a standardized autopsy form would be of considerable importance in this effort. This increases dramatically both the quality and comprehensiveness of the data collected across personnel and as a result substantially increases the usefulness of the data which are collected. The current death review team in Manitoba by the PDRC and the MEO utilizes an investigation protocol which includes total body x-rays of the dead infant, full toxicology screens, metabolic screens, examination for sexual abuse and a death scene investigation by the Medical Examiner or trained designee. Death scene investigation is another area where the use of a standardized protocol will greatly facilitate the completeness of the death scene investigation and substantially enhance the usefulness of the data. Deaths are then reported to the PDRC and SIDS Foundation. The PDRC then conducts a separate investigation and provies an annual report on the infant and childhood death in the province of Manitoba. These deaths are examined for comprehensiveness of medical care, preventability, changing patterns in the deaths, for example, increased SIDS deaths in certain sub-populations or areas. This data then provides useful material for the legislative and executive branches of government to utilize in implementing and funding system changes. As problems with medical care are identified, the College of Physicians can then notify either individual practitioners or implement wider spread corrective action by publication of best practices papers for physicians and other health care workers.

Programs of comprehensive access to prenatal care have estimated that for every dollar invested in prenatal care, $2 to $10 in health care costs can be saved (Racine, et al., 1992). This is especially important for the prevention of deaths in the PNM period, since these deaths are related to quality and access to health care for both mothers and infants (Mitchell, 1990). Beyond quality and access, serious attention would also have to be given to the involvement of high risk mother/child dyads in preventive programs, since having programs operational and having these programs utilized by women in the high risk groups for infant death are not necessairly synonomous.

The final step in this model of prevention, is for the community review of individual deaths to determine both "what went wrong" and "what can be changed" to prevent similar deaths in the future. This area is one of the most difficult to implement, since nearly all the funding and change resources are controlled at regional or national levels. An example of an effort of this type which has not yet been evaluated, but which appears to be a move in this direction conceptually is the Healthy Start project underway in the United States (Strobino et al., 1995). Communities with very high rates of IMR were selected and funded to implement community-developed programs to reduce IMR. The model described in figure 3 also provides for agency review of infant deaths if a change in incidence rates (or some other event) suggests that a change in the distribution of the deaths has occurred, and requires further investigation. Lastly, further funding of basic research activities need to be continued with some monies specifically earmarked for SIDS.

Appendix 1

This is a copy of the Maternal Nursing Database which is used in hospitals in Manitoba.

The form is completed by the nursing staff at the time of admission to the hospital.

Maternal Nursing Database

PRESENT PREGNANCY

First language _____
Second language interpreter _____

"Presenting Problem _____

Previous Admissions/Problems this Pregnancy

Bleeding _____ 1st Trimester
_____ 2nd _____ 3rd
Risk score ☐☐

Prenatal Care Yes ☐ No ☐
G _____ P _____ EDC ☐☐☐ (day mo. yr.) WKS _____
LNMP ☐☐☐ (day mo. yr.) Reg ☐ Irreg. ☐
Ultrasound No ☐ Yes ☐
EDC by Ultrasound ☐☐☐ (day mo. yr.)
Doctor's Office ☐☐☐ (day mo. yr.) FAU ☐☐☐ (day mo. yr.)

Rh Last WinRho Inj. ☐☐☐ (day mo. yr.)
Previous blood transfusions No ☐
Yes ☐ ☐☐☐ (day mo. yr.)

Prenatal Classes: Yes ☐ No ☐

Breastfeeding: Yes ☐ No ☐ Undecided ☐
Previous Experience: Yes ☐ No ☐

Exposures
Smoking _____ packs/day
Alcohol (amount) _____
Other drugs _____
Communicable diseases _____

Allergies ☐ None Known
Food _____
Drug _____
Other _____
Type of reaction _____

Surgical HX _____

HISTORY OF PREVIOUS PREGNANCIES

No.	Year	Sex	Gest. Age (Wks.)	Birth Weight	Dur. of Labour	Place of Birth	Comments: re Pregnancy, Type of Delivery, Anomaly, Perinatal Death, etc.

REVIEW OF SYSTEMS

Respiratory ☐ No difficulty

Neurological ☐ No difficulty
Seizure disorder _____
Migraines _____

Musculo-Skeletal ☐ No difficulty
Pre-gravid weight _____
Present weight _____ Height _____

Vision/Hearing ☐ No difficulty
Glasses ☐ Contacts ☐ Hearing aid ☐

Skin ☐ No difficulty

Cardiovascular ☐ No difficulty
Ess. hypertension _____
PIH _____ Edema _____
Heart disease _____
Varicosities _____ Hemmorrhoids _____

Gastro-Intestinal ☐ No difficulty
Dentures _____
Dietary restrictions _____
Constipation _____ Diarrhea _____
Jaundice _____ Hepatitis B _____

Genito-Urinary ☐ No difficulty
UTI _____ Kidney disorders _____
Vag. discharge _____ STD _____

Endocrine ☐ No difficulty
Diabetes _____
Thyroid disorder _____

PSYCHOSOCIAL ASSESSMENT

Present emotional status _____

Support person _____
Initial response to this pregnancy _____

Planned pregnancy Yes ☐ No ☐
Type of contraception _____
Hx of postpartum depression _____
S M W Sep. Div. C.L.
Keeping baby: Yes ☐ No ☐
Infant's physician _____
No. of children at home _____
Child care _____
Occupation _____
Returning to work: Yes ☐ No ☐
Economic concerns: Yes ☐ No ☐

Recent family stress _____

Referral to social worker needed:
Yes ☐ No ☐ To be reassessed ☐
Involved with: _____
Name of social worker _____
City welfare ☐ Prov. welfare ☐
Treaty # _____

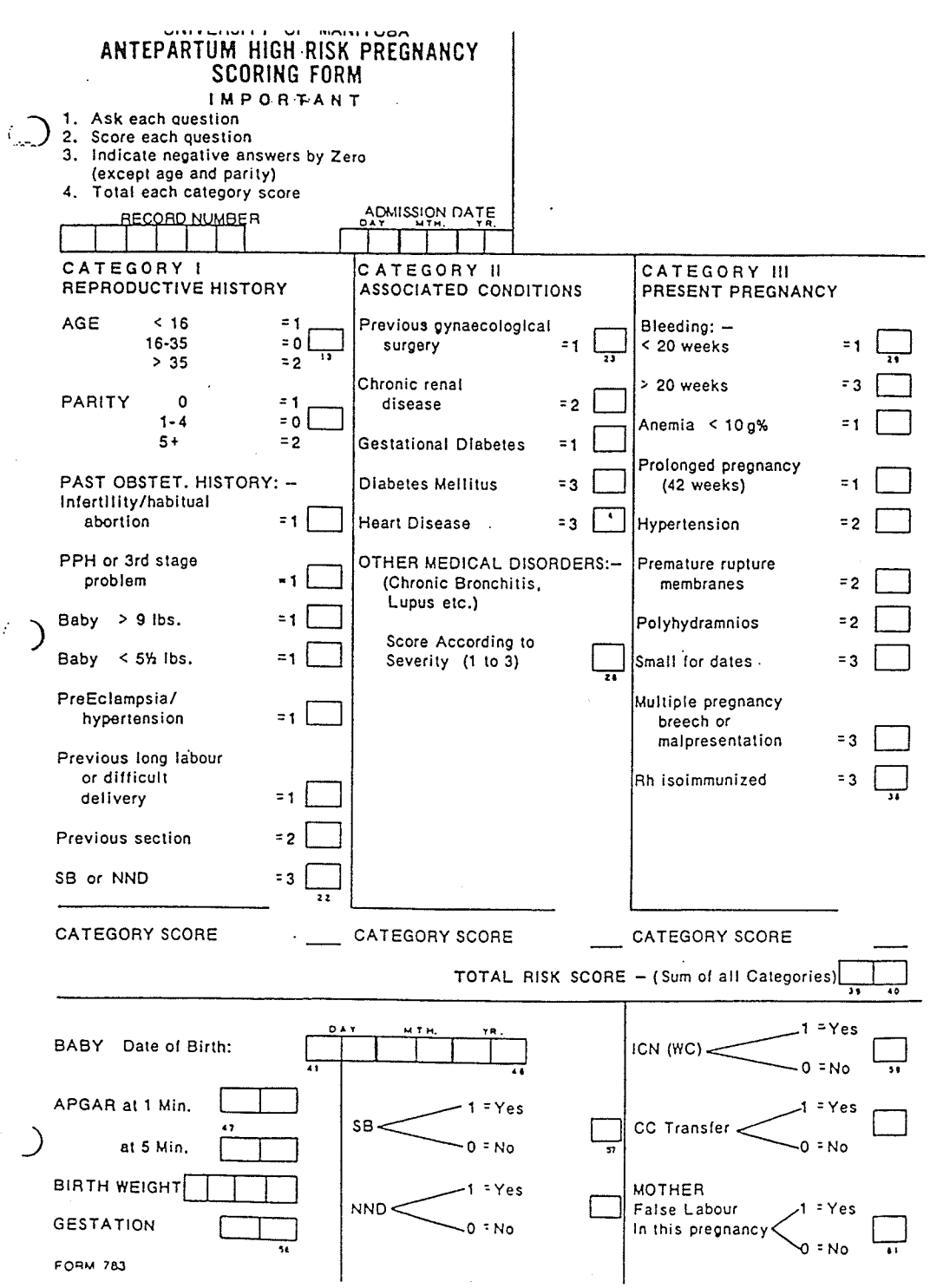
COMMENTS

Date reviewed _____
Signature _____

FORM 82251 ORIGINAL - WHITE COPY — To Mother's Chart YELLOW COPY — To Infant Chart October, 1988

Appendix 2

The Antepartum High Risk Pregnancy Scoring Form is completed by the nursing staff when a women is admitted for delivery.



UNIVERSITY OF MANITOBA

ANTEPARTUM HIGH RISK PREGNANCY SCORING FORM

IMPORTANT

1. Ask each question
2. Score each question
3. Indicate negative answers by Zero (except age and parity)
4. Total each category score

RECORD NUMBER

ADMISSION DATE DAY MTH. YR.

CATEGORY I REPRODUCTIVE HISTORY

AGE < 16 =1; 16-35 =0; > 35 =2 (13)

PARITY 0 =1; 1-4 =0; 5+ =2

PAST OBSTET. HISTORY: – Infertility/habitual abortion =1

PPH or 3rd stage problem =1

Baby > 9 lbs. =1

Baby < 5½ lbs. =1

PreEclampsia/ hypertension =1

Previous long labour or difficult delivery =1

Previous section =2

SB or NND =3 (22)

CATEGORY SCORE ___

CATEGORY II ASSOCIATED CONDITIONS

Previous gynaecological surgery =1 (23)

Chronic renal disease =2

Gestational Diabetes =1

Diabetes Mellitus =3

Heart Disease =3 (24)

OTHER MEDICAL DISORDERS:– (Chronic Bronchitis, Lupus etc.)

Score According to Severity (1 to 3) (25)

CATEGORY SCORE ___

CATEGORY III PRESENT PREGNANCY

Bleeding: – < 20 weeks =1 (26)

> 20 weeks =3

Anemia < 10 g% =1

Prolonged pregnancy (42 weeks) =1

Hypertension =2

Premature rupture membranes =2

Polyhydramnios =2

Small for dates =3

Multiple pregnancy breech or malpresentation =3

Rh isoimmunized =3 (28)

CATEGORY SCORE ___

TOTAL RISK SCORE – (Sum of all Categories) (39) (40)

BABY Date of Birth: DAY MTH. YR. (41) (46)

APGAR at 1 Min. (47)

at 5 Min.

BIRTH WEIGHT

GESTATION (56)

SB 1 =Yes 0 =No

NND 1 =Yes 0 =No

(57)

ICN (WC) 1 =Yes 0 =No (59)

CC Transfer 1 =Yes 0 =No

MOTHER False Labour In this pregnancy 1 =Yes 0 =No (61)

FORM 783

Appendix 3

Variable Descriptions

A description of each of the variables included in the data set which were included in the analysis follows:

CASENO: These were assigned at the time of data entry. One number was assigned to each subject.

DEATHYR: The year of death for each subject in the SIDS and the non-SIDS deaths control group.

SEX: Coded as 1 = males, 2 = females.

AGE: The age of the dead subjects in days. The range is from 14 to 363 days. Subjects were selected if they were 14 days of age and less than 365 days of age at time of their death.

DOB: The date of birth for the study subjects.

RACE: The study subjects were assigned to two groups: 1 = white/other, 2 = aboriginal. Subjects who were neither white nor aboriginal were assigned as white, since this is the larger group, and it was not feasible during analysis to have additional groups with very small numbers.

DATEDTH: The date of death for subjects in the two death groups (SIDS and non-SIDS)

HOSPITAL: The hospital of birth for all subjects. This data was obtained from the Division of Vital Records for the province, and for some subjects by searches of the hospital birth records. Coded from 1-23.

L&DCOMPL: This variable was used to record complications at labor or delivery that were extracted from the mothers' medical records. Coded as: 0 = none, 1 = multiple pregnancy, 2 = C-section, 3 = pre-eclampsia, 4 = prematurity, and 5 = other.

BIRTWT: Birthweights in grams from the infants' medical charts. Where different birthweights were listed, the average of the two weights was used. Coded from 410-4790.

APGAR1: The one minute Apgar score from the infants chart. Coded from 0-10.

APGAR5: The five minute Apgar score from the infants chart. Coded from 0-10.

BIRTHORD: The birth order of the subject (of the total number of live births recorded for the mother). Coded as: 1= first, 2 = second, 3 = third, 4 = fourth, 5 = fifth, and 6 = sixth or more.

INFCOMPL: Any medical complications of the subjects at birth (taken from the infants' chart). Coded as: 0 = none, 1 = congenital malformation, 2 = preterm birth, 3 = respiratory distress, and 4 = other.

BIRTHSGL: Records the parity of this pregnancy. Coded as: 1 = single, 2 = twins, and 3 = triplets.

MOTHAGE: The mothers' ages at the time of delivery from the mothers' medical charts. Coded in years: 14-43. For the 2 X 2 tables the score was less than 20 and 20 or more years of age.

PREVPG: The number of previous pregnancies listed in the mothers' medical charts. Coded as: 0 = none, 1 = one, 2 = two, 3 = three, 4 = four, and 5 = five or more.

BIRDEAD: The number of live births that were subsequently listed as dead in the mothers' medical charts. Coded as: 0 = none, 1 = one, 2 = two, and 3 = three.

PGBEF21: The number of pregnancies for each mother before she was 21 years of age. Coded as: 0 = none, 1 = one, 2 = two, 3 = three, 4 = four, and 5 = five or more.

SMOKE: The smoking status of the mother during pregnancy. If this data was not available, the infant was not included in the study. Coded as: 1 = yes (mother smoked), and 2 = no.

CIGDAY: The number of cigarettes smoked by mothers per day. Coded as: 0 = none, 1 = less than one pack per day, and 2 = one or more packs per day.

ALCOHOL: The mothers' use of alcohol during pregnancy. Coded as: 0 = no, and 1 = yes. This data was primarily obtained from the nurses' reports or from the prenatal care record.

DIABETES: The mothers' diabetic status prior to pregnancy. Coded as: 1 = positive, and 2 = negative.

PRNAVISRF: A categorical variable developed for data analysis. Coded as: 0 = less than 5 prenatal care visits, and 1 = five or more prenatal care visits.

PLANPG: The status of the pregnancy as planned/unplanned. Coded as: 0 = yes, and 1 = no.

REPHIST: This score was the Manitoba Antepartum Risk Score. The score utilized was the last recorded score before delivery. Coded from 0-10.

ASSOCOND: This score was the Manitoba Antepartum Risk Score. Coded from 0-4.

PRESPGSC: This score was the Manitoba Antepartum Risk Score. Coded from 0-9.

TOTRISK: This was the total risk score for the Manitoba Antepartum Risk Score. Coded from 0-17.

DEPRESS: History of postpartum depression in the mothers' medical records. Coded as: 1 = yes, and 0 = no.

MARITAL: The marital status of the mother at the time of delivery. Coded as: 1 = married, 2 = single, 3 = separated, 4 = divorced, 5 = widowed, and 6 = living with partner.

PREVABOR: The number of abortions before 20 weeks of gestation (prior to this pregnancy). Coded as: 1 = yes, 0 = no, 0-2

ABOR+20: The number of abortions after 20 weeks gestation (prior to this pregnancy). Coded as: 1 = yes 0 = no 0-2

GESTRF: The number of weeks of gestation. This data was from taken from the mothers' medical records. This was a derived categorical variable for data analysis. Coded as: 1 = less than 37 weeks, and 0 = 37 weeks or longer.

PRENAME: The month prenatal care was started. This data was taken from the prenatal care record. Coded from 1-9

PRENAVIS: The total number of prenatal visits. This data was taken from the prenatal record. Coded from 0-18.

PRECLASS: Attendance of mother at prenatal class(es) before the birth of this child. Coded as: 0 = yes, and 1 = no.

BRSTFED: Breastfeeding status of the infant prior to discharge from the hospital. This data was taken from the nurses notes and the Manitoba Maternal Nursing Database. Coded as: 0 = yes, and 1 = no.

PROTSER: Evidence of contact or referral to Social Services indicated by the nursing database or by the physician notes. Coded as: 1 = yes, and 0 = no.

CHLDHOME: The number of children reported to be living at home from data on the Maternal Nursing Database. Coded from 0-7.

PREDEATH: History of a previous SIDS, infant or child death reported in the family? Coded as: 1 = positive history, and 0 = negative history.

WKGEST: The reported length of gestation in weeks. Data taken from the Manitoba Maternal Nursing Database, the infants' medical records, or from the mothers' medical records. Coded from 22-43.

PDRCSIDS: Classification of death as SIDS by the PDRC. Coded as: 1 = yes, and 0 = no.

MEOSIDS: Classification of death as SIDS by the Medical Examiners Office. Coded as: 1 = yes, and 0 = no.

BOTHSIDS: Classification of death as SIDS by both the PDRC and the MEO. Coded as: 1 = yes, and 0 = no.

SEASBRTH: The season during which birth occurred. Coded as: 1 = DOB in December, January or February; 2 = DOB in March, April or May; 3 = DOB in June, July or August; and 4 = DOB in September October or November. This classification system is used by the PDRC.

SEASDEATH: The season during which death occurred. Coded as: 1 = death in December, January or February; 2 = death in March, April or May; 3 = death in June, July or August; and 4 = death in September, October or November.

The remaining variables were categorical variables derived for use in the analytic portion of the study and are defined below;

PRABRF Subjects whose mothers medical records listed previous abortions before 20 weeks of gestation. 1 = >abortion, 0 = >no abortion.

PRAB20RF Subjects whose mothers medical records listed previous abortions after 20 weeks gestation. 1 = >abortion, 0 = >no abortion recorded in the record.

PRNAMORF Subjects whose mothers were reported to have started prenatal care after the third month of pregnancy. 1 = >prenatal care started after 3 months, 0 = > prenatal care started in the first three months.

PRCLASSRF: A derived variable for data analysis. 1 = no prenatal, 0 = prenatal class.

BRSTFEDRF: A derived variable for data analysis. 1 = not breastfed 0 = was breastfed.

PRTSERRF: A derived risk factor for data analysis. 1 = protective service was notified 0 = not notified.

L&DCoRF:	1=> L&DCompl>=1,	0=> L&DCompl=0
BtWt25RF:	1=> BirthWt<2500,	0=> BirthWt>=2500
BtWt15RF:	1=> BirthWt<1500,	0=> BirthWt>=1500
Apg1RF:	1=> Apgar1<5,	0=> Apgar1>=5
Apg5RF:	1=> Apgar5<7,	0=> Apgar5>=7
CompLRF:	1=> CompL>=1,	0=> CompL=0
SingBtRF:	1=> BirthSgl>=2,	0=> BirthSgl=1
MoageRF:	1=> MothAge<20,	0=> MothAge>=20
ParityRF:	1=> PrevPg>2,	0=> PrevPg<=2
LBDeadRF:	1=> BirDead>=1,	0=> BirDead=0
PgBe21RF:	1=> PgBef21>=1,	0=> PgBef21=0
SmokeRF:	1=> Smoke=1,	0=> Smoke=0
AlcoholRF:	1=> Alcohol=1,	0=> Alcohol=0
DiabRF:	1=> Diabetes=1,	0=> Diabetes=0
PlPrgRF:	1=> PlanPg=0,	0=> PlanPg=1
RepHstRF:	1=> RepHist>=1,	0=> RepHist=0
AssConRF:	1=> AssoCond>=1,	0=> AssoCond=0
PrPgScRF:	1=> PresPgSc>=1,	0=> PresPgSc=0
TotRskRF:	1=> TotRisk>=1,	0=> TotRisk=0
DeprRF:	1=> Depress=1,	0=> Depress=0
MaritRF:	1=> Marital>=2,	0=> Marital=1

REFERENCES

Abel E L., Sokol R J. (1987). Incidence of fetal alcohol syndrome and economic impact of FAS related anomalies. Drug Alcohol Depend 19:51-70.

Abel E L. (1990). Fetal Alcohol Syndrome. Oradell, NJ Medical Economics Books.

Abel E L. (1982). Fetal Alcohol Syndrome (Vol. II). Boca Raton, FL: CRC.

Abel E L. (1984). Fetal Alcohol Syndrome and Fetal Alcohol Effects. New York: Plenum Press,.

Abel E L, Sokol R J., (1986). Fetal alcohol syndrome is now leading cause of mental retardation. Lancet II 1222.

Abel E L. (1988). Fetal alcohol syndrome in families. Neurobehav Toxicol Teratol 10:1-2.

Adams M M. (1985). The Descriptive Epidemiology of Sudden Infant Deaths among Natives and Whites in Alaska. Am J Epidemiol 122:637-43.

American Association for Study and Prevention of Infant Mortality. (1911). Transactions of the Second Annual Meeting. Baltimore, MD: Press of the Franklin Printing Co.

Anonymous. (1983). Alcohol and the fetus--is zero the only option? Lancet 1:682.

Arndt C, Huckabay L M D. (1980). Nursing Administration in Theory for Practice with a Systems Approach (2nd ed.). St. Louis, MO: C. V. Mosby Co.

Arntzen A, Moum T, Mangus P, Bakketeig, L S. (1995). Is the higher postneonatal mortality in lower social status due to SIDS? Acta Paediatr 84:188-192.

Balarjan, R., Raleigh, V. S., Bolling, B. (1989). Sudden Infant Death Syndrome and Postneonatal Mortality in Immigrants in England and Wales. BMJ 298:716-720.

Bass, D O, Kravath R E, & Glass L. (1986). Death-scene investigation in sudden infant death. NEJM, 315:100-105.

Bass, M. (1989). The fallacy of the simultaneous sudden infant death syndrome in twins. Am J Forensic Med Pathol 10(3):200-205.

Bass M, Hass R. (1991). Sudden Infant Death In Thailand and Alaska. Lancet 337:304-305.

Bergeson, M., et al. (1993). Linking multiple data sources in fetal alcohol syndrome surveillance-Alaska. MMWR Morb Mortal Wkly Rep. Vol 42:312-314.

Bergman A B. (1986). The Discovery of Sudden Infant Death Syndrome: Lessons in the Practice of Political Medicine. Prague, NY.

Bergman A B, Ray, G, Pomeroy MA, Wahl PW, Beckwith JB. (1972). Studies of the sudden infant death syndrome in King County, Washington: Epidemiology. Pediatrics 49:860-870.

Bergman A B, Wiesner L A. (1976). Relationship of passive cigarette smoking to sudden infant death syndrome. Pediatrics 58:605-668.

Blok J H. (1978). The incidence of Sudden Infant Death Syndrome in North Carolina's: Cities and counties: 1972-1974. Am J Public Health 68:367-372.

Bolumar F, Rebagliato M, Hernandez-Aguado I, Florey CD (1994). Smoking and drinking habits before and during pregnancy in Spanish women. J Epidemiol Community Health 48:36-40.

Bonati M, Fellin G. (1991). Changes in smoking and drinking behaviour before and during pregnancy in Italian mothers: implications for public health intervention. Int J Epidemiol 4:927-932.

Bonthius, D J, West J R. (1991). Permanent neuronal deficits in rats exposed to alcohol durng the brain growth spurt. Teratology 44:147-613.

Bray D L, Anderson P D. (1989). Appraisal of the Epidemiology of Fetal Alcohol Syndrome among Canadian native peoples. Can J Public Health 80: 40-45.

Bulterys M, Greenland S, Kraus J F. (1989). Chronic fetal hypoxia and sudden infant death syndrome: Interaction between maternal smoking and low hematocrit during pregnancy. Pediatrics 86:535-540.

Bulterys M. High incidence of sudden infant death syndrome among northern Indians and Alaska natives compared with southwestern Indians: possible roles of smoking. J Comm Health 1990; 15: 185-94.

Bulterys M. (1993). Passive tobacco exposure and sudden infant death syndrome. Pediatrics, 92:505-506.

Bulterys M, Morganstern H, Welty TK, Kraus JF. (1990). The expected impact of smoking cessation program for pregnant women on infant mortality among Native Americans. Am J. Prev Med 6:267-275.

Burd, L. (1994). Prevalence of prone sleeping position and selected infant care practices of North Dakota infants: a comparison of whites and Native Americans. Public Health Rep. 109(3):446-449.

Burd, L., Gregory J.M., Ford, R (1994) Incidence rates and Risk Factors for SIDS. Federal Practioner. 11:53-63.

Burd L, Moffett MEK. (1994). Epidemiology of Fetal Alcohol Syndrome in American Indians, Alaska Natives and Canadian Aboriginal Peoples: A Review of the Literature. Public Health Rep 109:688-693.

Campbell DT, Stanley JC. (1963). Experimental and Quasi-Experimental Designs For Research. Houghton Mifflin Co. Boston.

Canadian Institute of Child Health. (1990). The Health of Canada's Children. ACICH Profile, Canadian Institute of Child Health, Ottowa, Canada.

Carrol J L., Loughlin G M. (1993) Sudden Infant Death Syndrome. Pediatrics in Review. 14:83-93.

Chávez, G F., Cordero, J F., and Becerrant J E. (1988). Leading major congenital malformations among minority groups in the United States 1981-1986. MMWR Morb Mortal Wkly Rep 37:17-24.

Christensen, R. (1990). Health problems among Alaskan Eskimo infants and young children. Arctic Med Res 49:63-67.

Church M W, Gerkin K P, McPherson E W. (1986). The incidence of sudden death syndrome among the older siblings of children with fetal alcohol syndrome. Alcoholism: Clin Exp Res 10:93 (abstract).

Davies D P. (1984). Cot death in Hong Kong : a rare problem. Lancet ii 1346 - 1349.

Davis N, Sweeney L B, Peterson D R. (1988). Epidemiologic Comparisons of Sudden Infant Death Syndrome with Infantile Apnea. Ann NY Acad Sci 533:450-453.

Dehan M. (1988). Thoughts on ethical problems in neonatal and pediatric resuscitation. Presse Med. 17:503-508.

DiNicola A F. (1985). Might excessive maternal alcohol ingestion during pregnancy be a risk factor associated with an increased likelihood of SIDS. Clin Pediatr 24:659.

Drews C D, Kraus J F, Greenland S. (1990). Recall bias in a case-control study of SIDS. Int J Epidemiol 19:405-411.

DiFranza J J. (1995). Effect of maternal cigarette smoking on pregnancy complications and sudden infant death syndrome. J Fam Pract, 40(4):385-94.

Duimstra C, Johnson D, and Kutsch C., et al. (1993). A fetal alcohol syndrome surveillance pilot project in American Indian communities in the Northern Plains. Public Health Rep. 103:225-229.

Durfee M J, Gellert G A, Tilton-Durfee D. (1992). Origins and Clinical Relevance of Child Death Review Teams JAMA, 267:3172-3175.

Dwyer T, Ponsonby A L B, Blizzard L, Newman N M, & Cochrane J A. (1995). The Contribution of Changes in the Prevalence of Prone Sleeping Position to the Decline in Sudden Infant Death Syndrome in Tasmania. JAMA; 273(10):783-789.

Emery J L . (1989). Is sudden infant death syndrome a diagnosis? Or is it just a diagnostic dustbin? BMJ, 299:1240.

Filley A, House R, Kerr S. (1976). Managerial Process and Organizational Behavior (2nd ed.).

Fleming P J, Filbert R, Azaz Y, Berry P J, Rudd P T, Stewart A, Hall E. (1990). Interaction between bedding and sleeping position in the sudden infant death syndrome: a population based case-control study. BMJ 301:85-89.

Fleshman J K, Peterson D R. (1977). The Sudden Infant Death Syndrome among Alaskan natives. Am J Epidemiol 105:555-558.

Fox H E, Steinbrecher M, Pessel D, Inglis J, Medvid L, & Angel E. (1978). Maternal ethanol ingestion and the occurrence of human fetal breathing movements. Am J Obstet Gynecol 132:354-358.

Friede A, Rhodes R H, Guyer B, Binkin N J, Hannan M T, & Hougue C J. (1988). The postponement of neonatal deaths into the postneonatal period: evidence from Massachusetts.Am J Epidemol 127:161-170.

Frost F. & Shy, K K. (1980). Racial differences between linked birth and infant death records in Washington state. Am J Pub Hlth, 70:974-976.

Gantley M, Davies D P, & Murcatt A. (1993). Sudden infant death syndrome: links with infant care practices. BMJ 306:16-20.

Gilbert R, Rudd P, Berry P J, et al. (1992). Combined effect of infection and heavy wrapping on the risk of sudden infant death. Arch Dis Child 67:171-7.

Guntheroth W G, Spiers P S. (1992). Sleeping prone and the risk of Sudden Infant Death Syndrome. JAMA 267:2359-2362.

Guntheroth W G & Spiers P S. (1990). Bedding and sleeping position in the sudden infant death syndrome. BMJ 301:494.

Guntheroth W G. (1989). Crib Death: The Sudden Infant Death Syndrome. New York: Futura Publishing Co.

Guntheroth, W G. (1982). Crib Death: The Sudden Infant Death Syndrome. New York: Futura Publishing Co.

Haas J E, Taylor J A, Bergman A B ,et al. (1993). Relationship Between Epidemiologic Risk Factors and Clinicopathologic Findings in the Sudden Infant Death Syndrome. Pediatrics 91:106-112.

Haglund B. (1993). Cigarette smoking and sudden infant death syndrome: some salient points in the debate. Acta. Paediatr. Suppl 389:37-39.

Hahn R A. (1992). The state of federal health statistics on racial and ethnic groups. JAMA 267:268-271.

Hahn R A, Mulinare J, & Teutsch S M. (1992). Inconsistencies in coding of race and ethnicity between birth and death in U.S. infants: A new look at infant mortality, 1983 through 1985. JAMA 267:259-263.

Halperin W & Baker E L. (1992). Public Health Surveillance. NY Von Nostranad Reinhold.

Harrison H. (1984). Infant Apnea Syndromes: Part I. Alaska Med 26:104-108.

Havlicek V, Childaeva R, Chernick V. (1977). EEG Frequency spectrum characteristics of sleep states in infants of alcoholic mothers. Neuropaediatrie 8:360-373.

Hayward J D & Alessio D J. (1990). SIDS: Race as a factor. Wis Med J 89:11-14.

Heller J, Anderson H R, Bland J M, Brooke O G, Peacock J L, & Stewart C M. (1981). Alcohol in pregnancy: Patterns and association with socio-economic, psychological and behavioral factors. Brit J Addict 83:541-551.

Helweg-Larson K., Knudsen L B, Gregersen M, & Simonsen J. (1992). Sudden infant death syndrome (SIDS) in Denmark: evaluation of increasing incidence of registered SIDS in the period 1972 to 1983 and results of a prospective study in 1987 throught 1988. Pediatrics 89:855-859.

Healthy People 2000. (1992). National Health Promotion and Disease Prevention Objectives. US Government Printing Office. Jones and Barttell Pub Co. Boston, MA.

Henry M M, Getzen E S, & McNeill-Harkins K A. (1984). Update - The Sudden Infant Death Syndrome in North Carolina. North Carolina Medical Journal 45:543-546.

Hoffman H J, D'Amus K, Hillan L,& Krongrad E. (1988). Risk factors for SIDS. Results of the National Institute of Child Health and Development SIDS Cooperative Epidemiological Study. Ann NY Acad Sci 533:13-30.

Hoffman H J, Hunter J C, Ellish N J, et al. (1988). Adverse reproductive factors and the sudden infant death syndrome. In Harper RM, Hoffman HJ (eds). Sudden Infant Death Syndrome: Risk Factors and Basic Mechanisms. New York PMA publishing Group.

Hogue C J R.& Hargraves M A. (1993). Class, race and infant mortality in the United States. Am J Pub Hlth, 83:9-11.

Honigfeld L S & Kaplan D W. (1987). Native American post-neonatal mortality. Pediatrics; 80(4):575-578.

Illing E M & Kaiserman MJ. (1995) Mortality Attributable to Tobaccco Use in Canada and its Regions,1991. Can J of Public Health. 86:257-265.

Indian Health Service. (1990). Trends In Indian Health 1990. Washington, D.C.: U.S. Government Printing.

Jones K L & Smith D W. (1973). Recognition of the Fetal Alcohol Syndrome in Infancy. Lancet II: 999-1001.

Kandall S R & Gaines J. (1991). Maternal substance use and subsequent sudden infant death syndrome (SIDS) in offspring. Neurotoxicol-Teratol. 13(2):235-40.

Kaplan D W, Bauman A E, & Krous H F. (1984). Epidemiology of Sudden Infant Death Syndrome in American Indians. Pediatrics 74:1041-1046.

Kemp J S & Thach B T. (1991). Sudden infant death in infants sleeping on polystyrene-filled cushions. NEJM, 324:1858-1864.

Kesby G, Parker G S, & Barrett E. (1991). Personality and coping style as influences on alcohol intake and cigarette smoking during pregnancy. Med J Aust 155: 229-233.

Kinney H. C, Filiano J J, & Harper R M. (1992). The Neuropathology of the Sudden Infant Death Syndrome. A Review. J. Neuropath and Expt Neurol 51:115-126.

Kinney H C, Filano J J, Sleeper L A, Mandell F, Valdes-Dapenu M, & White, F. (1975). Decreased Muscarinic receptor binding in the Arcuate Nucleus in Sudden Infant Death Syndrome. Science 269:1446-1450.

Klaucke D L. (1992). Evaluating Public Health Surveillance Systems In: Halperin W. Baker E.L. (eds.) Public Health Surveillance NY Von Nostrand Reinhold pp 26-39.

Kleinman J C & Kiely J L. (1990). Postneonatal Mortality in the United States: An International Perspective. Pediatrics. 86:1091-1097.

Klonoff-Cohen H S, Edelstein S L, Lefkowitz E S, Srinivasan I P, Kaegi D, Chang J C, & Wiley K J. (1995). The effect of passive smoking and tobacco exposure through breast milk on sudden infant death syndrome. JAMA 273(10):795-8.

Kraus J F & Borhami N O. (1972). Post-neonatal sudden unexplained death in California: A cohort study. Am J Epidemiol 95:497-510.

Kraus J F & Bulterys M. (1991). The epidemiology of sudden infant death syndrome. In: Kiely M, ed. Reproductive and Perinatal Epidemiology. Boca Raton, FL: CRC Press Inc:219-249.

Lee N Y, Chan,Y F, Davies D P, Lau E, & Yip D C P. (1989). Sudden Infant Death Syndrome in Hong Kong:Confirmation of Low Incidence. BMJ 298;721.

Lewis P & Boylan P. (1979). Fetal breaching: A review. Am J Obstet Gynecol 134(5):587-598.

Li D K & Daling J R. (1991). Maternal smoking, low birthweight, and ethnicity in relation to sudden infant death syndrome. Am J Epidemiol 134:958-964.

Lum M K, Knutson L R, Hall D B, Margolis H S, & Bender T R. (1986). Decline in infant mortality of Alaskan Yupik Eskimos from 1960 to 1980. Public Health Rep 101: 309-314.

Malloy, M H & Hoffman J J. (1995). Prematurity, sudden infant death syndrome, and age of death. Pediatrics. 96:464-471.

Malloy M ., Hoffman H J, & Peterson D R. (1992). Sudden infant death and maternal smoking. Am J Pub Hlth. 82:1380-1382.

May P A, Hymbaugh K J, Aase J M, & Samet J M. (1983). Epidemiology of fetal alcohol syndrome among American Indians of the southwest. Soc Biol 30:374-387.

McKenna J J. (1986). An Anthropological Perspective on the Sudden Infant Death Syndrome (SIDS): The Role of Parental Breathing Cues and Speech Breathing Adaptions. Med Anthro 10:9-53.

McKenna J & Mosko S. (1990). Evolution and the Sudden Infant Death Syndrome (SIDS) Part III: Infant arousal and Parent-Infant Co-Sleeping. Human Nature 1: 291-330.

McLeod W, Brien J, Loomis C, Carmichael L, Probert C, & Patrick J. (1983). Effect of maternal ethanol ingestion on fetal breathing movements, gross body movements, and heart rate at 37 to 40 weeks' gestational age. Am J Obstet Gynecol 145(2):252-257.

Meadow R. (1990). Suffocation, recurrent apnea, and sudden infant death. J Pediatrics 117:351-357.

Millar W J & Hill G B. (1993). Prevalence of and risk factors for sudden infant death syndrome in Canada. Can Med Assoc J 149(5):629-635.

Mitchell E A, Ford R P K, Stewart A W, Taylor B J, Becroft, D MO, Thompson, J M D, Scragg R, Hassall I B, Barry D M J, Allen E M, & Roberts A P. (1993). Smoking and the Sudden Infant Death Syndrome. Pediatrics, 91:893-896.

Mitchell E A, Scragg L, & Clements M. (1995). Location of smoking and the sudden infant death syndrome (SIDS). Aust NZ J Med 25:155-56.

Mitchell E A, Steward AW, Scragg R, Ford R PK, Taylor B J, Becroft D M O, Thompson J M D, Hassall I B, Barry D M J, Allen E M, & Roberts A P. (1993). Ethnic differences in mortality from sudden infant death syndrome in New Zealand. BMJ 306:13-15.

Mitchell E A, Taylor B J, Ford R P, Stewart A W, Becroft D M, Thompson J M, Scragg, R, Hassall I B, Barry D M, & Allen E M. (1992). Four modifiable and other major risk factors for cot death: the New Zealand study. J. Paediatr. Child Health, 28 Suppl 1:S308.

Mitchell E.A. (1990). International Trends in Postneonatal Mortality. Arch Dis Child 65:607-609.

MMWR. (1992). Sudden Infant Death Syndrome -- United States, 1980-1988. 41(28):515.

MMWR. (1992). Infant Mortality -- United States, 1989. 41(5):81-85.

MMWR. (1992). 41:313-315.

MMWR. (1992). 41:18.

Moffatt M E K, Gray D, & Poslt BD. (1988). Sudden infant death syndrome in Manitoba: Confirmation of high rates for Indians. Artic Med Res 47: (Suppl 1) 526-528.

Morrison H I, Semenciw R M, Mao Y, & Wigle D T. (1986). Infant mortality on Canadian Reserves 1976-1983. Can J Public Health 77:269-73.

Nakamura, R M, King R, Kimball E H, Oye R K, & Helgerson S D. (1991). Excess infant mortality in an American Indian population, 1940 to 1990. JAMA, 266: 2244-2248.

Nelson E A S, Taylor B J, & Weatherall I L. (1989). Sleeping position and infant bedding may predispose to hyperthermia and the sudden infant death syndrome. Lancet, 8:199-201.

Nordstrom M, Cnattinguis S, & Haglund. (1993). Social Differences in Swedish Infant Mortality by Cause of Death 1983-1986. Am J. Pub Hlth 83:26-30.

Olegard R, Sabel K G, Aronsson M, et al. (1979). Effects on the child of alcohol abuse during pregnancy. Acta Paediatr Scand Suppl. 275: 112-121.

Oyen N, Bulterys M, Welty T K, & Kraus J F. (1990). Sudden unexplained infant deaths among American Indians and Whites in North and South Dakota. Ped Perinatal Epidemiol 4:175-183.

Pediatric Death Review Committee. Annual Report 1986. College of Physicians and Surgeons of Manitoba. Winnipeg, Manitoba.

Pediatric Death Review Committee. Annual Report 1987. College of Physicians and Surgeons of Manitoba. Winnipeg, Manitoba.

Pediatric Death Review Committee. Annual Report 1988. College of Physicians and Surgeons of Manitoba. Winnipeg, Manitoba.

Pediatric Death Review Committee. Annual Report 1989 & 1990. College of Physicians and Surgeons of Manitoba. Winnipeg, Manitoba.

Peterson L P, Leondardeson G, Wingert R I, Stanage W, Gergen J, & Gilmore H T. (1984). Pregnancy Complications in Sioux Indians. Obstet Gynecol 64: 519-23.

Peterson D R. (1984). Sudden infant death syndrome. In Perinatal Epidemiology, MB Bracken (Ed). Oxford University Press.

Pharoah P O D & Morris J N. (1979) . Postneonatal Mortality. Epidemiol Rev 1:170-183.

Pietrantoni M & Knuppel R A. (1991). Alcohol use in pregnancy. Clin Perinatol 18(1): 93-111.

Racine A D, Joyce T J, & Grossman M. (1992). Effectiveness of health care services for pregnant women and infants. The Future of Children 2:40-58.

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. Can Med Assoc J 137: 203-207.

Reed F W & McBroom W H. (1992). Implications of inaccurate infant mortality data. The IHS Primary Care Provider 17:29-32.

Sandor G, Smith D, MacLeod P, Tredwell S, Wood B, & Newman D. (1981). Intrinsic Defects in the Fetal Alcohol Syndrome: Studies on 76 cases from B.C. and the Yukon. Neurobehav Toxicol Teratol 3:145-152.

Savitt T. (1979). Social and medical history of crib death. The Journal of the Florida Medical Association 66(8):853-859.

Schlesselman J J. (1982). Case control studies: Design, conduct and analysis. Oxford University Press. NY.

Schoendorf K C, Houge C, Kleinman J C, & Rowley D. (1992). Mortality among infants of Black as compared with while college educated compared with while college educated parents. NEJM 326:1522-1526.

Schoendorf KC & Kiely J L. (1992). Relationship of sudden infant death syndrome to maternal smoking during and after pregnancy. Pediatrics 90:905-908.

Scragg R, Mitchell E A, Taylor B J, Stewart A W, Ford R P K, Thompson J M D, Allen E M, & Becroft D M O on behalf of the New Zealand Cot Death Study Group (1993). Bed sharing, smoking, and alcohol in the sudden infant death syndrome. BMJ 307:1312-18.

Seligman P J, Frazier T N. (1992). Surveillance: The Sentinel Health Event Approach. In: Halperin, W., Baker, E.L. (eds). Public Health Surveillance NY Von Nostrand Reinhoold pp 17-24.

Serdula M, Williamson D F, Kenrick J S, Anda R F, & Byers T. (1991). Trends in Alcohol Consumption by pregnant women. JAMA 265:876-79.

Sitthi-amorn C & Poshyachinda V. (1993). Bias. Lancet 342:286-288.

Single E W, Brewster J M, MacNeil P, Hatcher J, & Trainor C. (1995). The 1993 Geneal Social Survey I: Alcohol Use in Canada. Canad J Pub Health 86:397-401.

Smart R G. (1990). Health warning labels for alcoholic beverages in Canada. Can J Public Health 81:280-284.

Smialek J E & Lambros Z. (1988). Investigation of sudden infant deaths. Pediatrician 15:191-197.

Smialek J E, Smialek P Z & Spitz W U. (1977). Accidental bed deaths in infants due to unsafe sleeping situations. Clin Ped 16, 1031-1036.

Southall D P, Alexander J R, Stebbens V A, Taylor V G, & Janczynski R E. (1987). Cardiorespiratory patterns in siblings of babies with sudden infant death syndrome. Arch Dis Child 62:721-726.

Spector P E. (1985). Research Designs. Sage Publications. Beverley Hills CA

Spohr H L, & Steinhausen H C. (1987). Follow-up studies of children with Fetal Alcohol Syndrome. Neuropediatrics 18:13-17.

Steel R, Langworth J T. (1966). The relationship of antenatal and postnatal factors to sudden unexpected death in infancy. Can Med Assoc J 94:1165-72.

Steinschneider A L. (1972). Prolonged apnea and the sudden infant death syndrome: Clinical and laboratory observations. Pediatrics 50:646.

Stockwell E G, Swanson A, & Wicks W. (1988). Economic status differences in infant mortality by cause of death. Pub Hlth Rep 103:135-142.

Streissguth A P, Aase J M, Clarren S K, & Jones K L. (1985). Natural history of the fetal alcohol syndrome: A 10-year follow-up of eleven patients. Lancet II: 85-92.

Streissguth P A, Clarren S K, Randels S P, LaDue R A, & Smith D F. (1991). Fetal Alcohol Syndrome in Adolescents and Adults. JAMA 265:1961-1967.

Strobino D, O'Campo P, & Schoendorf K C. (1995). A Strategic Framework for Infant Mortality Reduction: " Implications for Healthy Start". Milbank Quarterly. 73:507-533.

Strychar I M, Griffith W S, & Conroy R F. (1990). The relationship among learning, health beliefs, alcohol consumption and tobacco use of prima gravidas. Can J Public Health 81:462-467.

Tasaki H,Yamashita M, & Miyazaki S. (1988). The incidence of SIDS in Saga Prefecture (1981-1985) Journal of the Pediatric Association of Japan 92;364-368.

Taylor J A & Sanderson M. (1995). A reexamination of the risk factors for the sudden infant death syndrome. Journal of Pediatrics. 126:887-891.

Taylor E M & Emery J L. (1990). Categories of preventable, unexpected infant deaths. Arch Dis Child 65:535-539.

Templeman C. (1892). Two hundred and fifty-eight cases of suffocation in infants. Edinburgh Medical Journal 38:322-34.

Torrez D J. (1990). Sudden Infant Death Syndrome In New Mexico (1975-1985). Ph.D. Dissertation: University of New Mexico, Albuquerque, NM.

U.S. Department of Health and Human Services. (1986). Black and Minority Health. Vol. VI: Infant Mortality and Low Birthweight. Washington, D.C.: U.S. Government Printing Office.

Valdes-Dapena M A. (1967). Sudden and unexpected death in infancy:A review of the world literature (1954-66). Pediatrics 39:123-138.

Vanlandingham M J, Buehler J W, Hogue C J R, & Strauss L T. (1988). Birthweight - specific infant mortality for Native Americans compared with Whites, six states. Am J Public Health 78:499-503.

Waterson E J, Evans C S, & Murroy-Lyon I M. (1990). Is pregnancy a time of changing drinking and smoking patterns for fathers as well as mothers? An initial investigation. Brit J Addict 85:389-396.

West J R. (1993). Acute and long-term changes in the cerebellum following developmental exposure to ethanol. Alcohol-Alcohol-Suppl 2:199-202.

West J R (Ed). (1986). Alcohol and Brain Development. New York: Oxford University Press.

Williams D R, Lavizzo-Mourey R, & Warren R C. (1994). The Concept of Race and Health Status in America. Public Health Reports; 109(1):26-41.

Willinger M, James L S, & Catz C. (1991). Defining the sudden infant death syndrome (SIDS):Deliberations of an expert panel convened by the National Institute of Child Health and Human Development. Ped Path 11:677-84.

Wilson E. (1990). Sudden Infant Death Syndrome in China. Lancet 336:1199-1200.

Young T K. (1983). Mortality Pattern of Isolated Indians in Northwestern Ontario: A 10-year Review. Public Health Rep 98:467-475.