THE EFFECTS OF MATERNAL SMOKING DURING PREGNANCY AND YOUNG MATERNAL AGE ON SUDDEN INFANT DEATH SYNDROME MORTALITY IN OREGON, 1991-1996

By

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CERTIFICATE OF APPROVAL

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TABLE OF CONTENTS

TABLES

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ABSTRACT

Sudden Infant Death Syndrome (SIDS) is a major cause of postneonatal death in the United States accounting for 2991 deaths in 1997 (CD Wonder). SIDS is especially troubling in Oregon, which consistently has had one of the highest SIDS rates in the country. In the early 1990s, several countries began national intervention programs to reduce SIDS rates by discouraging the use of the prone sleep position. In the United States, a national "Back to Sleep" campaign was initiated in 1994 to discourage the use of the prone infant sleep position and it has been successful in lowering SIDS rates and the prevalence of the prone sleep position (Willinger, et al., 2000). In 1997, the SIDS rate in the United States was 0.77 SIDS deaths per 1000 live births and in Oregon it was 0.86 deaths per 1000 live births (CD Wonder). Other important risk factors for SIDS include maternal smoking during pregnancy and young maternal age.

A retrospective cohort study was conducted to assess for changes in the epidemiology of SIDS that may have occurred in Oregon between 1991-1993 and 1994-1996. The first hypothesis of this study was that the population attributable risk percent (PAR%) for the effect of maternal smoking during pregnancy on SIDS mortality has increased from 1991-1993 to1994-1996. The second hypothesis of this study was that the risk of SIDS for infants born to younger mothers (<20 years old) did not decline as rapidly as the risk for infants of older mothers (≥20 years old).

Data on SIDS mortality and risk factors was obtained from Oregon birth certificate, matched birth/death certificate, and death certificate data sets. The adjusted PAR% for the effect of maternal smoking during pregnancy on SIDS mortality was calculated using the method presented by Bruzzi and colleagues (1985) after obtaining an

adjusted odds ratio estimated by multivariate logistic regression analysis. Multivariate logistic regression analysis was also used to test whether the risk of SIDS for infants born to younger mothers (<20 years old) did not decline as rapidly as the risk of SIDS for infants born to older mothers (≥20 years old).

We found that the adjusted PAR% for maternal smoking during pregnancy decreased from 36% in 1991-1993 to 28% in 1994-1996. The odds ratio for the association between SIDS mortality and maternal smoking during pregnancy versus none also decreased from 3.16 (95% CI 2.40, 4.14) in 1991-1993 to 2.59 (95% CI 1.85, 3.62) in 1994-1996.

We also observed that infants born to young mothers (<20 years old) in 1994-1996 were only 9% less likely to die of SIDS when compared to infants born to young mothers in 1991-1993 (OR=0.91; 95% CI 0.62, 1.33). In comparison, infants born to mothers 20-29 years old in 1994-1996 were 39% less likely (OR=0.61; 95% CI 0.46, 0.81) to die from SIDS compared to this group in 1991-1994. Finally, infants born to mothers >29 years old experienced a 44% decline in their risk of SIDS (OR=0.56; 95% CI 0.35,0.90) from 1991-1993 to 1994-1996.

These findings indicate that efforts should be continued to discourage maternal smoking during pregnancy. Reducing the prevalence of maternal smoking during pregnancy could prevent a significant number of SIDS deaths. In addition, the results of this study show that teen mothers have not experienced the large reductions in SIDS rates that older women have experienced. A focused effort to reach teen mothers may be necessary in order to more effectively deliver the "Back to Sleep" message to this population.

INTRODUCTION

The sudden, unexpected, and unexplainable deaths of apparently healthy infants have a written history dating back to biblical times when an infant's death in the night was attributed to overlaying by a co-sleeping mother (Russel-Jones, 1985). However, sudden infant death remained a largely unknown subject in the medical community until the 1960s, although important epidemiologic facts had been identified by that time. In the 1960s, parents of sudden infant death syndrome (SIDS) victims organized several parent groups in the United States and their efforts, in addition to an increased interest in studying SIDS by the National Institute of Child Health and Human Development (NICHD), resulted in two international SIDS conferences and a proliferation of major research projects (Valdes-Dapena, 1995). The first conference took place in 1963, the second in 1969. These conferences were seminal developments in SIDS research and the published proceedings of both conferences have been invaluable references ever since. An important accomplishment of the second conference in 1969 was the creation of the American term "sudden infant death syndrome " and the drafting of the first official American definition for SIDS: "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 of death" (Little and Peterson, 1990). This definition is still commonly used to define SIDS although more specific definitions have been proposed.

Because there are no clinically distinguishing characteristics of SIDS, it is a diagnosis made by exclusion and it can be difficult to distinguish between SIDS and other causes of death such as accidental suffocation and intentional smothering. Some have

proposed more restrictive definitions in order to refine the cases studied, although many experts support the continued use of the 1969 definition (Cordner and Willinger, 1995). The following definition has been adopted for research purposes in the United States:

> "Sudden infant death syndrome (SIDS) is the sudden death of an infant <1 year of age that remains unexplained after a complete postmortem investigation including autopsy, examination of the death scene, and review of the case history." (Cordner and Willinger, 1995)

The implementation of this restrictive definition is uneven, particularly in regards to the death scene investigation. It is possible that the use of the strict case definition may help to improve specificity in regards to SIDS diagnosis because some infant deaths due to causes such as accidental suffocation or Munchausen syndrome by proxy may be misclassified as SIDS deaths. However, a loss in sensitivity may result because the proper diagnosis of SIDS may not be made if all components of the definition are not met. For example, death scene investigations are not commonly performed internationally and not uniformly performed in the United States (Cordner and Willinger, 1995).

Some have questioned whether SIDS is really one disease or actually a term that encompasses several disease entities including misdiagnosed deaths. Some deaths that have been categorized as SIDS due to incorrect diagnoses include child abuse, acidemias, and fungal diseases. In fact, the original definition envisioned that SIDS represented a common final pathway of more than one disease process and was a syndrome in the proper sense of the word: a pattern of symptoms and signs without necessarily the same cause (Limerick, 1992).

Risk factors for Sudden Infant Death Syndrome

The epidemiology of SIDS has been thoroughly investigated and is abundantly represented in the literature. While the etiology of SIDS is unknown, researchers have assessed a wide range of risk factors such as race, age, maternal smoking, birth weight and infant sex. One of the major epidemiologic studies was conducted in the 1980s when the National Institute of Child Health and Human Development conducted an ambitious, large-scale research project, selecting SIDS cases from six centers across the country. 757 SIDS cases and 1514 controls were included in the study. Social workers interviewed the families of all the SIDS victims and controls. The findings were published in 1988 and presented an "analyses of risk factors and basic mechanisms" (Rognum, 1995). From this study and many others much has been learned about the risk factors for SIDS. The following section describes some of the more consistently reported and relevant risk factors.

Prone sleep position

Infant sleeping position is the most important epidemiologic finding to date in the study of SIDS. Prior to 1990, several studies had reported a substantial increase in the risk of SIDS for infants sleeping in the prone, or face down, position (Jonge, et al., 1989). In the late 1980s and early 1990s, population-based case-control studies on environmental factors were conducted in New Zealand, Australia, and the United Kingdom in hopes of explaining their high SIDS rates and all three studies reported strong associations between prone sleeping and SIDS (Fleming, et al., 1990; Mitchell, et al., 1991; Dwyer, et al., 1991).

The accumulating evidence that the prone sleep position increased the risk of SIDS prompted medical communities in several countries to advocate a nonprone sleep position, even as early as 1987 when the medical society for maternal and child care in the Netherlands recommended that infants be placed nonprone (Jonge, et al., 1989; Willinger, et al., 1994). Maternity and health care providers in Hordland County, Norway were encouraged to use a nonprone sleep position in early 1990 and the recommendations gained support from national medical societies and the national media. In 1991, campaigns were initiated in Tasmania and the rest of Australia to promote placing infants on their side to sleep. Also in 1991, the "National Cot Death Prevention Program" was begun in New Zealand which promoted infants sleeping on their backs, breast feeding and discouraged parental smoking and bundling of infants. A national intervention program titled "Back is Best" was started first in Avon and then the remainder of England in the fall of 1991 (Willinger, et al., 1998; Hunt, 1995).

The intervention programs resulted in immediate and dramatic reductions of 50% or more in the SIDS rates (Hunt, 1995). No increases in other causes of infant mortality were observed nor were there any decreases in any other important epidemiological risk factors, and it was concluded that sleep position was the most likely cause of the decreases in SIDS mortality. Based on the strength of the accumulated evidence, the American Academy of Pediatrics (AAP) officially recommended the use of nonprone sleeping positions in 1992, but it was not until 1994 that a national initiative was recommended following a meeting of experts held by the National Institute of Child Health and Human Development (Willinger, et al., 1994).

The national public education campaign "Back to Sleep" was initiated in late June 1994 under the joint sponsorship of the United States Public Health Service, American Academy of Pediatrics, and several SIDS advocacy groups (Willinger, et al., 2000). The "Back to Sleep" campaign is an ongoing project with the ultimate goal of reducing the prevalence of prone sleeping to 10% or less (Lesko, et al., 1998). Strategies used in the Back to Sleep campaign have included: 1) disseminating information to hospital nurseries and physicians, 2) targeting child care programs, and 3) initiating public media campaigns.

The mechanisms for the association between prone sleeping and SIDS have not been conclusively determined, but may be related to rebreathing of expired air in susceptible infants with impaired ventilatory and arousal responses. Animal studies conducted in the early 1990s support the hypothesis that prone sleeping places infants at risk of rebreathing their own expired air (Kemp, et al., 1996). It has also been reported that arousal mechanisms and airway protective mechanisms are more robust when infants sleep in the supine position versus the prone position (Jeffrey, et al., 1999). Prone sleeping may also be dangerous because infants have more episodes of quiet sleep and sleep for longer periods with fewer arousals (Oyen, et al., 1997). A review of published reports reveals that 20% to 50% of SIDS deaths occur in the prone sleeping position with the nose and mouth into bedding (Kemp, et al., 1996; Willinger, et al., 1994).

The prone sleep position may also increase the risk of SIDS by promoting hyperthermia. Infants sleeping in the prone position lose heat less efficiently. Hyperthermic conditions have been hypothesized to alter the response to hypercapnic or hypoxic conditions (Oyen, et al., 1997).

Studies have found that the prone position, along with sleeping on soft surfaces, overwrapping or swaddling of the infant, and excess bedding, produced a higher risk of SIDS than found with the use of the prone position alone (Ponsonby, et al., 1994; Kemp and Thach, 1991; Fleming, et al., 1990). In addition, several items of bedding have been shown to be associated with an increased SIDS risk and it is hypothesized that these softer items of bedding (comforters, sheepskins, pillows, quilts, porous mattresses) may limit carbon dioxide dispersal (Kemp, et al., 1996; Anonymous, 2000).

Several studies have assessed the impact of national interventions in various countries by examining changes in the prevalence of sleep position, SIDS incidence, and the prevalence of other risk factors. A Tasmanian cohort study found that the proportion of infants sleeping prone declined from 29.9% in the three years preceding the intervention to only 5.4% in the year following the intervention (Ponsonby, et al., 1994). Skadberg and associates reported that prone sleeping had declined from 74% to 43% among SIDS victims and from 64% to 8% among controls (Skadberg, et al., 1995). In addition, the SIDS rate had dropped from 3.5 SIDS deaths per 1000 live births to 0.3 SIDS deaths per 1000 live births (Skadberg, et al., 1995).

Maternal smoking

Maternal smoking has been consistently found to increase the risk of SIDS mortality numerous studies (DiFranza and Lew, 1995; Anderson and Cook, 1997; Mitchell, et al., 1993). In 1990, a large prospective study in Sweden reported that maternal smoking doubled the risk for SIDS and that a clear dose-response effect was observed (Haglund and Cnattingius, 1990). In the United States, maternal smoking has

been found to increase the risk of SIDS for all race/ethnic groups in a dose-response relationship. (MacDorman, et al., 1997).

Anderson and Cook (1997) pooled the results of 39 studies on maternal smoking and found that maternal smoking during pregnancy was consistently associated with an increased risk of SIDS when compared to mothers who did not smoke during pregnancy (pooled OR=2.08, 95% CI 1.83, 2.38). DiFranza and Lew (1995) conducted a similar meta-analysis of eleven studies and reported a pooled odds ratio of 2.98 (95% CI 2.51, 3.54).

These studies did not control for the effects of potential postpartum smoking by either the group who smoked during pregnancy or the group who did not smoke during pregnancy. It is possible that mothers in both groups began or continued smoking in the postpartum period which may introduce confounding of the relationship between maternal smoking during pregnancy and SIDS mortality.

Maternal smoking is a particularly important risk factor not only because of its strong association with SIDS but also because it is readily amendable to intervention. Maternal smoking includes smoking during both pregnancy and in the postpartum period. It is important to recognize that prenatal and postpartum exposure to cigarette smoking are highly correlated and that most women who smoke during pregnancy continue to do so in the postpartum period (Anderson and Cook, 1997). This makes it difficult to isolate the relationships between SIDS and maternal smoking during pregnancy or maternal smoking in the postpartum period. Most studies have not been large enough to separate the two time periods since it is difficult to find women who only smoke in the prenatal period (Dwyer, et al., 1999). Those mothers that do smoke in only one of the time

periods tend to be lighter smokers and thus have a lower risk of SIDS than the mothers who smoked in both periods (Mitchell, et al., 1993).

Some studies have attempted to disentangle the increased risk of SIDS mortality associated with maternal smoking during pregnancy from the risk attributable to maternal smoking in the postpartum period. Anderson and Cook (1997) pooled the data from four studies that examined maternal postpartum maternal smoking after controlling for prenatal maternal smoking and reported an increased risk of SIDS mortality associated with postpartum maternal smoking compared to nonsmokers (pooled OR = 1.94, 95% CI 1.55, 2.43). A different study found that smoking during and after pregnancy was associated with a threefold increased risk of SIDS, while exposure to cigarette smoke only after delivery resulted in a twofold increased risk (DiFranza and Lew, 1995).

It is likely that maternal smoking during pregnancy has an independent effect on SIDS mortality after controlling for maternal smoking in the postpartum period. In a review of the literature, Spiers (1999) concluded that there appears to be no quantitatively reliable estimate of the association between maternal smoking during pregnancy alone and the risk of SIDS. Its importance, however, can be inferred from the appreciably greater adjusted odds ratio for SIDS mortality in infants whose mothers smoked in the prenatal and postpartum period than the adjusted odds ratio associated with postpartum smoking alone.

Cigarette smoking by household members other than the mother has also been associated with SIDS. The effects of environmental tobacco smoke (ETS) on infants has been examined in several studies. Nicholl and O'Cathain (1992) reported an increased risk for SIDS when the mother was a nonsmoker and the partner a smoker compared with

households in which both were nonsmokers. However, Mitchell and others (1993) found that paternal smoking only increased the risk of SIDS in infants whose mothers also smoked (OR=2.41, 95% CI 1.92, 3.02). In another study, it was found that ETS exposure from persons other than the mother increased the risk of SIDS among white infants but not among black infants (DiFranza and Lew, 1995). It has been hypothesized that postpartum smoking exposure and ETS may increase the risk of SIDS through direct irritation of infants' airways or by increasing the risks of respiratory infection (Anderson and Cook, 1997).

Important research on the biologic effects of maternal smoking during pregnancy has been conducted. It is well established that maternal smoking during pregnancy lowers infant birth weight and that low birth weight increases the risk of SIDS. However, most studies have found that maternal smoking during pregnancy remains a risk factor for SIDS even after controlling for birth weight (Mitchell, et al., 1993). Maternal smoking may increase the risk of SIDS by affecting the arousal response in infants.

It has been proposed that maternal smoking during pregnancy may cause the delayed development of arousal or cardiorespiratory control and that this may cause SIDS in certain infants (Lewis and Bosque, 1995; Hunt, 1995; Willinger, et al., 1994; Ponsonby, et al., 1993). In pathological studies, it has been found that infants who died of SIDS have hypoplasia of the arcuate nucleus which is believed to function in the hypercapnic ventilatory response, chemosensitivity, and blood pressure response (Anonymous, 2000). It has been proposed that these infants are unable to respond appropriately to hypoxia and hypercapnia that may occur during sleep due to some noxious insult or condition.

Proposed causes of hypoxic stress which may be involved in the pathogenesis of SIDS include the rebreathing of expired gases when sleeping in the prone position, especially on soft sleeping surfaces (Kemp and Thach, 1991). Inability to mount an appropriate arousal response to a carbon dioxide rich and oxygen poor environment may result in fatal asphyxia. A normal sleeping infant would react readily and unconsciously to the noxious environment by lifting and turning its face. It has also been proposed that hyperthermia could provide the noxious insult leading to physiologic instability (Oyen, et al., 1997).

The evidence for a causal relationship between maternal smoking during pregnancy and SIDS is substantial. The increased risk remains after controlling for variables such as birth order, date of birth, sex, gestational age, low birth weight, race, maternal age, parity, occupation, and socioeconomic status (DiFranza and Lew 1995). A dose-response effect has been reported in many studies, with an increasing odds ratio for SIDS associated with the more cigarettes smoked by the mother per day. (MacDorman, et al., 1997; Mitchell, et al., 1991) Finally, plausible biological mechanisms have been proposed. These factors, in addition to the overwhelming consistency of the findings and strength of associations, all argue for a causal association between maternal smoking and SIDS.

Socioeconomic status

It has been consistently observed that the incidence of SIDS and of infant mortality overall is elevated among lower socioeconomic groups, whether measured by occupation, income, or education (Hoffman and Hillman, 1992). Low socioeconomic status may be regarded as a marker for other risk factors such as inadequate housing,

nutritional deficiencies, inadequate prenatal or medical care (MacDorman, et al., 1997). Some studies have controlled for potential confounders such as maternal smoking and found an attenuated association between socioeconomic status and SIDS (MacDorman, et al., 1997).

Race

The SIDS rate varies considerably among racial and ethnic groups. In the United States, Black and American Indian populations have the highest incidence rates, ranging from 2.6 to 6.0 per 1000 live births, while rates for Asians, Hispanics, and Whites have ranged from 1.0 to 2.5 per 1000 live births (Hoffman and Hillman, 1992). It has often been suggested that these racial differences are caused by a greater prevalence of risk factors in Blacks and American Indians, such as low birth weight, lower socioeconomic status, and young maternal age. Adjusting for different risk factors has produced varying results and it seems that the associations cannot be simply explained by confounding.

Maternal age

Numerous studies have reported an increased risk of SIDS for infants born to young mothers. The relative risks for SIDS mortality for infants born to young mothers compared to infants born to older mothers have been from about 2.0 to 2.8 and persists after controlling for low birth weight and race (Little and Peterson, 1990). Adjustment for parity increases the relative risk. Adjusting for socioeconomic status attenuates the association between young maternal age and SIDS but does not entirely eliminate it (Dwyer, et al., 1991).

Marital status

The NICHD study reported that 59% of SIDS mothers were single and infants born to single mothers had 2.5 times the risk of dying from SIDS than infants born to married women (Hoffman and Hillman, 1992). The excess risk associated with single mothers may operate through maternal age and socioeconomic status (Guntheroth, 1995). Single mothers are generally younger, have less education, and lower incomes.

Low birth weight and prematurity

Low birth weight and prematurity have been found to increase the risk of SIDS and this risk increases with decreasing gestational age or birth weight (Anonymous, 2000). In the NICHD study, the low birth weight infants were 4.5 times as likely to die from SIDS than normal birth weight infants (Hoffman, et al., 1988). It is noteworthy that the majority of SIDS infants are not low birth weight and it is unclear how birth weight works to increase the incidence of SIDS (Hoffman and Hillman, 1992). Low birth weight is not only an important risk factor for SIDS, but for many other causes of postneonatal deaths (Hoffman and Hillman, 1992).

Inadequate prenatal care

Most studies have reported an association between inadequate prenatal care and SIDS (Hoffman and Hillman, 1992). Inadequate prenatal care may reflect a negative or indifferent attitude towards the value of healthcare or health in general that may be of associated for SIDS (Guntheroth, 1995). In the NICHD study, the odds ratio was 2.5 for SIDS mortality comparing infants whose mothers had a late onset of prenatal care (third trimester or later) to infants whose mothers initiated prenatal care in the first or second trimester. Freed and associates (1994) suggest that most of the effect of late prenatal care

can be explained by confounding by maternal characteristics such as high parity, maternal smoking, single marital status, and young maternal age.

Maternal drug abuse

Maternal drug abuse has also been examined as a potential risk factor for SIDS. The use of marijuana, methadone, cocaine, heroin, or psychedelics during pregnancy doubled the risk of SIDS in the NICHD study but 75% of this drug use was for marijuana only (Hoffman and Hillman, 1992).

Seasonality

A seasonal variation in the incidence of SIDS has consistently been found in studies from numerous countries. The SIDS rate in cold weather months is approximately double the rate in warmer months (Hoffman and Hillman, 1992). Multiple risk factors may be involved, including infectious agents, temperature, nutritional or metabolic processes, infant care practices, or other behavioral factors. It has been postulated that the higher incidence of respiratory infections during cold months can explain the seasonal differences in SIDS occurrence (Guntheroth, 1995). The role of respiratory infections in SIDS will be discussed later. Petersen (1988), however, concluded that the underlying rates of infection could not fully explain the doubling of risk from warmer to colder months. Some studies have suggested that the age of the infant and the season interact, such that infants who are three months of age in the winter are at greater risk than other infants (Leiss and Suchindran, 1993)

Several reports have found a positive association between the SIDS and low temperature several days earlier (Hoffman and Hill, 1992). As in seasonality, the mechanisms are not understood, but it has been suggested that parents may

overcompensate for the cold weather by overdressing the infants, causing overheating which may trigger SIDS. The role of thermal stress will be discussed in greater detail later.

Maternal urinary tract infections

Maternal urinary tract infections during pregnancy have been an area of concern that have not been as thoroughly examined as other risk factors. Infants whose mothers had a urinary tract infection during pregnancy had an increased risk of SIDS (OR=1.80) compared to infants whose mothers did not have maternal urinary tract infections during (Hoffman and Hillman, 1992). However, the association may be confounded by other common risk factors such as low socioeconomic status and low birth weight. Some data suggests an increase in neurologic impairment associated with maternal urinary tract infections during pregnancy (Hoffman and Hillman, 1992).

Age of infant

Age at death is a distinguishing characteristic shared by SIDS cases and has been consistently been identified wherever it has been adequately studied (Hoffman, et al., 1988). Although people of all ages die suddenly and unexpectedly without any lethal pathology at autopsy, it is generally agreed that the upper age limit for SIDS deaths is one year. The age distribution for SIDS shows that it is most common from one to six months with a peak from two to three months and published curves are clearly asymptotic to eleven or twelve months of age (Leiss and Suchindran, 1993). SIDS is also relatively uncommon in neonates.

Breast feeding

Several retrospective studies have reported that breastfeeding is protective against SIDS. Mitchell and associates (1991) reported that lack of breast feeding was associated with a substantially increased risk of SIDS compared to infants who were breastfed (OR= 2.45, 95% CI 1.32, 4.55) after controlling for an extensive list of other variables. However, other prospective cohort studies have reported no association between breast feeding and SIDS after adjusting for confounding variables (Mitchell, et al., 1997). The American Academy of Pediatrics stated that the evidence supporting the promotion of breast feeding to reduce SIDS is inconclusive although breast feeding should be encouraged for numerous other health benefits (Anonymous, 2000).

Infections

Infection has been implicated in numerous reports on SIDS, both through histologic evidence and interviews of parents (Guntheroth, 1995). In the NICHD study, there was no difference reported in the frequency of upper respiratory infections in the last 2 weeks of life for African-American SIDS cases compared to controls but Caucasian SIDS infants had significantly more colds than the controls. An important issue when considering the role of infections is the varying ability of parents to recognize signs or symptoms. It is also important to recognize that the infection need not be severe since illness may act only as a trigger for prolonged apnea or to inhibit arousal from sleep.

Prolonged QT interval

The prolongation of the QT interval recently gained renewed attention as a risk factor for SIDS. In 1998, a large prospective cohort study in Italy reported that a significantly greater number of SIDS cases had prolonged QT intervals on a screening electrocardiogram compared to controls (Schwartz, et al., 1998). They proposed that the

prolonged QT interval on the electrocardiogram was the result of a developmental abnormality in sympathetic cardiac innervation which may increase the risk of ventricular arrhythmias. The evidence at present does not appear to support the implementation of widespread electrocardiographic screening to identify at-risk infants (Anonymous, 2000)

Co-sleeping

The safety of bed sharing or co-sleeping has been a point of controversy in the SIDS debate. There have been four large studies of SIDS and co-sleeping (Rosenberg, 2000). A 1993 New Zealand case-control study reported that the risk of SIDS associated with bed sharing was only increased among infants whose mothers smoked (Scragg, et al., 1993). In 1996, an English case-control study reported an increased risk of SIDS for infants who shared a bed with a smoking mother but no increase in risk was observed if the mother was a nonsmoker (Fleming, et al., 1996). In 1997, a New Zealand prospective study also reported an increased risk of SIDS for bed sharing by smoking mothers and that there was no increased risk for bed sharing among nonsmoking mothers (Mitchell, et al., 1997). Finally, a California case-control study found no association between SIDS and bed sharing (Klonoff-Cohen and Edelstein, 1995)

While at one time it was commonly believed that the risk of SIDS associated with bed sharing was due to overlay (asphyxiation), this argument fails to explain why bed sharing only increases the risk of SIDS in infants whose mothers smoked. The increased risk may be related to deficient hypoxic arousal responses in some infants caused by maternal smoking during pregnancy (Lewis and Bosque, 1995). There may be complex interactions involved between parent and child in bed sharing that need to be better

understood. In addition, there may be potential psychological, social or health benefits associated with bed sharing such as increased breast feeding (O'Hara, et al., 2000).

The complexity of co-sleeping was demonstrated in a large English case-control study (325 cases and 1300 controls) which found that only certain co-sleeping arrangements and parental behaviors (sofa sleeping with infant, bed sharing with prior alcohol use, bed sharing for smoking mothers, and bed sharing with parental exhaustion) were associated with an increased risk of SIDS (McAfee, 2000). In fact, the English study reported that the risk for SIDS mortality in infants who slept on the sofa with a parent was increased fifty-fold compared to infants who slept alone and the authors believed that sofa sleeping may cause accidental suffocation (Blair, et al., 1999).
At the current time the evidence does not justify a recommendation for or against the practice of bed sharing (Anonymous, 2000).

Summary of risk factors

Demographic characteristics and maternal and infant risk factors have been well documented for SIDS. The prone sleeping position was the most important epidemiologic finding concerning SIDS and national intervention campaigns have reduced SIDS rates worldwide. The next modifiable risk factor to be addressed seems to be maternal smoking.

METHODS

Study Aims

The overall aim of this study is to assess the effect of the Back to Sleep campaign on the epidemiology of SIDS mortality in Oregon. Specifically, changes in the contribution of maternal smoking during pregnancy, as reported on birth certificate data, on SIDS mortality in Oregon will be examined. Maternal smoking during pregnancy has been consistently identified as having one the strongest associations with SIDS mortality. Since the Back to Sleep campaign has reduced the prevalence of the prone sleep position, maternal smoking during pregnancy may now be associated with an even greater proportion of SIDS deaths.

In addition, this study will also examine whether the risk for SIDS has decreased among mothers of all age groups. It is hypothesized that younger mothers have not experienced the same reductions in SIDS risk than older mothers.

This study tested the following hypotheses:

1. (*a priori* hypothesis) Population attributable risk per cent (PAR%) for the effect of maternal smoking during pregnancy on SIDS mortality has increased from 1991-1993 to1994-1996.
2. (*a posteriori* hypothesis) The risk of SIDS for infants born to younger mothers (<20 years old) did not decline to the same extent as the risk of SIDS for infants born to older mothers (≥20 years old).

1. Population attributable risk percentage (PAR%)

The first hypothesis is concerned with assessing the role of maternal smoking on SIDS mortality. Quantifying the risk attributable to a specific factor in a population is a useful idea from an epidemiologic standpoint. As with any measure of association drawn from non-experimental epidemiologic studies, it is important to consider possible confounding by other factors when drawing conclusions from measures of PAR%. PAR% represents the proportion of the total disease incidence that would be eliminated if the risk factor could be removed from the population, under the assumptions that the risk factor is indeed a causal agent of the disease and that the risk factor is distributed in the population independently of other causal risk factors for the disease. If confounding exists, then the PAR% estimate will be greater or lesser than the reduction that would actually occur, depending on whether the confounding is positive or negative.

Calculations of PAR% that do not account for confounding should be considered crude estimates of the proportion of disease related to the risk factor. Statistical methods have been developed that adjust for confounding in the calculation of PAR%. An adjusted PAR% based on a logistic model was utilized in this project to calculate the proportion of SIDS mortality that was attributable to maternal smoking during pregnancy (Bruzzi, et al., 1985).

Bruzzi and others (1985) present a method to calculate an adjusted PAR% estimate which requires estimates of the relative risk for each level of the risk factor and the proportion of the cases that are in each level of the risk factor. The adjusted odds ratio obtained from a logistic regression model can be used to estimate relative risk when the disease in rare in the population (Bruzzi, et al., 1985). In this paper, the proportion of SIDS caused by maternal smoking will be estimated using this method.

Various parameters are utilized in epidemiology to assess the association between diseases and risk factors. Relative risk is perhaps most commonly used and can be viewed as measuring the strength of the association between exposure and disease. Much of the popularity of the relative risk as a measure of association between disease and risk factor is due to the ability to calculate or estimate relative risk from the three standard epidemiologic study designs: cross-sectional, prospective, and retrospective (Walter, 1978). However, relative risk does not take into account the number of persons exposed to the risk factor under study. A risk factor may have a high relative risk but cause few cases of disease due to a low prevalence of exposure to the risk factor in the population. Such a risk factor would probably not be considered a public health priority and in this situation relative risk would not be very useful from the perspective of public health planning (Walter, 1978).

As an alternative measure, population attributable risk percent, does take into account both the strength of association and the number of persons exposed to the risk factor in question, and is therefore a more useful measurement of the public health importance of a risk factor. PAR% can be a powerful epidemiologic tool in public health planning. For policymakers, PAR% is an easily understood measure of the potential impact of public health interventions and is useful in judging public health priorities.

However, PAR% is limited because it does not provide a direct measure of absolute numbers of lives saved or diseases prevented (excess risk would be the appropriate measure). In some instances the PAR% could be misleading if used to assess the importance of a risk factor from a cost-efficiency perspective. For a rare disease, a high PAR% for a risk factor may still mean only a few cases of diseases could be

prevented. For a common disease, eliminating a risk factor with a small PAR% could prevent many cases of disease. In these cases, additional information such as the excess risk would be useful in making policy decisions.

In the case of SIDS, the determination of the PAR% of maternal smoking helps us to better understand the impact of maternal smoking on SIDS mortality. While it is important to continue efforts to discourage the prone sleep position, it is also important to find ways to further lower SIDS rates. The PAR% findings may be useful in guiding future efforts towards preventing SIDS mortality.

2. Maternal Age

The second hypothesis of this project examines the changes in the risk for SIDS mortality that occurred for different maternal age groups after the Back to Sleep campaign that began in 1994. The investigation of maternal age and SIDS mortality was not one of the initial aims of this study. While it would have been methodologically preferable for the maternal age hypothesis to have been stated *a priori*, we felt it was important to pursue the *a posteriori* hypothesis with the intention that any findings would only be used to encourage the need for further research into this area. It may be important to focus prevention efforts on mothers of certain age groups if it is found that they have not received and followed previous recommendations.

Study population

A retrospective cohort study was conducted to examine maternal smoking during pregnancy and young maternal age as risk factors for SIDS. The study population included all infants born in Oregon to Oregon resident mothers from 1991 to 1996. Infant birth, matched infant birth and death, and infant death databases are maintained by the

Center for Health Statistics (CHS) at the Oregon Health Division and these were the sources of data for this study. The birth certificate records are an extensive source of maternal, prenatal, and infant information that includes numerous known risk factors for SIDS such as low birth weight, maternal use of tobacco and prenatal health care utilization. The matched infant birth /death data set and the infant death data set were used to identify all SIDS deaths to infants in the study population. The CHS links infant death certificates to birth certificates by birth certificate number to create the matched birth and death database.

An exemption from a full Oregon Health Division/Multnomah County Institutional Review Board was received on January 13, 1998 on the grounds that the study involved the collection and study of existing data, documents, and records. In addition, the subjects could not be identified directly or through identifiers linked to the subjects.

The birth data sets were the source of data on SIDS risk factors. The dichotomous variable "SIDS death" was added to the data set and infants who had died of SIDS were coded as "yes" and those who had not were coded as "no." SIDS deaths were identified using both the matched birth and death and infant death data sets. No names are included in any of the data sets but individuals are given unique birth certificate and death certificate numbers. In this manner the anonymity of the study population was ensured.

SIDS deaths were those for which an International Classification of Diseases, Ninth Revision, (ICD-9) code of 798.0 was given as the underlying cause of death. In Oregon, the process of determining the cause of death and filing the death certificate is as follows. The county medical examiner is notified of any death of an infant (except if the

death occurred at the hospital under the care of a physician) and a death scene investigation is generally conducted by the medical examiner and local law enforcement agency. In addition, the district attorney is generally notified and an autopsy is generally performed. Ultimately, the medical examiner determines the cause of death that will be reported in the death certificate that is filed in the county of death.

Infants with incomplete data were not removed from the study population. Instead, individuals were excluded from certain analyses only when they were missing information on specific risk factors required for a particular analysis.

The matched infant death data set was only available through 1996. Thus, infants who were born in 1996 but died of SIDS in 1997 were not identified through this method. In order to capture all the SIDS cases among infants born in 1996, the 1997 infant death certificate records were used. The infant death data file for 1997 that was initially obtained was provisional but a finalized data set was obtained in June 1999 that added ten SIDS cases to the initial set.

The data was analyzed using birth cohorts so that all infants born between January 1, 1991 and December 31, 1993 were included in the 1991-93 birth cohort and all infants born between January 1, 1994 and December 31, 1996 were included in the 1994-96 birth cohort. Alternatively, death cohorts could have been utilized so that infants were assigned to the cohort corresponding to the year in which the death occurred. Birth cohorts were utilized in this study for methodological reasons. When calculating SIDS mortality rates by birth cohorts all the SIDS cases in the numerator are drawn from the denominator. This differs from the death cohort method used by the Center for Health Statistics. They calculate annual SIDS rates using the year in which the death occurred so their numerator

includes all SIDS cases in one year and their denominator includes the total number of live births for that same year. In their calculation, cases in the numerator may not be represented in the denominator. In addition, we felt that separating infants born 1991-93 and born 1994-96 was the most accurate method of measuring changes due to the Back to Sleep campaign. Using death cohorts would have included infants in the post Back to Sleep cohort that were actually born before the Back to Sleep campaign was initiated.

Preliminary analysis

All analyses were performed using SPSS 9.0. Initial analysis began with an examination of established risk factors that were identified in the literature. The full data set from 1991-1996 was utilized in order to identify significant risk factors over the entire time period. The frequency distribution of values for each risk factor of interest was examined. Certain variables such as mother's education, birth order, and maternal age were re-coded into categorical variables. The scale of each risk factor was chosen by what was commonly used in the literature and/or by examining SIDS rates for different levels of each risk factor.

Most of the variables were directly obtained from the birth certificate data while other required some modifications. Table 1 describes the variables and the coding that was used.

In order to determine the final categorization of the mother's age variable, SIDS rates according to mother's age were calculated for three-year age categories. The resulting SIDS rates for the three year age categories suggested that three distinct risk categories for SIDS existed and were most appropriately represented by the three age categories <20, 20-29, ≥30 years old.

Season of birth was dichotomized into infants born in August through December and those born in January through July. We felt that this grouping would allow us to make inferences upon the hypothesis that infants who are two to three months old in the winter are at greater risk than other infants (Leiss and Suchindran, 1993). Infants born in August through December would be at their peak age risk for SIDS in October through March while those born in January through July are at their peak risks from March through October.

The variable "time period" was also created to measure the decrease in the risk of SIDS for infants born in 1994-1996 that has been attributed to the Back to Sleep campaign. Infants born in 1991-1993 are assumed to be at a higher risk for sleeping prone because they were born prior to the Back to Sleep campaign and are coded as "1." Infants born in 1994-1996 are assumed to be at a lower risk for sleeping prone because they were born after the intervention and are coded as "0". Discuss why babies born in first part of 94 in period 1 and mediating factor in small effect of AAP.

Pearson's chi-square test was used to explore the association between each risk factor and SIDS. Associations that were significant at the $p<.25$ level were considered in a multiple logistic regression model (Hosmer and Lemenshow, 1989). Fifteen variables were screened in this manner and all were found to be significantly associated with SIDS mortality. In addition to the chi-square tests, univariate logistic regressions were performed to obtain estimates of crude odds ratios.

The prevalence of each risk factor was assessed for each time period. The prevalence of each risk factor in 1991-1993 was compared to the prevalence observed in

1994-1996. Pearson Chi-Square test was used to test whether each risk factor and time period were independent (Norusis, 1998).

Maternal smoking during pregnancy

In order to determine a crude estimate of the proportion of SIDS cases attributable to maternal smoking, a crude PAR% for each time period was calculated using the equation (Northridge, 1995):

$$PAR\% = \frac{(Rate_{\text{total population}} - Rate_{\text{unexposed}})}{Rate_{\text{total population}}} \times 100$$

All rates in this study are per 1000 live births. In the formula above,

$$Rate_{\text{total population}} = \frac{\#\,SIDS}{\text{Total live births}} \times 1000$$

The PAR% calculated using the above equation does not consider how the association between maternal smoking and SIDS may be confounded by other factors. We used a method presented in Bruzzi and colleagues (1985) to calculate an adjusted PAR% ($PAR\%_{adj}$). Th method requires estimates of the relative risk for each level of the risk factor and the proportion of the cases that are in each level of the risk factor, the details of which are as follows.

The equation presented by Bruzzi and colleagues (1985) assumes that the risk factor of interest has j levels with the level of lowest risk designated as j=0. A logistic regression model with SIDS as the dependent variable was used to estimate regression coefficients, each representing the log odds ratio for a unit change in a variable adjusted for the other variables. We were interested in the odds ratio for maternal smoking during pregnancy adjusted for important confounders and j = 0 was designated as mothers who

did not smoke during pregnancy while j = 1 was designated as mothers who smoked during pregnancy. The adjusted odds ratio was then used in the following equation to determine $PAR\%_{adj}$ (Bruzzi, et al., 1985):

$$PAR\%adj = 1 - \sum_{j} \frac{P_j}{RR_j}$$

In the equation, P_j is the proportion of cases that are in the level j of risk factor and RR_j is the relative risk estimated from logistic regression model for level j of the risk factor. It is appropriate to approximate this relative risk with the odds ratio obtained through logistic regression models for a disease that is uncommon in the population (Bruzzi, et al., 1985). Interaction terms must be assessed and if statistically significant the odds ratio for the risk factor would have to consider the effects of the interactions. For example, for a risk factor A with factor C as a confounding variable, we would include terms for the main effects of the risk factor A and also any interactions between A and C (Bruzzi, et al., 1985). All individuals would then be classified into j mutually exclusive strata formed by cross-classifying A and C. RR_j becomes the risk for stratum j and P_j is the proportion of cases within stratum j.

In order to calculate adjusted odds ratios associated with maternal smoking during pregnancy we first identified which risk factors were significant confounders during the 1991-1996 time period using logistic regression models. The analysis of confounders was not assessed separately for the 1991-1993 time period and the 1994-1996 time period in order to adjust for the same confounders in both time periods and because we did not expect that the main confounders changed in a statistically significant manner between

the time periods. The dependent variable in all the logistic regression models was SIDS death which was coded yes = 1 and no = 0.

The crude odds ratio associated with maternal smoking during pregnancy was calculated from the univariate logistic regression model with maternal smoking during pregnancy (1 = yes, 0 = no) as the independent variable. Next, fourteen bivariate logistic regression models were constructed. Each model included maternal smoking during pregnancy as one risk factor and one of the fourteen other risk factors (Table 2) assessed in this study as the other risk factor.

Confounding by each risk factor on the association between maternal smoking and SIDS was assessed by comparing the crude odds ratio for maternal smoking to the adjusted odds ratio for maternal smoking in the bivariate model. The proportionate change in the between the crude odds ratio and the adjusted odds ratio for maternal smoking was calculated. Any risk factor which demonstrated confounding of the relationship between maternal smoking and SIDS by more than 5% was considered for further analysis.

In the next step, logistic regression models were assessed that included different combinations of maternal smoking and two of these risk factors. Additional risk factors were added, creating larger models, until the coefficient for smoking no longer changed substantially (5%). Correlation between the potential confounders was analyzed with the gamma statistic (Agresti, 1990) to determine if any pairs of confounders were highly correlated. For highly correlated pairs (gamma > 0.6) only one pair was included in the logistic regression model unless the combined effect of the pair was substantially larger

than the effect from including only one of the pair. A final set of confounders was selected based upon greatest confounding effect after screening for correlations.

Time period specific (1991-1993 and 1994-1996) adjusted odds ratios for maternal smoking during pregnancy were calculated through logistic regression models which included maternal smoking during pregnancy and the final set of confounders as independent variables. Each time period was assessed separately at this point. The adjusted odds ratios were then used to calculate the adjusted population attributable risk percent for maternal smoking during pregnancy for each time period in order to assess whether any change had occurred.

Maternal age

The SIDS rate per 1000 live births for each maternal age category was calculated for each time period. The proportionate change in SIDS rates between 1991-1993 and 1994-1996 for each maternal age group was quantified and compared.

To assess whether the odds ratios for the mother's age categories changed significantly between 1991-1993 and 1994-1996, a logistic regression model was constructed using the backward elimination method of selecting independent variables for a logistic regression model. All fifteen risk factors listed in Table 1 were candidates for inclusion in this logistic regression model. The backward elimination process began by including all 15 variables in the model, and then testing for removal using the log likelihood ratio test criteria. Conservative significance levels were chosen so that only significant risk factors were maintained in the model ($p<.10$). In addition, variables that were removed during the backward elimination process were assessed for any

confounding of other variables in the model. If any significant confounding was observed the confounding variable was placed back into the model.

This approach clearly differs from and potentially includes more risk factors in the final logistic regression model than the approach used to assess maternal smoking. For maternal smoking, it was felt that including risk factors that were not strong confounders of the association between maternal smoking and SIDS in the logistic regression model would only add unnecessary complexity to the model and make it difficult to identify which risk factors were the major confounders. In the examination of maternal age, it was felt that the added complexity was justified because the final model would identify all statistically significant risk factors and that this was more important than highlighting which risk factors were the most significant confounders of the relationship between maternal age and SIDS. In addition, odds ratios for all the significant risk factors could be determined. While not directly related to the assessment of maternal age, these finding would be useful to guide further research and public health efforts.

The dichotomous variable "time period" was created. Infant births in 1991-1993 were coded as "1" to reflect their greater risk of sleeping prone because they were born before the Back to Sleep campaign and infants born in 1994-1996 were coded as "0". The interaction term between maternal age and time period was added to the model in order to determine whether the odds ratios for mother's age were homogenous across the time periods. The 1994-1996 was the referent category and was coded as " 0". Testing the homogeneity of the odds ratios across the time periods was performed through the log

likelihood ratio test of the model without the interaction term versus the model with the interaction term (Hosmer and Lemeshow, 1989).

To calculate the odds ratios for the maternal age groups, a separate logistic regression model was analyzed in which the variable "timeage" was created to reflect the cross-classification of the three maternal age groups and two time periods into six mutually exclusive strata. The strata were coded 0-5.

The variable "timeage" and was included in a multivariate logistic regression along with the statistically significant risk factors identified from the initial multivariate logistic regression model constructed using the backward elimination method of selecting independent variables. We wanted to measure the risk of SIDS for each maternal age group in 1994-1996 compared to its own risk in 1991-1993. Odds ratios were calculated for each maternal age category that quantified this risk using logistic regression with tests of contrasts. The 1991-1993 maternal age groups were specified as the reference groups for their respective groups in 1994-1996. For example, the risk of SIDS for infants born to mothers <20 years old in 1994-1996 was compared to the risk of SIDS for infants born to mothers <20 years old in 1991-1993.

Changes in the prevalence of risk factors were assessed for each maternal age group. We were particularly interested in knowing if maternal smoking during pregnancy had increased among mothers <20 years old in 1994-1996. We used Chi-square to test the independence of maternal smoking during pregnancy and time period of birth. Other risk factors tested were Medicaid/OHP coverage, alcohol use, and marital status.

RESULTS

Preliminary analyses

A total of 254,157 infants were included in the study. 125,965 of these infants were born in 1991-1993 (time period 1) and 128,192 (time period 2) were born in 1994-1996. There were 243 SIDS deaths for time period 1 and 160 SIDS deaths for time period 2. The SIDS rate declined 33% from 1.87 SIDS deaths per 1000 live births in 1991-1993 to 1.25 SIDS deaths per 1000 live births in 1994-1996. Table 2 shows the prevalence of the risk factors in 1991-1993 and 1994-1996. Risk factors that showed increases in prevalence in 1994-1996 were young motherhood (<20 years old), older motherhood (>29 years old), and single/divorced marital status. Risk factors with reduced prevalence in 1994-1996 include maternal smoking during pregnancy and maternal alcohol use during pregnancy. The racial/ethnic composition of infants born in Oregon underwent substantial changes from 1991-93 to 1994-96. The proportion of births that were Hispanic grew from 9% to 12%. In addition, the proportion of births that were Caucasian fell from 85% to 81%.

Crude odds ratios for all fifteen risk factors are listed in Table 3. Maternal smoking during pregnancy (OR=4.14), maternal age <20 years old (OR=3.64), and alcohol use during pregnancy (OR=3.44) were most strongly associated with SIDS.

Maternal smoking during pregnancy

The prevalence of maternal smoking during pregnancy fell from 20% in 1991-1993 to 18% in 1994-1996 (Table 2). In addition, the SIDS rates for both smokers and nonsmokers decreased, although smokers had a proportionately greater reduction. The SIDS rate among smokers fell 35% from 4.88 per 1000 live births to 3.15 per 1000 live

births. The SIDS rate among nonsmokers fell from 1.11 per 1000 live births to 0.83 per 1000 live births, a 25% reduction. The crude relative risk for SIDS mortality comparing infants whose mothers smoked during pregnancy to infants whose mothers did not smoke during pregnancy was reduced from 4.39 in 1991-1993 to 3.79 in 1994-96. This reduction in crude relative risk reflects the disproportionate reduction in the rate of SIDS among infants whose mothers smoked during pregnancy compared to infants whose mothers did not smoke during pregnancy.

Assessing risk factors over the total time period (1991-1996), infants born to mothers who smoked during pregnancy were four times as likely to die from SIDS compared to infants whose mothers did not smoke during pregnancy. Single marital status, Medicaid/OHP coverage, mother's education <12 years, alcohol use during pregnancy, and low birth weight substantially confounded this relationship. Marital status and Medicaid/OHP coverage were the most important confounders (Table 4). Controlling for the combined confounding effects of marital status and Medicaid/OHP, the odds ratio for SIDS mortality comparing infants whose mothers smoked during pregnancy to infants whose mothers did not smoke during pregnancy was adjusted from 4.14 to 2.97. Adding education, maternal age, and maternal alcohol use during pregnancy to the model only provided a minimal further adjustment of the odds ratio for maternal smoking (adjusted OR=2.74) suggesting that these risk factors were not significant confounders. Thus, maternal education, maternal age, and maternal alcohol use were not included in the final model.

Although marital status and Medicaid/OHP coverage were highly correlated with one another (gamma = 0.78) both were included in the final model because together they

demonstrated substantial confounding compared to each individually. The adjusted odds ratio for maternal smoking when adjusting for marital status alone was 3.25, a 22% change.

The odds ratio for SIDS mortality comparing infants whose mothers smoked during pregnancy to infants whose mothers did not smoke during pregnancy was 3.28 after controlling for Medicaid/OHP coverage, a 21% change. Adjusting for both marital status and Medicaid/OHP coverage resulted in an adjusted odds ratio of 2.97, a 28% change compared to the crude odds ratio for maternal smoking.

In 1991-93, the odds ratio for SIDS mortality comparing infants whose mothers smoked during pregnancy to infants whose mothers did not smoke during pregnancy 3.16 after adjusting for marital status and Medicaid/OHP coverage (Table 5). This odds ratio fell to 2.59 in 1994-96 (Table 5).

Using these adjusted odds ratios it was estimated that the adjusted PAR% for maternal smoking during pregnancy was 36% in 1991-1993 and the adjusted PAR% fell to 28% in 1994-1996 (Table 6). Finally, the proportion of SIDS cases whose mothers smoked during pregnancy decreased (52% versus 45%) and the crude PAR% fell from 41% to 34%.

Maternal age

The proportion of infants born to mothers in each of the age categories remained similar between the two time periods. For 1994-1996, a slightly greater proportion of infants were born to mothers <20 years old and to mothers >29 years old while the proportion of infants born to mothers age 20-29 was slightly reduced (Table 2). The SIDS

rates for each age category dropped, although the percentage decrease was nearly three times as great for the 20-29 and >29 age group than for the <20 age group (Table 7).

Statistically significant risk factors (1991-1996) from multivariate logistic regression are presented in Table 8. The risk factors most strongly associated with SIDS mortality include maternal smoking (OR=2.47), low birth weight and birth order. Season of birth and Medicaid/OHP coverage were weaker predictors of SIDS. The variables heroin, cocaine, and methamphetamine use, mother's race, inadequate prenatal care and urinary tract infection were not significant predictors of SIDS in the multivariate logistic regression model for 1991-1996.

The interaction term maternal age*time was not statistically significant at the $p< 0.05$ level indicating that there were not statistically significant changes in the odds ratios for the maternal age categories between the time periods. However, the likelihood ratio test value was large enough (Chi-square=3.61, p=.16) to warrant further a exploration of this interaction (Hosmer and Lemeshow, 1989).

Table 8 presents the risk of SIDS for each maternal age group in 1994-1996 compared to its risk in 1991-1993. The risk of SIDS for young mothers (<20 years old) in 1994-1996 compared to young mothers in 1991-1993 was only slightly decreased (OR=0.91, 95% CI 0.62, 1.33). In comparison, both the 20-29 year old age group (OR=0.61, 95% CI 0.46, 0.81) and the >29 year old age group (OR=0.56, 95% CI 0.35, 0.90) had significant decreases in their risks for SIDS compared to 1991-1993. In addition, the confidence intervals for both older age groups are below unity while the confidence interval for the <20 age group includes unity. Clearly, the evidence suggests a

much less dramatic decrease in the risk of SIDS from 1991-1993 to 1994-1996 for mothers <20 years old.

The prevalence of maternal smoking during pregnancy among mothers <20 years old fell 2.8% from 30.6% of mothers in 1991-1993 to 27.8%. The prevalence of maternal smoking during pregnancy for mothers>29 years old fell from 14.4% to 13.0%.

DISCUSSION

Maternal Smoking During Pregnancy

It was hypothesized that the proportion of SIDS in Oregon that could be attributed to maternal smoking would be larger after the 1994 Back to Sleep campaign because of the expected reduction in the proportion of SIDS deaths associated with prone sleeping. The results do not support this hypothesis. Both the crude and adjusted PAR% for maternal smoking decreased from 1991-1993 to 1994-1996.

The crude PAR% for maternal smoking decreased from 40% in 1991-1993 to 34% in 1994-1996. Factors which may have contributed to the observed decrease in PAR% can be assessed by examining the components of the crude PAR% equation (Northridge, 1995):

$$PAR\% = \frac{(Rate_{\text{total population}} - Rate_{\text{unexposed}})}{Rate_{\text{total population}}} \times 100$$

The equation consists of two SIDS rates: the SIDS rate in the total population and the SIDS rate among the unexposed. In this study, the unexposed are the infants whose mothers who did not smoke during pregnancy who will be referred to as nonsmokers and the exposed group consists of infants whose mothers smoked during pregnancy, who will be referred to as smokers. The SIDS rate in the total population is determined by the SIDS rate among nonsmokers and the SIDS rate among smokers. In Oregon, the total population SIDS rate for the two time periods dropped from 1.92 per 1000 live births to 1.25 per 1000 live births.

The effect of this 35% drop in the overall SIDS rate on PAR% cannot be predicted without assessing the SIDS rate changes that occurred among smokers and

nonsmokers. If both groups experienced proportionately equal reductions in their respective SIDS rates, there would be no change in the PAR% for maternal smoking. If smokers experienced a greater reduction in SIDS rates than nonsmokers then the PAR% for maternal smoking would be expected to decrease. Finally, if nonsmokers experienced a greater reduction in SIDS rates than smokers then the PAR% for maternal smoking would have increased.

In fact, SIDS rates for nonsmokers fell from 1.16 per 1000 live births to 0.83 per 1000 live births, a 28% reduction. The SIDS rate for smokers fell by 36%, from 4.96 SIDS cases per 1000 live births to 3.15 SIDS cases per 1000 live births. Corresponding to the change in SIDS rates, the relative risk for maternal smoking during pregnancy fell from 4.27 to 3.79. The change in the association between maternal smoking and SIDS mortality was unexpected and will be discussed later.

From the results above we can state that the decreased PAR% for maternal smoking during pregnancy can be attributed in part to a disproportionate change in SIDS rates among smokers and nonsmokers. In addition, the prevalence of maternal smoking decreased from 20.2% in 1991-1993 to17.9% in 1994-1996, which also contributed to the decreased PAR% for maternal smoking during pregnancy.

The PAR% that would have been expected if the SIDS rates had changed in the manner that was observed but the maternal smoking prevalence had remained constant was calculated as follows. The 1991-1993 maternal smoking prevalence of 17.9% was applied to the 1994-1996 population and it was determined that an additional 2954 mothers who smoked during pregnancy would have been expected for 1994-1996 if the smoking prevalence had remained the same as in 1991-1993. Nine SIDS deaths would

have been predicted to occur among these 2954 mothers based on the SIDS rates for smokers 1994-1996. In 1991-1993, 79.1% of pregnant women did not smoke compared to 81.7% in 1994-96. Applying the 1991-1993 prevalence to the 1994-1996 resulted in 3163 less nonsmokers in 1994-96 than was actually observed. Based on the SIDS rate of 0.83 per 1000 live births for nonsmokers, three less SIDS deaths would have been expected to occur among nonsmoking women. A net increase of six SIDS cases would have occurred if the maternal smoking during pregnancy prevalence had remained constant. The SIDS rate in the total population would have been 1.30 per 1000 live births and the PAR% would have been 36%. Thus, the disproportionate change in SIDS rates would have resulted in the PAR% for maternal smoking during pregnancy to decrease from 40% in 1991-1993 to 36% in 1994-1996, instead of the observed PAR% of 34%, if the prevalence of maternal smoking during pregnancy had not changed.

The reduction in PAR% caused by the reduced prevalence of maternal smoking during pregnancy was determined by applying the 1991-1993 SIDS rates for smokers and nonsmokers to the 1994-1996 group. Applying these rates, there would have been 114 SIDS cases among the 22890 mothers who smoked during pregnancy in 1994-1996. There were 104691 nonsmokers in 1994-1996 which results in an expected 121 cases of SIDS based on the SIDS rate for nonsmokers in 1991-1993. A total of 235 cases of SIDS would have been expected, resulting in a total SIDS rate of 1.83 per 1000 live births. Using these figures, the hypothetical crude PAR% for maternal smoking in 1994-1996 would have been 37% if no changes in SIDS rates had occurred among smokers or non-smokers. Thus, the decrease in the prevalence of maternal smoking would have resulted in a decrease in PAR% from 40% to 37% if the SIDS rates for smokers and nonsmokers

had not changed. In addition to the crude PAR%, it was found that the adjusted PAR% decreased from 36% to 28% using the equation (Bruzzi, et al., 1985):

$$PARadj\% = 1 - \sum_j \frac{P_j}{RR_j}$$

P_j is the proportion of cases that are in level j of the risk factor. RR_j is the relative risk estimated by the odds ratio from logistic regression for level j of the risk factor.

In this equation, a decrease in the proportion of SIDS cases whose mothers smoked during pregnancy and/or a decrease in the relative risk for maternal smoking would result in a lower PAR%. 52% of SIDS cases were positive for maternal smoking in 1991-1993 compared to 45% of SIDS cases in 1994-1996 and this decrease explains part of the decrease in the adjusted PAR%. In addition, the adjusted odds ratio for smoking decreased from 3.16 to 2.59 which further reduced the PAR% of maternal smoking during pregnancy.

Our initial hypothesis was incorrect because we profoundly misunderstood the determinants of PAR%. To illustrate our reasoning, consider a hypothetical situation in which there were two hundred SIDS cases in one year and fifty of the cases were exposed to maternal smoking during pregnancy, one hundred cases slept prone, and fifty cases were exposed to other risk factors. We envisioned PAR% as a pie chart of the 200 SIDS cases in which each section of the pie represented the cases exposed to a particular risk factor. For the previously described hypothetical situation the pie would consist of three sections with 50% of the pie representing SIDS cases who slept prone, 25% representing maternal smoking during pregnancy, and 25% representing other causes. We believed

that each section of the pie was also representative of the PAR%. Therefore, reducing the prevalence of prone sleeping would make the section of the pie representing prone sleeping smaller while the other sections representing maternal smoking during pregnancy and other risk factors would become proportionately larger. In fact, this was an erroneous way to conceptualize PAR%.

The pie chart we envisioned does not accurately represent PAR% nor can it represent all SIDS cases because it does not account for SIDS cases who were exposed to multiple risk factors. For example, it has no representation of infants who slept prone and whose mothers smoked during pregnancy. This is an important point because the prevalence of the prone sleep position was relatively high before the Back to Sleep campaign so it is likely that a substantial proportion of infants whose mothers smoked during pregnancy also slept in the prone position. Therefore, if the prevalence of the prone sleep position was reduced in the total population, one would expect that the prevalence of the prone sleep position would also be reduced among infants whose mothers smoked during pregnancy. This would result in a reduction of SIDS mortality among infants born to mothers who smoked during pregnancy that was not accounted for in the pie chart conceptualization of PAR%.

The PAR% should not have increased or decreased due to a reduction in the prevalence of the prone sleep position if there is no confounding or interaction between maternal smoking during pregnancy and the prone sleep position. Confounding would occur if the prevalence of prone sleeping was not the same between smokers and nonsmokers. An interaction effect would be observed if the effect of prone sleeping on SIDS mortality was not the same among smokers and nonsmokers.

Since no data on sleep position was available for this study it was not possible to control for the effects of sleep position on the association between maternal smoking and SIDS or to assess for a possible interaction between prone sleeping and maternal smoking. However, Oyen and others (1997) have reported such an association. They reported that the effects of maternal smoking and the prone sleep position on SIDS mortality are multiplicative. The odds ratio for prone sleeping compared with supine sleeping were 13.9 (8.2-24). The odds ratio for maternal smoking in pregnancy compared with no maternal smoking in pregnancy was 4.1 (95% CI 3.0, 5.7). However, for infants who slept prone and whose mothers smoked during pregnancy compared to infants who slept supine and whose mothers did not smoke during pregnancy, the odds ratio was 55.3 (95% CI 24,127).

In our study we could not assess for the interaction. If there had been an unmeasured statistically significant interaction in our study, then a reduced prevalence of prone sleeping would have caused the odds ratio for SIDS mortality comparing smokers to nonsmokers to decrease. Thus, the decreased odds ratio we observed from 1991-93 to 1994-96 may be explained by an interaction between maternal smoking during pregnancy and prone sleeping.

The results from this study are contrary to those of a Scandinavian study which reported that the odds ratio for SIDS mortality comparing infants born to mothers who smoked during pregnancy to infants whose mothers who did not smoke during pregnancy increased from 4.3 to 6.5 following national SIDS interventions in Sweden, Denmark, and Norway (Wennergren, et al., 1997). The authors hypothesized that as the prevalence of the prone sleeping position was reduced, SIDS cases may have been increasingly

drawn from a group in whom other risk factors, such as smoking and bottle-feeding, were more frequent. They observed that among the cases, bottle-feeding (a risk factor for SIDS in their study) increased from 35% to 55% but they did not report if the prevalence of smoking changed among the cases.

In this study we found that it was important to adjust for Medicaid/OHP coverage and marital status when calculating the odds ratio for maternal smoking during pregnancy. Recently, several studies have examined the association between publicly funded health insurance and SIDS mortality. Data from the Pregnancy Risk Assessment Monitoring System (PRAMS) showed that prone sleeping was more common among mothers with publicly funded health insurance than among mothers with privately funded health insurance (Center for Disease Control, 1999). A study of Philadelphia clinics and private practices reported that 66% of the infants seen in clinics slept in a nonprone sleep position while 84% of infants in private practices slept in the prone sleep position (Gibson, et al., 2000). 93% of clinic patients had publicly funded medical assistance compared to 3% of the private patients so that the type of practice was used as a proxy for type of insurance in their analysis.

In our study, it was observed that the risk of SIDS associated with Medicaid/OHP coverage was even higher in 1994-1996 than it was in 1991-1993 (Table 5). Perhaps women with Medicaid/OHP coverage were less likely to receive or follow recommendations regarding sleep position from their health providers.

Marital status was also found to be a significant confounder of the association between maternal smoking and SIDS. It is generally believed that marital status operates through other risk factors such as young maternal age and lower socioeconomic status

(Guntheroth, 1995). However, after controlling for maternal age, Medicaid/OHP and other risk factors, marital status remained as a significant risk factor for SIDS with infants born to single or divorced women at a higher risk than infants born to married or separated women. The increased risk of SIDS associated with a single marital status may be related to infant sleep position practices. Taylor and Davis (1996) found that single mothers were less likely to be aware of the danger associated with the prone sleep position and were also more likely to use the prone sleep position than married mothers.

Despite the reduction in PAR% it is clear that maternal smoking during pregnancy remains an important modifiable risk factor for SIDS in Oregon. The drop in PAR% was only moderate and the adjusted odds ratio was still high in 1994-1996. 44 of the 160 SIDS deaths in 1994-1996 can be attributed to maternal smoking. It remains important to encourage smoking cessation for women during and after pregnancy. The results of a study examining the effects of smoking cessation suggest that it may be potentially beneficial to reduce the number of cigarettes smoked during pregnancy among people who are unable to quit (Alm, et al., 1998).

Efforts to reduce maternal smoking during pregnancy are particularly relevant to Oregon where the prevalence of maternal smoking has consistently been higher than the national average in Oregon. Nationally, maternal smoking during pregnancy has declined every year since 1990, from 18.4% in 1990 to 13.6% in 1996 (Mathews, 1998). In comparison, the Oregon prevalence of maternal smoking during pregnancy was 22.3% in 1990 and only declined to 17.8% in 1996 (Mathews, 1998).

Maternal age

Early in the course of analyzing the data to answer the maternal smoking hypothesis, it became clear that the decrease in SIDS rates in infants born to mothers<20 years old was lagging behind the dramatic drops observed among infants born to older mothers (Table 7). In addition, while the prevalence of young motherhood only changed from 12.4% in 1991-1993 to 13.0% in 1994-1996, the proportion of SIDS deaths that were born to mothers<20 years old rose from 23% to 33%. We felt it was valid to further explore this hypothesis with the understanding that any findings could not be viewed as conclusive due to the *a posteriori* nature of the hypothesis but solely as a hypothesis generating endeavor.

The risk of SIDS in 1994-1996 compared to 1991-1993 was calculated for each maternal age group (Table 8). While each age group showed a decreased risk in 1994-1996 compared to its respective maternal age group in 1991-1993, the decrease was not statistically significant for infant born to mothers<20 years old. In contrast, infants born to mothers 20-29 years old and infants born to mothers >29 years old experienced statistically significant declines in their risk of SIDS in 1994-1996 compared to their risks in 1991-1993.

These results raise the question as to why infants born to younger mothers did not decrease their risk of SIDS in 1994-1996. It does not appear that trends in the prevalence of risk factors for younger mothers differed dramatically from other mothers. For example, the prevalence of maternal smoking during pregnancy showed similar decreases in all three maternal age groups. Maternal smoking among mothers<20 years old declined from 30.6 % in 1991-1993 to 27.8 % in 1994-1996, a 9.2 % decrease. In mothers 20-29

years old, the prevalence dropped from 21.4 % to 18.6 %, a 13% decrease. Among mothers>29 years old the prevalence of maternal smoking during pregnancy fell from 14.4 % to 13.0 %, a 9.7 % decrease. Furthermore, potential confounding by numerous risk factors was controlled for in the multivariate logistic regression analysis when calculating the odds ratios for SIDS mortality associated with maternal age (Table 8).

While it does not appear that differences in smoking habits during pregnancy can explain why the risk of SIDS did not drop for young mothers, it is still a source of concern. Nationally, 17.2% of mothers age 15-19 reported that they smoked during pregnancy in 1996 (Matthews, 1998). In Oregon, 28.4% of mothers age 15-19 reported smoking during pregnancy in 1996. It is also important to note that smoking rates declined steadily in mothers<20 years old from 1991-1994 but these declines reached a plateau in 1994 and even rose from 1995 to 1996. Averaging the smoking prevalence over the time periods masked these disturbing trends.

It is possible that infant sleep position may be confounding the relationship between maternal age and SIDS. If young mothers were more likely than older mothers to continue using the prone sleep position in 1994-1996 then it would be expected that that the SIDS risks for young mothers would remain elevated. There is evidence that the increasing importance of maternal age as a risk factor can be explained by infant sleep position trends. Some studies have reported results that suggest that young mothers are more likely to place their babies in the prone sleep position.

Lesko and others (1998) studied sleep position in infants at one month of age and also assessed changes in the sleep position used at one month to the sleep position used at three months of age. Comparing mothers <18 years old to mothers ≥35 years old, they

observed that younger mothers were one and a half times more likely to place their infants in the prone position at one month (OR=1.6, 95% CI 1.0, 2.3) and were over twice as likely to change from using a nonprone position to the prone position by 3 months (OR=2.2, 95% CI 1.4,2.2). Willinger and associates (1998) reported that younger women were more likely to use the prone sleep position compared to women ≥30 years old; the odds ratio for mothers age 20-29 years old was 1.28 (95% CI 1.09, 1.50) and the odds ratio mothers<20 years old was 1.09 (95% CI 0.72, 1.66).

Studies by Taylor and Davis (1996) and Ponsonby and others (1994) suggest that young mothers are less likely to have changed their infants' sleep position as a result of national campaigns. Ponsonby and others (1994) conducted a study in Tasmania following a national campaign and showed that young mothers (<20 years old) were more likely to be unaware of the increased risk associated with the prone sleep position than older mothers. However, they did not report the association between maternal age and the actual sleep position used. Taylor and Davis (1996) found that among parents who were aware of the dangers of the prone sleep position, mothers <20 years old were ten times as likely to still use the prone sleep position when compared to women ≥ 20 years old (95% CI 1.1-107.0). They also found that among mothers who were not aware of the recent advice, young motherhood was no longer associated with the prone sleep position.

These findings provide some support to the hypothesis of this study that younger women have not learned to put their babies to sleep on their backs. The prone position has clearly declined in prevalence, starting as early as 1992, which provides evidence that the AAP recommendations and the Back to Sleep campaign have had a substantial effect

on sleep position in the total population. However, the risks for SIDS mortality among teen mothers remains alarmingly high and may be related to the continued use of the prone sleep position among this subgroup. Perhaps the most disturbing finding is that even after becoming aware of the danger of the prone sleep position, young mothers are more likely to continue to place their infants to sleep in the prone position (Taylor and Davis, 1996).

It is also disturbing that physicians may not be actively promoting the use of a non-prone sleep position. Willinger and associates (2000) observed that in 1997-1998, 40.7% of caregivers received no recommendation of sleep position from the infant's physician. In the same study it was found that a physician's recommendation was the strongest predictor of not using the prone sleep position. The medical community in the United States was slow to respond to the AAP recommendations and the Back to Sleep message in comparison to other countries who experienced much more dramatic and immediate changes in infant sleep practices (Gibson, et al., 2000). This was due to concerns about possible adverse effects of the supine sleep position, the methodological limitations of the existing studies supporting the increased risk of the prone sleep position, and the differences in risk factors and infant care practices between the United States and other countries (Gibson, et al., 2000).

A 1996 survey of family practitioners revealed that only 62% of physicians surveyed usually or always recommended the supine sleep position while 21% actually discouraged the supine sleep position (Gibson et al., 2000). Increased efforts to reduce the prone sleep position by physicians have a large potential impact on SIDS mortality.

Studies have reported that sleep position choice is significantly associated with race/ethnicity. Willinger and associates (2000) found that African-American mothers were positively associated with using the prone sleep position; compared to Caucasian mothers the odds ratio for using the prone sleep position was1.91. The association is independent of whether or not the caregiver reported receiving a recommendation of using the supine position or of sociodemographic characteristics such as maternal age, education level, and family income. Willinger and associates (2000) suggested that cultural factors may play a role based on the observation that having a grandmother in the home almost doubled the risk of the prone sleep position. PRAMS data indicates that African-American mothers were twice as likely as white mothers to place their infants on their stomachs (Centers for Disease Control, 1998).

A study of Philadelphia clinics and private practices found that 67% of African-American infants slept in the nonprone sleep position compared to 82% of non-African-American infants, a statistically significant difference (Gibson, et al., 2000). It is important that further research is conducted to determine the reasons for the higher prevalence of prone sleeping reported by Black mothers in order to effectively reach this group of mothers with the "Back to Sleep" message. In our study, race/ethnicity was not a statistically significant predictor of SIDS mortality in the final multivariate logistic regression model because of the small number African-American infants in the study population.

Conclusions

It is vital that efforts to further reduce the prevalence of prone sleeping and SIDS mortality are implemented. Prone sleeping remains far too common in the United States.

While recent data on sleep position is encouraging, further reductions in the prevalence of the prone sleep position can be achieved. In the United States, the National Institutes of Health (NIH) estimates that the prevalence of the prone sleep position has decreased from 70% in 1992 to 24% in 1996 (Willinger, et al., 2000). According to unpublished Oregon PRAMS data, the use of the prone sleep position in Oregon was 9.3% in 1998-1999. The national SIDS rate has fallen from 1.16 per 1000 live births in 1993 to 0.77 per 1000 live births in 1997 (CD Wonder, 2000). In Oregon, the SIDS rate has fallen from 2.14 per 1000 live births in 1993 to 0.86 in 1997 (CD Wonder, 2000).

In comparison to the United States, New Zealand was able to reduce the prevalence of prone sleeping to 0.7% following a national SIDS reduction campaign (Mitchell, et al., 1997). Continuing and increasing the spread of the Back to Sleep message is of particular importance to Oregon, which has the second highest SIDS rate among Caucasian infants in the United States (CD Wonder, 2001).

The results of this study provide guidance in expanding and improving efforts to reduce the prevalence of SIDS. Although the PAR% for maternal smoking during pregnancy did show a decrease, it continues to be an important risk factor for SIDS and should be emphasized. It was the strongest modifiable risk factor for SIDS with an odds ratio of 2.47 for smokers compared to nonsmokers. Prenatal alcohol use was also strongly associated with SIDS (Table 8) and it may be important to include the cessation of alcohol use in future recommendations to prevent SIDS.

The results also suggest that young mothers may not have received or adopted the recommendations advising against the prone sleep position. Although the reasons for this are unclear, it may be that teenage behavior requires a more intensive intervention or that

teenagers are not fully cognizant of the seriousness of the outcome associated with prone sleeping (Taylor and Davis, 1996). Additional research is necessary and appropriate interventions should be developed to increase the nonprone sleep position practices in teenage mothers. Finally, it appears that mothers<20 years old did not reduce their prevalence of smoking during pregnancy from 1994-1996. Efforts to assist these mothers to quit smoking may prevent many SIDS deaths.

Limitations

The accuracy of substance abuse information on birth certificates has been questioned. A study of 1994 Oregon birth certificates clearly showed that prenatal tobacco and alcohol use prevalence was substantially underreported (Glick, et al., 1998). While birth certificate data indicated that 18.1% of women smoked during pregnancy in 1994, it was estimated that the true prevalence of prenatal tobacco use was 24.0% (Glick, et al., 1998). The estimated prevalence of maternal alcohol use during pregnancy was 21.1% compared to 2.7% reported on birth certificates. Underreporting of prenatal maternal smoking and alcohol use could lead to biased estimates of the increased risk for SIDS mortality associated with these risk factors.

The maternal smoking during pregnancy analyses of this study was also limited by the format of the data regarding tobacco use. The birth certificate question regarding tobacco use is in the form of a Yes/No question that does not include any information on the number of cigarettes smoked per day. We were also unable to control for post partum smoking.

A potential problem of this study is in using the 1991-1993 and 1994-1996 birth cohorts as the time periods to assess the epidemiology of SIDS before and after the Back

to Sleep campaign. The AAP first made recommendations in 1992 and the actual "Back to Sleep" campaign was not officially kicked off until June of 1994. It may not be possible to date precisely the onset of effective publicity and implementation of the campaign.

Finally, SIDS research has always had the potential problem of misclassification of SIDS deaths. SIDS deaths have been misclassified as accidental suffocation and infanticide among other causes.

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Table 1- Risk Factor Coding and Description

*all data obtained from infant birth data sets for 1991-1996

Risk Factor	Coding
Any Maternal Smoking During Pregnancy	1 = yes 0 = no
Maternal Age	1= < 20 years old 2 = 20-29 years old 3 = >29 years old
Single Maternal Marital Status	1 = single or divorced 0 = married or separated
Mother's Race/Ethnicity	1=Hispanic 0= Non-Hispanic (NH): 1 = NH African-American 2 = NH Native American 3 = NH Asian/Pacific Islander 4 = Other NH Races 5 = NH White
Medicaid/Oregon Health Plan Coverage of Birth	1 = yes 0 = no
Any Alcohol Use During Pregnancy	1 = yes 0 = no
Any Heroin, Cocaine, or Methamphetamine Use During Pregnancy	1 = yes 0 = no
Inadequate Prenatal Care	1 = <5 prenatal visits or began in month 7-9 0 = >4 prenatal visits or began in month 1-6
Urinary Tract Infection During Pregnancy	1 = yes 0 = no
Birth Order	1 = first live birth 2 = second live birth 3 = third or higher birth
Low Birthweight	1 = low birthweight (<2500 g) 0 = normal birthweight (>2499 g)
Infant's Sex	1 = male 0 = female
Season of Birth	1 = born August - December 0 = born January - July

Table 1- Risk Factor Coding and Description

Time Period of Birth	1 = born 1991-1993 0 = born 1994-1996
Maternal Education	1 = <12 years 0 = >11 years

Table 2. Prevalences of Risk Factors for SIDS

	1991-1993		1994-1996		Total		P-value*
	number	%	number	%	number	%	
Maternal Age							p<.001
<20	15593	12.38%	16663	13.00%	32256	12.69%	
20-29	69716	55.35%	69149	53.94%	138865	54.64%	
30+	40631	32.26%	42361	33.04%	82992	32.65%	
unknown	25	0.02%	19	0.01%	44	0.02%	
Maternal Smoking During Pregnancy							p<.001
Yes	25394	20.16%	22890	17.86%	48284	19.00%	
No	99768	79.20%	104691	81.67%	204459	80.45%	
unknown	803	0.64%	611	0.48%	1414	0.56%	
Marital Status							p<.001
married/separated	91603	72.72%	90815	70.84%	182418	71.77%	
single/divorced	34341	27.26%	37301	29.10%	71642	28.19%	
unknown	21	0.02%	76	0.06%	97	0.04%	
Mother's Ethnicity							p<.001
Hispanic	10803	8.58%	14819	11.56%	25622	10.08%	
Non-Hispanic (NH)							
NH African-American	2715	2.20%	2661	2.10%	5376	2.12%	
NH Native American	1721	1.40%	1787	1.40%	3508	1.38%	
NH Asian/Pacific Islander	3907	3.10%	4646	3.60%	8553	3.37%	
NH Other Races	71	0.10%	3	0.00%	74	0.03%	
NH White	106659	84.70%	104205	81.30%	210864	82.97%	
Unknown	89	0.10%	71	0.10%	160	0.06%	
Maternal Education							
Less <12 years	26709	21.20%	27030	21.10%	196161	77.18%	p=.665
12 or more years	97288	77.20%	98873	77.10%	53739	21.14%	
unknown	1968	1.60%	2289	1.80%	4257	1.67%	
Medicaid/OHP coverage							p=.320
Yes	43960	34.90%	45051	35.14%	89011	35.02%	
No	79774	63.33%	81078	63.25%	160852	63.29%	
unknown	2231	1.77%	2063	1.61%	4294	1.69%	

*Chi-square test

Table 2 cont.

	1991-1993		1994-1996		Total		P-value
	number	%	number	%	number	%	
Maternal Alcohol Use During Pregnancy							p<.001
Yes	4795	3.81%	3195	2.49%	7990	3.14%	
No	119427	94.81%	122910	95.88%	242337	95.35%	
unknown	1743	1.38%	2087	1.63%	3830	1.51%	
Maternal Heroin, Cocaine, methamphetamine use during pregnancy							p<.001
Yes	507	0.40%	650	0.50%	1157	0.46%	
No	125458	99.60%	127542	99.50%	253300	99.54%	
Inadequate Prenatal Care							
Yes	6973	5.54%	7227	5.64%	14200	5.59%	p=.166
No	118720	94.25%	120437	93.95%	239157	94.41%	
unknown	272	0.21%	528	0.42%	800	0.31%	
Low Birth Weight							p=.003
Yes	6445	5.12%	6898	5.38%	13343	5.25%	
No	119507	94.87%	121282	94.61%	240789	94.74%	
Unknown	13	0.01%	12	0.01%	25	<.01%	
Infant's Sex							p=.691
Male	64535	51.20%	65575	51.15%	130110	51.19%	
Female	61430	48.80%	62617	48.85%	124047	48.81%	
Birth Order							
1	50558	40.10%	53221	41.50%	103779	40.83%	p=.003
2	40884	32.50%	41004	32%	81888	32.22%	
3 or more	34486	27.40%	33933	26.50%	68419	26.92%	
unknown	37	0%	34	0%	71	0.03%	
Season of Birth							p=.002
August-December	51780	41.10%	53476	41.70%	105256	41.41%	
January-July	74185	58.90%	74716	58.30%	148901	58.59%	

Table 2 cont.

	1991-1993		1994-1996		Total		P-value
	number	%	number	%	number	%	
Maternal Urinary Tract Infection							p=.003
Yes	810	0.60%	951	0.70%	1761	0.69%	
No	125155	99.40%	127241	99.30%	252396	99.31%	
TOTAL	125965		128192		254157		

Table 3. Crude Odds Ratios of Risk Factors for SIDS from Univariate Logistic Regression Analyses, 1991-1996

Risk Factor	Odds Ratio	95% CI
Maternal Smoking During Pregnancy	4.14	3.41, 5.04
Maternal Age (>29 years old = referent)		
< 20 years old	3.64	2.72, 4.89
20-29 years old	1.68	1.30, 2.18
Marital Status (married/separated=referent)		
single/divorced	2.94	2.41, 3.58
Mother's Race/Ethnicity (Non-Hispanic Caucasian=referent)		
Hispanic	0.70	0.48, 1.02
Non-Hispanic (NH):		
NH African-American	2.41	1.55, 3.75
NH Native American		
NH Asian/Pacific Islander	0.44	0.19, 0.97
Medicaid/ Oregon Health Plan Coverage	2.78	2.27, 3.40
Maternal Alcohol Use During Pregnancy	3.44	2.48, 4.77
Heroin, Cocaine, or Methamphetamine Use During Pregnancy	2.24	0.83, 6.00
Inadequate Prenatal Care (>prenatal visits or began in month 1-6=referent)		
<5 prenatal visits or began in month 7-9	1.85	1.33, 2.57
Maternal Urinary Tract Infection During Pregnancy	2.59	1.22, 5.47
Birth Order (first birth=referent)		
first birth	referent	
second birth	1.36	1.07, 1.73
third or higher birth	1.64	1.29, 2.08
Low Birth weight(>=2500g=referent)		
low birthweight (<2500 g)	3.17	2.40, 4.18
Infant's Sex (female=referent)		
male	1.39	1.14, 1.70
Season of Birth (Born January-July=referent)		
born August - December	1.19	0.98, 1.45
Time Period of Birth (born 1994-1996=referent)		
born 1991-1993	1.50	1.23, 1.84

Table 4. Adjusted Odds Ratio for SIDS Mortality comparing Maternal Smoking During Pregnancy to No Smoking, Adjusting for Selected Confounders.

Maternal smoking + one risk factor	Maternal smoking adjusted OR (% change*)	Confounder crude OR	Confounder adjusted OR
Smoking + No risk factor	4.14	N/A	N/A
Smoking + Single Marital Status	**3.25 (21.5%)**	2.94	2.04
Smoking + Medicaid/OHP coverage	**3.28 (20.8%**	2.78	2.04
Smoking + Mother's education <12	3.59 (13.3%)	2.44	1.80
Maternal Age (>29 years old=referent)	3.73 (9.9%)		
<20 years old		3.64	2.78
20-29 years old		1.68	1.49
Maternal Alcohol Use During Pregnancy	3.87 (6.5%)	3.44	1.93
Low birthweight	3.90 (5.8%)	3.17	2.65
Mother's Ethnicity/Race (Caucasian=referent)	4.03 (2.7%)		
Hispanic		0.70	0.91
Non-Hispanic (NH)			
NH African-American		2.41	2.25
NH Native American		0.88	0.74
NH Asian/Pacific Islander		0.44	0.62
Birth Order (First birth=referent)	4.05 (2.2%)		
2nd		1.36	0.68
3rd or higher		1.64	0.90
Inadequate Prenatal Care (>4 prenatal visits or began in months 1-6)			
Yes (<5 prenatal visits or began in months 7-9)	4.05 (2.2%)	1.85	1.35
Urinary Tract Infection During Pregnancy	4.13 (0.2%)	2.59	2.30
Heroin, Cocaine, or Methamphetamine Use During Pregnancy	4.14 (0%)	2.24	1.00
Season of Birth (Jan-July=referent)			
Aug-Dec	4.15 (0.2%)	1.19	1.26

* % change represents percent change in crude odds ratio for SIDS mortality comparing maternal smoking during pregnancy to no maternal smoking during pregnancy (rude OR=4.14) after controlling for specified risk factor.

Table 5. Adjusted Odds Ratios for Maternal Smoking During Pregnancy, Marital Status, and Medicaid/OHP coverage*

Variable	1991-1993 Odds Ratio (95% CI)	1994-1996 Odds Ratio(95% CI)
Maternal smoking during pregnancy	3.16 (2.40, 4.14)	2.59 (1.85, 3.62)
Single marital status	1.83 (1.37, 2.46)	1.48 (1.03, 2.10)
Medicaid/OHP coverage	1.37 (1.02, 1.85)	2.23 (1.54, 3.23)

*Marital status and Medicaid/OHP coverage were strongest confounders of the association between maternal smoking and SIDS.

Table 6. Maternal Smoking During Pregnancy and Adjusted Population Attributable Risk Percent for SIDS Mortality.

1991-1993

PAR* = {1 – [(126/242)/3.16+ (116/242)/1] } x 100

= 36%

1994-1996

PAR = {1 – [(72/159)/2.59 + (87/159)/1]} x 100

= 28%

$$*\,\mathrm{PAR} = \left\{1 - \left[\frac{\text{proportion of SIDS cases whose mothers smoked}}{\text{Adjusted Odds Ratio for Maternal Smoking}} + \frac{\text{proportion of SIDS whose mothers did not smoke}}{1}\right]\right\} \times 100$$

Table 7. Maternal Age SIDS Rates per 1000 live births.

Age categories	1991-96	1991-93	1994-96	% decrease 1991-93 to 1994-96
<20	3.38	3.66	3.12	14.75%
20-29	1.56	1.97	1.16	41.12%
>29	.93	1.21	0.66	45.45%

Table 8. Adjusted Odds Ratios of Risk factors for SIDS mortality from Multivariate Logistic Regression Analysis.

Risk Factor	95% CI Odds Ratio	Lower	Upper	Significance
Maternal age				
<20 years old, 1994-1996	0.91	0.62	1.33	0.6354
<20 years old, 1991-1993	referent			
20-29 years old, 1994-1996	0.61	0.46	0.81	0.0005
20-29 years old, 1991-1993	referent			
>29 years old, 1994-1996	0.56	0.35	0.90	0.0153
>29 years old, 1991-1993	referent			
Single Marital Status	1.52	1.20	1.92	.0005
Maternal Smoking	2.47	1.99	3.07	<.0001
Maternal Alcohol use	1.85	1.32	2.61	.0004
Medicaid/OHP coverage	1.35	1.06	1.70	.0134
Season of Birth	1.26	1.04	1.54	.0192
Birth Order				
1	referent			
2	1.91	1.47	2.47	<.0001
3 or more	2.41	1.82	3.19	<.0001
Low birth weight	2.57	1.95	3.39	<.0001
Infant's sex (male)	1.44	1.18	1.76	.0003