Incidence and determinants of sudden infant death syndrome: a population-based study on 37 million births

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Background: **The objective of our study is to measure the incidence of sudden infant death syndrome (SIDS), estimate the birth to death interval, and identify associated maternal and infant risk factors.**

Methods: **We carried out a population-based cohort study on 37 418 280 births using data from the Centers for Disease Control and Prevention's "Linked Birth-Infant Death" and "Fetal Death" data files from 1995 to 2004. Descriptive statistics and cox-proportional hazard models were used to estimate the adjusted effect of maternal and newborn characteristics on the risk of SIDS.**

Results: **There were 24 101 cases of SIDS identified for an overall 10-year incidence of 6.4 cases per 10 000 births. Over the study period, the incidence decreased from 8.1 to 5.6 per 10 000 and appeared to be most common among infants aged 2-4 months. Risk factors included maternal age <20 years, black, non-Hispanic race, smoking, increasing parity, inadequate prenatal care, prematurity and growth restriction.**

Conclusions: **While the incidence of SIDS in the US has declined, it currently remains the leading cause of post-neonatal mortality, highlighting an important public health priority. Educational campaigns should be targeted towards mothers at increased risk in order to raise their awareness of modifiable risk factors for SIDS such as maternal smoking and inadequate prenatal care.**

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Introduction

Sudden infant death syndrome (SIDS) is one of the leading causes of death in the first year of life in newborns and infants.[1] According to the 1989 definition, SIDS is defined as the sudden and unexpected death of an infant aged less than 1 year, with onset of the lethal episode apparently occurring during sleep, that remains unexplained after a thorough investigation including performance of a complete autopsy, and review of the circumstances of death and the clinical history.[2] The cause of SIDS is unknown, however several risk factors have been found to play an important role in the etiology of SIDS.

Few studies have been sufficiently powered to accurately estimate the incidence of SIDS and the effect of maternal and infant characteristics as well as pregnancy and labor/delivery factors on SIDS risk. The present large population-based study aims 1) to describe the incidence of SIDS in the US and measure the trend over the last decade, 2) to profile the timing of SIDS occurrence during the course of the year following birth, and 3) to examine the baseline and clinical factors associated with SIDS as potential risk factors.

Methods

This retrospective cohort study was based on 10 years of birth and infant death records compiled by the National Center for Health Statistics (NCHS) at the Center for Disease Control and Prevention (CDC). The NCHS database contains birth certificates for all births of residents and non-residents occurring in the United States, linked with death certificate records for all infant deaths less than one year of age. We extracted records from 1995 to 2004 from the "Birth Cohort Linked Birth-Infant Death" data files which contain a denominator file (for all live births) and a numerator file (for all infant deaths with birth certificate information in addition to more extensive death certificate details such as cause of death). For our final data set, we combined the numerator files with the denominator files and deleted records in the denominator file that were also contained in the numerator file. We excluded all births with a reported or unknown congenital or chromosomal abnormality

(*n*=2 475 473) as the co-existing congenital malformation and heart deformity were the likely causes of death. We additionally excluded all births below 22 weeks of gestation (*n*=42 160), as the survival rate below 22 weeks is extremely rare secondary to severe prematurity.

SIDS was identified as ICD-9 code "798" or "798.0" for years 1995-1998 and ICD-10 code "R95" for years 1999-2004. Age of infant death was recorded as the number of weeks since birth. Baseline characteristics were measured and maternal comorbidities were examined. We used an index developed by Kotelchuck and VanderWeele[3-5] to characterize prenatal care, which combined the adequacy of initiation of prenatal care and the adequacy of received prenatal care. Normal weight, intrauterine growth restriction, small for gestational age, and large for gestational age were defined according to gender-specific cut points for birth weight and weeks' gestation proposed by Kramer,[6] and were mutually exclusive categories. The following variables in the National Center for Health Statistics database contained imputed values to fill in missing birth certificate information: maternal age, race, marital status, and infant gender. Imputed values were less than 1% for all of these variables.[7]

Cumulative incidence was estimated as the number of SIDS cases divided by the number at risk in a given period. For descriptive purposes, we tabulated risk factors for cases with SIDS compared to those without SIDS and reported *P* values calculated by the chi-square test. Rate ratios for SIDS were calculated using a cox-proportional hazards model. In this model, the time from birth until occurrence of SIDS was used as the outcome variable. Infants who died from other causes within the first year were censored at the time of death. Those who were alive after one year of follow-up were censored at one year.

We constructed multivariable models in order to adjust for confounding factors. It has been shown that combining certain variables in a model, such as pregnancy factors with baseline characteristics to predict SIDS risk, results in biased risk estimates for baseline factors.[8] Under the assumption that baseline factors have a causal effect on pregnancy factors as well as on SIDS occurrence, the effects of baseline factors would be hidden in the effects of pregnancy factors for SIDS risk. For this reason, we created models at different stages of the causal pathway from baseline characteristics to SIDS occurrence. The three stages that we defined were: baseline characteristics, pregnancy, and labor/delivery factors. At each stage, we created a model adjusting for all variables at that stage and at previous stages. For each model, we reported only the risk ratios pertaining to the variables in that stage. Risk factors with rate ratios estimated to be between 0.9 and 1.1 and without a plausible mechanism of effect were considered to be clinically insignificant. Analysis was conducted using SAS statistical software and graphs were created with Excel 2010 and SAS.

Results

An initial 39 929 022 records were extracted, with the number of records ranging from 3.5 to 3.9 million for a single year. Fetal deaths were not included. There were 37 418 280 remaining records that met our study inclusion criteria. We identified a total of 24 101 SIDS infants between 1995 and 2004 inclusively. The annual incidence of SIDS is plotted in Fig. 1. In 1995, the incidence was 8.3 cases per 10 000 deliveries (CI=8.0-8.6) which declined to 5.6 cases per 10 000 deliveries (CI=5.2-5.7) in 2001 and remained stable through 2004. Among all SIDS cases, we calculated the number of weeks between birth and

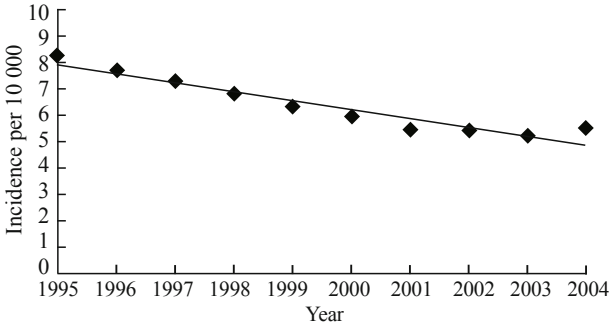

Fig. 1. Annual incidence of sudden infant death syndrome (per 10 000 deliveries) from 1995 to 2004.

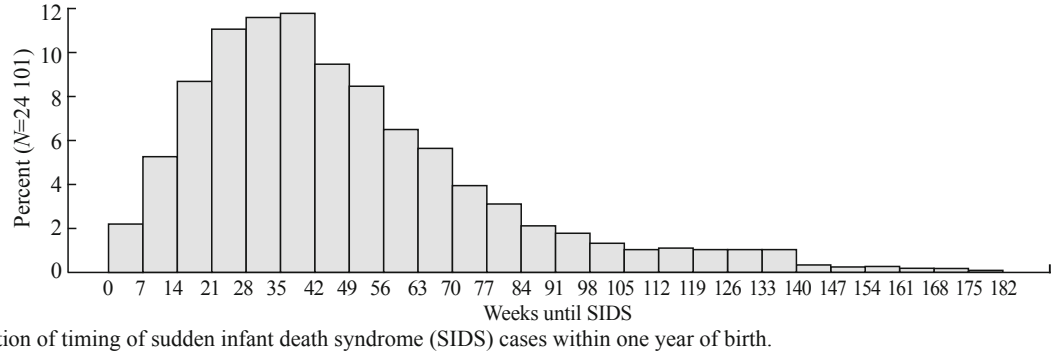

Fig. 2. Distribution of timing of sudden infant death syndrome (SIDS) cases within one year of birth.

Table 1. Baseline characteristics, pregnancy, labor and delivery factors

Variables	Cohort of live births without SIDS, *n*=37 394 179		SIDS, *n*=24 101		Chi-square test
	n	%	*n*	%	*P* value
Maternal age, y					
<20	4 401 930	12	5706	24	<0.001
20-24	9 307 726	25	9162	38	
25-29	10 113 530	27	4923	20	
30-34	8 663 242	23	2845	12	
35-39	4 067 213	11	1252	5	
40+	840 538	2	213	1	
Maternal race					
White, non-Hispanic	21 910 331	59	13 198	55	<0.001
Black, non-Hispanic	5 496 254	15	7082	29	
Hispanic	7 560 631	20	2588	11	
Other	2 052 094	5	1020	4	
Unknown	374 869	1	213	1	
Maternal education					
Less than high school diploma	8 088 349	22	9279	39	<0.001
Graduated from high school	11 783 984	32	8834	37	
1-3 y of college	8 033 924	21	3763	16	
4+ y of college and beyond	8 982 829	24	1810	8	
Marital status					
Married	24 999 226	67	9888	41	<0.001
Unmarried	12 394 953	33	14 213	59	
Maternal tobacco use					
No	27 236 346	73	13 467	56	<0.001
Yes	3 796 208	10	7715	32	
Unknown	6 361 625	17	2919	12	
Maternal alcohol use					
No	31 521 369	84	21 016	87	<0.001
Yes	321 187	1	579	2	
Unknown	5 551 623	15	2506	10	
Number of prior births					
0	15 015 009	40	6980	29	<0.001
1	12 110 201	32	7968	33	
2	6 183 468	17	4841	20	
3+	3 952 283	11	4211	17	
Prior c-section					
No	33 107 788	89	20 996	87	<0.001
Yes	4 148 202	11	3022	13	
Prenatal care, Kotelchuck's index					
Inadequate	4 191 373	11	5355	22	<0.001
Intermediate	5 027 002	13	3048	13	
Adequate	15 584 439	42	7267	30	
More than adequate	11 186 354	30	7176	30	
Plurality					
Single	36 271 081	97	22 870	95	<0.001
Twin	1 060 552	3	1195	5	
Triplet+	62 546	0.2	36	0.2	
Maternal comorbidities					
Pregnancy-associated hypertension	1 367 356	4	939	4	0.045
Diabetes	1 063 033	3	555	2	<0.001
Induced labor	7 173 736	19	4001	17	<0.001
Complications					
Rupture	920 671	2	977	4	<0.001
Abruption	197 584	1	272	1	<0.001
Placenta previa	119 826	0.3	102	0.4	0.005
Infant gender					
Male	19 092 723	51	14 300	59	<0.001
Female	18 301 456	49	9801	41	
Weeks of gestation					
22≤wk<28	216 900	1	319	1	<0.001
28≤wk<32	432 129	1	895	4	
32≤wk<37	3 578 596	10	4257	18	
37≤wk<40	18 085 026	48	10 726	45	
40≤wk<41	7 906 930	21	3858	16	
≥41 wk	6 798 399	18	3756	16	
Birth weight (kg)					
<2.5	2 745 106	7	4773	20	<0.001
2.5≤weight<4	31 016 949	83	18 124	75	
≥4	3 623 382	10	1196	5	
Weight adjusted for gestational age					
Normal weight	28 601 023	76	16 744	69	<0.001
Intrauterine growth restriction	1 547 958	4	2264	9	
Small for gestational age	2 795 349	7	2873	12	
Large for gestational age	4 449 849	12	2220	9	

SIDS: sudden infant death syndrome.

Table 2. Effect of baseline characteristics on sudden infant death syndrome

Baseline characteristics	Unadjusted RR (95% CI)	Adjusted RR (95% CI)	Adjusted *P* value
Maternal age, y			
<20	2.67 (2.57, 2.77)	2.74 (2.61, 2.87)	<0.001
20-24	2.02 (1.95, 2.09)	1.78 (1.72, 1.85)	<0.001
25-29	1 (reference)	1 (reference)	
30-34	0.68 (0.64, 0.71)	0.71 (0.68, 0.75)	<0.001
35-39	0.63 (0.60, 0.67)	0.61 (0.57, 0.65)	<0.001
40+	0.52 (0.45, 0.60)	0.47 (0.41, 0.54)	<0.001
Maternal race			
White, non-Hispanic	1 (reference)	1 (reference)	
Black, non-Hispanic	2.15 (2.09, 2.21)	1.41 (1.36, 1.46)	<0.001
Hispanic	0.57 (0.55, 0.59)	0.46 (0.44, 0.48)	<0.001
Other	0.83 (0.77, 0.88)	1.03 (0.97, 1.10)	0.349
Unknown	0.95 (0.83, 1.08)	0.88 (0.76, 1.00)	0.056
Maternal education			
Less than high school diploma	1 (reference)	1 (reference)	
High school diploma	0.65 (0.64, 0.67)	0.83 (0.81, 0.86)	<0.001
1-3 y of college	0.41 (0.39, 0.42)	0.74 (0.71, 0.77)	<0.001
4+ y of college and beyond	0.18 (0.17, 0.19)	0.55 (0.52, 0.59)	<0.001
Marital status			
Married	1 (reference)	1 (reference)	
Unmarried	2.90 (2.83, 2.98)	1.46 (1.42, 1.51)	<0.001
Maternal tobacco use			
No	1 (reference)	1 (reference)	
Yes	4.11 (4.00, 4.23)	2.58 (2.50, 2.66)	<0.001
Unknown	0.93 (0.89, 0.97)	1.10 (1.01, 1.20)	0.032
Maternal alcohol use			
No	1 (reference)	1 (reference)	
Yes	2.71 (2.50, 2.94)	1.45 (1.34, 1.58)	<0.001
Unknown	0.68 (0.65, 0.71)	1.05 (0.96, 1.15)	0.311
Number of prior births			
0	1 (reference)	1 (reference)	
1	1.41 (1.37, 1.46)	1.95 (1.88, 2.02)	<0.001
2	1.68 (1.62, 1.75)	2.54 (2.43, 2.64)	<0.001
3+	2.29 (2.21, 2.38)	3.59 (3.43, 3.76)	<0.001
Prior c-section			
No	1 (reference)	1 (reference)	
Yes	1.15 (1.11, 1.19)	1.08 (1.04, 1.13)	<0.001

RR: relative risk; CI: confidence intervals.

Table 3. Effect of pregnancy factors on sudden infant death syndrome adjusted for baseline and labor and delivery factors

Pregnancy factors	Unadjusted RR (95% CI)	Adjusted RR (95% CI)	Adjusted *P*-value
Prenatal care, Kotelchuck's index			
Inadequate	2.75 (2.65, 2.85)	1.50 (1.45, 1.56)	<0.001
Intermediate	1.30 (1.25, 1.36)	1.12 (1.08, 1.17)	<0.001
Adequate	1 (reference)	1 (reference)	
More than adequate	1.38 (1.34, 1.43)	1.24 (1.20, 1.28)	<0.001
Plurality			
Single	1 (reference)	1 (reference)	
Twin	1.81 (1.71, 1.92)	1.66 (1.57, 1.77)	<0.001
Triplet+	0.95 (0.69, 1.32)	1.48 (1.06, 2.05)	0.020
Maternal comorbidities			
Pregnancy-associated hypertension	1.07 (1.00, 1.14)	1.19 (1.11, 1.27)	<0.001
Diabetes	0.81 (0.74, 0.88)	1.04 (0.96, 1.13)	0.346
Infant gender			
Male	1.40 (1.36, 1.44)	1.40 (1.37, 1.44)	<0.001
Female	1 (reference)	1 (reference)	

RR: relative risk; CI: confidence intervals.

Table 4. Effect of labor and delivery factors on sudden infant death syndrome adjusted for baseline and pregnancy factors

Labor & delivery factors	Unadjusted RR (95% CI)	Adjusted RR (95% CI)	Adjusted *P* value
Induced labor	0.84 (0.81, 0.87)	0.96 (0.93, 1.00)	0.025
Complications			
Premature rupture of membrane	1.70 (1.60, 1.81)	1.16 (1.09, 1.24)	<0.001
Abruption	2.24 (1.98, 2.52)	1.04 (0.92, 1.18)	0.519
Placenta Previa	1.34 (1.10, 1.62)	1.03 (0.85, 1.26)	0.739
Weeks of gestation			
22≤wk<28	4.30 (3.84, 4.82)	2.04 (1.80, 2.32)	<0.001
28≤wk<32	4.37 (4.07, 4.70)	2.32 (2.12, 2.53)	<0.001
32≤wk<37	2.44 (2.34, 2.55)	1.69 (1.61, 1.79)	<0.001
37≤wk<40	1.22 (1.17, 1.26)	1.16 (1.12, 1.21)	<0.001
40≤wk<41	1 (reference)	1 (reference)	
≥41 wk	1.13 (1.08, 1.19)	1.00 (0.95, 1.04)	0.828
Birth weight (kg)			
<2.5	3.08 (2.98, 3.18)	1.56 (1.49, 1.64)	<0.001
2.5≤weight<4	1 (reference)	1 (reference)	
≥4	0.57 (0.53, 0.60)	0.85 (0.79, 0.91)	<0.001
Weight adjusted for gestational age			
Normal weight	1 (reference)	1 (reference)	
Intrauterine growth restriction	2.53 (2.42, 2.64)	1.51 (1.44, 1.59)	<0.001
Small for gestational age	1.76 (1.69, 1.83)	1.33 (1.28, 1.39)	<0.001
Large for gestational age	0.85 (0.82, 0.89)	0.85 (0.80, 0.90)	<0.001

RR: relative risk; CI: confidence intervals.

SIDS death and its distribution is presented in Fig. 2. Fifty percent of all cases occurred between 7.6 and 17.6 weeks after birth (25th and 75th percentiles). Only 10% of cases occurred after 24.7 weeks.

Maternal baseline characteristics of infants with and without SIDS were measured (Table 1). Factors most strongly associated with an increased risk of SIDS in the adjusted model for baseline characteristics were maternal age <20 years, tobacco use, having two and three or more prior births. The most protective baseline factors against SIDS were maternal age ≥40, Hispanic race and four years or greater of college and beyond. Maternal alcohol use increased the risk of SIDS by 45%. Infants of unmarried mothers had a increased risk of 46% for SIDS (Table 2).

Inadequate prenatal care and male infant gender increased the risk of SIDS by 50% and 40% respectively. A multiple birth was also found to be a significant risk factor for SIDS, with twin births increasing SIDS risk by 66% (Table 3). Twenty-two to 28 weeks gestation and 28 to 32 weeks gestation doubled the risk of SIDS. Occurrence of SIDS was greater among infants weighing less than 2500 g and growth restricted, small for gestational age infants. Birth weight of 4000 g or greater and a large weight adjusted for gestational age decreased the risk of SIDS by 15% (Table 4).

Discussion

We created a large population-based cohort using the CDC's NCHS database, enabling us to obtain sufficiently powered and reliable estimates of incidence rates and risk

factors associated with SIDS in the US between 1995 and 2004. Our results showed a temporal decline in SIDS incidence between 1995 and 2004. While decreasing post-neonatal mortality rates were observed in conjunction with declining SIDS rates, recent studies[9,10] have proposed the decline in SIDS in the US to be an artifact caused by changes in reporting practices and diagnoses. As SIDS is diagnosed by excluding other identifiable causes of death using death scene investigation and autopsy reports, a "diagnostic shift" has been noted where the incidence of SIDS was observed to be decreasing in parallel to rising rates of other sleep related deaths, such as suffocations and strangulations in bed.[10]

SIDS currently remains the leading cause of post-neonatal mortality in the US, despite the overall decline in SIDS rates in Canada and the US by more than 50% following the Back to Sleep (BTS) campaign launched in 1994 by the National Institute of Child Health and Development, which advocated a supine sleeping position for infants.[11,12] The US features among the countries with the highest incidence of SIDS, highlighting an important national public health priority.[9] Hauck et al[9] have offered several possible explanations for the significant variability in SIDS rates between countries such as differences in SIDS definitions and risk factors and the age of inclusion for SIDS. The latter likely does not have a large effect as very few SIDS deaths occur in the first month after delivery, reaching a peak at 10.7 weeks and falling markedly after 23.6 weeks.[9,13]

There have been noticeable changes to the SIDS risk profile following the effective public health campaign in the 1990s. Several hypotheses have been published regarding the involvement of different classes of risk factors and their interaction in the etiology of SIDS.[13,14] Both socio-demographic and prenatal risk factors were found to be associated with a significantly increased SIDS risk in our study. The emergence of modifiable risk factors in addition to prone sleeping placement, such as smoking, is important particularly in countries that have seen a significant decrease in prone sleep positioning.[9] Since the implementation of the BTS campaign, prenatal and postnatal exposure to cigarette smoke has become one of the leading risk factors for SIDS.[15-19]

In our study, maternal tobacco use increased the risk of SIDS by more than two-fold, which is similar to the finding from a meta-analysis by Zhang et al[16] that found a significantly increased risk of SIDS associated with prenatal maternal smoking (OR=2.25, 95% CI=2.03-2.50) and postnatal maternal smoking (OR=1.97, 95% CI=1.77-2.19). Exposure to nicotine, leading to decreased uteroplacental blood flow and chronic intrauterine hypoxia is one of the mechanisms linking maternal cigarette smoking to SIDS.[16,18,19] Another mechanism linking SIDS to cigarette smoke has looked at the interaction between nicotine and nicotinic acetylcholine receptors in the brainstem and its effect on the central nervous system. In-utero exposure to nicotine can lead to changes in the brainstem sites responsible for cardiorespiratory control and arousal from sleep. It has been previously established that impairment of both cardiorespiratory control and arousal can increase an infant's risk of SIDS.[16,18,20]

Similarly, studies[12,14,21] have suggested that prenatal exposure to alcohol can negatively affect the developing brainstem responsible for homeostatic control. In a large population-based cohort study,[21] maternal alcohol use disorder was found to increase the risk of SIDS through direct effects on the fetus and indirectly through environmental risk factors. An alcohol diagnosis during pregnancy increased SIDS risk by 3-fold and a maternal alcohol diagnosis within 1 year post-pregnancy resulted in more than an 8-fold increased risk,[21] whereas our results showed that maternal alcohol use increased the risk of SIDS by 45%.

Other baseline characteristics found to increase the risk of SIDS in our study include young maternal age, increasing number of prior births and black, non-Hispanic race. A maternal age younger than 20 years nearly tripled the risk of SIDS in our study. Younger mothers are at increased risk of having low birth weight babies, a risk factor which was found to significantly increase the risk of SIDS in our study.[22] The number of prior births was also noted to increase the risk of SIDS, with 3 or more prior births more than tripling SIDS risk. A possible explanation for this finding is that multiparous women are more likely to practice bed sharing, a known risk factor for SIDS.[23,24] In a study by Ball et al,[23] mothers with three or more prior births were 72% more likely to regularly practice bed sharing.

Black, non-Hispanic race increased the risk of SIDS by 41%, whereas Hispanic race was shown to decrease SIDS risk by 54%. Findings from the 2004 National Vital Statistics Report from the CDC noted that a higher percentage of live births in the US among black, non-Hispanic mothers as compared to white, non-Hispanic and Hispanic mothers had the following maternal and infant characteristics known to increase SIDS risk: births less than 2500 g; preterm births; births to mothers under 20 years; fourth and higher order births; and births to unmarried mothers.[25] An additional risk factor which places infants of black, non-Hispanic mothers at a greater risk of SIDS is sleep position and bed sharing. According to a study by Smith et al looking at race and infant sleep positioning in South Carolina, 60.2% of white, non-Hispanic infants were found to be placed in the supine sleep position whereas only 38.8% of black, non-Hispanic infants were positioned supinely in 2004.[26] In another study by Colson et al[27] mothers of black race were found to be 3 times more likely

to practice infant bed sharing.

Infants whose mothers received inadequate prenatal care and who suffered from pregnancy associated hypertension were more likely to have SIDS. Adequate prenatal care has been shown to result in a lower risk of prematurity and a low birth weight, both of which have been shown to increase the likelihood of SIDS.[28] Adequate prenatal care is dependent on the time at which prenatal care was initiated, the number of visits and the quality of care that was received.[28] Schlaud et al[29] found 0-3 antenatal consultations tripled the risk of SIDS (OR=2.9, 95% CI=1.44-5.84). Our results showed that inadequate prenatal care increased SIDS risk by 50%. Poor health insurance coverage in the US resulting in barriers to access and underutilization of antenatal care services may be a contributing factor to the observed increased rates of SIDS among women receiving inadequate care.

Pregnancy-associated hypertension was also shown to have a moderate effect on SIDS occurrence, possibly a consequence of the increased susceptibility to pregnancy-induced hypertension complications, such as placenta abruption, which is itself a cause of intrauterine hypoxia.

A multiple gestation is considered a form of high risk pregnancy that can be associated with many complications, most commonly prematurity and low birth weight babies, both of which are known risk factors for SIDS.[30] In our study, pregnancies with twins increased SIDS risk by 66% and with triplets the risk increased by 48%. We observed in infants, birth weight lower than 2500 g increased the risk of SIDS by 56% whereas a larger birth weight appeared to be slightly protective, reducing the SIDS risk by 15%. Bigger et al[31] also found this association and that infants with a birth weight lower than 2500 g were more than 3 times likely to have SIDS.

Preterm induction and preterm caesarean delivery have both been shown to play a major role in the observed temporal increase in preterm births in Canada and the US, particularly at 34 to 36 weeks gestation.[32] Hypoxic conditions in-utero have been found to be involved in the etiology of both unexplained stillbirths and SIDS, both of which share similar risk factors such as male gender, race and, maternal smoking.[30,32] As the majority of complications in pregnancy requiring medical intervention during delivery involve a fetal hypoxic state, the observed increase in medically indicated deliveries in Canada and the US was shown to correspond with a large reduction in stillbirth and SIDS rates.[30] Our results showed that weeks of gestation between 22 and 28 weeks and 28 to 32 weeks doubled the risk of SIDS, with a rate ratio of 2.04 and 2.32, respectively. The risk of SIDS dropped to 1.69 for infants of gestational age 32 to 37 weeks, which corresponds to the range (34 to 36 weeks) during which most medically indicated deliveries take place.[32]

There were several limitations in our study. No definitive definition for SIDS exists as it's a disease of exclusion and not a cause of death, increasing the likelihood of misclassification. Differences in reporting practices of SIDS coding may have introduced bias.[33] Shapiro-Mendoza et al[33] looked at all US NCHS mortality files from 2003 to 2004 and analyzed all deaths assigned an ICD-10 code for SIDS (R95). Death certificates were examined and they found that a range of terminology was used by certifiers to code SIDS deaths. We believe that sleep related deaths or sudden unexpected infant death should be coded similarly to SIDS, since they occur during the same period as SIDS, and their mechanism remains unknown.[34-37] It is important to note that the ICD coding for SIDS changed throughout the study period. ICD-9 code "798" was changed to "R95" following the implementation of the 10th revision of the ICD in 1999. A report examining the comparability of ICD-9 and ICD-10 coding for cause of death mortality statistics in the US found a 4% increase in SIDS attributable to the implementation of ICD-10.[38]

When defining SIDS in our study, we excluded all births with a reported or unknown congenital or chromosomal abnormality and gestation below 22 weeks, which may have introduced possible bias, however it is likely to be minimal. We acknowledged that not all risk factors for SIDS such as infant sleep position were available in the linked birth/infant death database and we could not possibly adjust for all important confounding variables. To our knowledge this is the first population-based study using the CDCs' linked birth/infant death files to yield the most number of SIDS cases and to examine the effect of maternal and infant characteristics as well as pregnancy and labor/delivery factors on SIDS risk.

While the incidence of SIDS has notably decreased over the past decade, rates remain high in countries such as the US,[9] reflecting the need to implement educational campaigns that target mothers at increased risk in order to raise their awareness of modifiable risk factors for SIDS. Reducing maternal smoking, discouraging infant bed sharing and prone sleep placement practices in addition to ensuring access to and use of adequate prenatal care services could help further lower SIDS incidence in the US.

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Contributors: HGF, OL and AHA drafted the article. HCA was involved in the acquisition and analysis of the data.

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