



The Children and Infant Sudden Death

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Abstract

The sudden unexpected infant death (SUID) usually occurs in a healthy infant and children, and the cause of death remains unclear. Sudden infant and children death syndrome (SICDS) cannot be completely prevented, but it is thought that risk can be decreased by sleeping safely. SICDS is associated with a variety of risk factors, including maternal, infant, and environmental factors. The sudden infant death rates were significantly declined from the late 1980s to early 1990s, after prevention campaigns were introduced across many countries. These campaigns appear to affect some part of our population and resulted in behavioral change; this is not totally successful. The principles and evidence for public health approaches to prevention are based on different strategies. The interventions must focus on a limited number of simple feasible interventions and deliver through programs. The programs should be resourced appropriately, based on the long term and taken on long-term leadership which could make the target communities engagement and authorization. These programs must have been performed in robust monitoring and evaluation.

Keywords

Sudden unexpected infant death (SUID) · Sudden infant and children death syndrome (SICDS) · Preventive strategy

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10.1 Epidemiology of Sudden Death in Infant and Children

The sudden unexpected infant death (SUID) usually occurs in a formerly healthy infant and children, and the cause of death remains hard to fully explain despite a thorough case analysis, including autopsy, death investigation, and evaluation of the clinical history.

In the USA, about 4000 infants die per year due to sleep-related deaths, indicated as sudden unexpected infant deaths (SUIDs) [1]. These deaths take place suddenly and may be explained or unexplained among infants less than 1 year of age, and they are usually reported as sudden infant and children death syndrome (SICDS), death from unexplained cause, and death from accidental apnea and asphyxia in bed. SICDS includes about 50% of all SUIDs and is characterized by the sudden death during the sleep of the infants and children. Most SICDS deaths take place during the first 6 months of age, especially between ages 2 and 4 months. Since the sponsor of the Back to Sleep campaign in 1994 by the American Academy of Pediatrics (AAP), the total SICDS rate has declined by more than 50% in the USA. The number of death has declined from 130 per 100,000 live births in 1990 to 40 per 100,000 live births in 2015 [1]. Nonetheless, SICDS still is the second leading cause of early infant death and the fourth leading cause of infant and children mortality in the USA [2]. In recent years, the mortality rate of SICDS has become invariant unless major public health efforts were made to improve infant's sleep environment and aimed at focusing on high-risk groups. SICDS affects families of all social, economic, culture, and ethnic spheres. However, it is more likely to occur in infants born to mothers with few or inadequate antenatal care, mothers smoking during pregnancy, male infants, prone and side-lying position during sleep, and premature and low birth weight infants [3]. SICDS is not completely prevented, but it is considered that the risks could be avoided by following the basic "ABC" of safe sleep pattern. It means having the infant sleep "alone" and not with the parents or other people and not sleep with too soft pillows or unfixed blankets, having the infant on his or her "back" and not in prone or side-lying position during sleep, and having the infant sleep in his or her own "crib" and not on an adult bed, sofa, or other very soft surface.

10.2 Etiology and Risk Factors of Sudden Death in Children and Infant

A lot of risk factors are known to cause SICDS. They can be summarized as maternal, infant, and environmental factors (see Table 10.1). Statistically, the proportion of SICDS cases related to at least one risk factor exceeds 95%. And in a number of cases, factors leading to SICDS are modifiable. Among these factors, sleep posture, sleep environment, and parental smoking are more often to be altered [4]. We will discuss these risk factors and related protection factors in detail in this chapter.

Table 10.1 Risk factors of SICDS

Maternal factors	Infant and environmental factors
Young age	Premature delivery and/or low birth weight
Cigarette smoke	Prone sleep posture
Alcohol or drug abuse	Twins
Pregnancy complications	Genetic polymorphisms
	Sleeping environment
	Sibling of SICDS victim
	History of apnea
	Overheating

10.2.1 Maternal Risk Factors

10.2.1.1 Young Maternal Age

Many studies have found that young maternal age is related to the risk of SICDS [5, 6]. A research conducted by the USA points out that after the neonatal period, SICDS occurred in 5.2 out of every 1000 infants born to teenage mothers, while the incidence in infants born by the older mothers was 1.0 per 1000 [6]. Another case-control study conducted by the Netherlands shows that the younger age of mother is an important risk factor for SICDS [7]. Compared with older mothers, very young mothers may differ in the way they take care of their children, and perhaps they have more things to worry about.

10.2.1.2 Maternal Smoking

Maternal smoking can significantly increase the risk of SICDS and is positively correlated with the amount of smoking [8]. Cigarette smoking during pregnancy would bring the most distinct impact on babies, and second-hand smoke exposure is another independent risk factor for SICDS [9]. A great deal of studies have suggested that the babies were more susceptible to SICDS if their mothers smoke during the gestation period, and smoking prevention/intervention program could greatly reduce the risk of SICDS [8, 10]. According to a case-control study of KC Schoendorf et al., infants who were surviving had less exposure to maternal smoking, compared with those who died of SICDS. They also concluded that the odds ratio (OR) for passive exposure among normal birth weight infants of black race and white race was 2.4 and 2.2, respectively, but that of combined exposure was 2.9 and 4.1. By adjusting the demographic risk factors, the ratio for SICDS in these two races went consistent. The ratio for passive exposure was approximately 2, and the value for passive exposure became 3 [11]. Another prospective follow-up study found that there were 0.8 cases of SICDS per 1000 live births ($n = 20$). Maternal cigarette smoke significantly increased the children's risk of SICDS by more than three times (OR = 3.5; 95% CI 1.4–8.7), and the higher the number of cigarettes smoked every day, the higher the risk of SICDS ($p < 0.05$) [12]. Whether in the fetal period or after birth, tobacco can hinder the normal development of physiological functions and anatomical structures, thereby greatly increasing the risk of SICDS.

10.2.1.3 Maternal Alcohol Use or Drug Abuse

Alcohol abuse by mothers can significantly increase SICDS risk [13]. A population-based case-control study of Northern Plains American Indians showed the significant associations between SICDS and pre-pregnancy maternal drinking (adjusted OR = 6.2; 95% CI 1.6–23.3), and between SICDS and early pregnancy bibulosity (adjusted OR = 8.2; 95% CI 1.9–35.3) [14]. Drug abuse by mothers can also increase the risk of SICDS [15]. Infants of mothers abusing methadone, heroin, cocaine, and other drugs during pregnancy can reach an incidence of SICDS of 5.89%, which is 4.19 times higher than infants born to mothers without drug abuse. Different drugs have different risks of increasing SICDS. Among them, SICDS risk for babies of cocaine-abusing mothers has increased threefold, heroin has increased fivefold, and methadone has increased sevenfold.

10.2.1.4 Pregnancy Complications

A variety of complications may occur during pregnancy, including placenta previa, placental abruption, premature rupture of membranes, and elevated maternal alpha-fetoprotein. The ones listed above are currently regarded as maternal-related risk factors of SICDS [15–18]. Premature delivery is another common complication of pregnancy, but does not appear to increase the risk of SICDS.

10.2.2 Infant Risk Factors

10.2.2.1 Prematurity

Compared with full-term delivery, premature delivery puts the babies at higher risk of SICDS [19, 20]. One study indicated that SICDS occurred three to four times more frequently in infants who are born with a low weight or a very low weight than that of full-term infants [21]. Donna R. Halloran et al. reported that length of gestation was closely related to the incidence of SICDS. Among all groups, infants between 28 and 32 weeks of gestation had the highest risk of SICDS (adjusted OR, 2.9; 95% CI, 2.6–3.2). The adjusted average age of postnatal SICDS death for infants born at 22 to 27 weeks was 20.9 (SD = 0.8) weeks, 28 to 32 weeks was 15.3 (SD = 0.5) weeks ($p \leq 0.002$), and 40 to 41 weeks was 14.5 (SD = 0.4) weeks [22]. From the above data, it can be seen that the mean death age of babies born at older gestational age decreased instead. Compared with term ones, very preterm infants died at an age 6 weeks later.

10.2.2.2 Low Birth Weight

SICDS risk is raised in small for gestational age infants [23, 24]. Gestational age, maternal cigarette smoke, and hypertension are all related to low birth weight. And regardless of whether these factors are adjusted or not, birning at a low weight is still weakly but significantly associated with the risk of SICDS.

10.2.2.3 Twins

Cohort studies indicated that the sudden death risk in twins was about twice as high as that in singletons [25–27]. The risk elevates partly because the proportion of premature births and low birth weight of twins is higher than that of singletons. However, other studies found that the SICDS risk for twins with gestational age of 37w and birth weight of 3000 g or more was still higher [28].

10.2.2.4 Genetic Polymorphisms

Sudden infant death syndrome has a certain correlation with genes. The existence of some unknown gene polymorphisms and gene mutations may lead to a series of different responses of individuals to the external environment and the internal body, thus increasing the rate of sudden death. Neubauer et al. recently sequenced 161 SICDS infants in 2017 and found potential pathogenic gene variations in 20% of SICDS cases. These diseases are related to ion channel diseases (9%), myocardial diseases (7%), and metabolic diseases (1%) [29].

10.2.2.5 Sleep Position

The majority of sudden deaths in infants are sleep-related, but it doesn't matter when they sleep. Sudden infant death syndrome deaths do not occur at a specific time within a day [30]. Prone sleep posture, which can cause extra physiological pressure on the cardiopulmonary system, leads to SICDS and plays a more important role than any other environmental or "external" factors. In fact, an infant's risk of SICDS in this sleeping position may be 14 times that of other sleeping positions [31]. The death mechanism attributed to prone sleep is usually asphyxia, but asphyxia cannot explain all deaths. Other mechanisms involve the changes of the blood flow, body temperature, etc. Firstly, the composition of air inhaled by the infants in the prone position changes. The oxygen they inhale decreases and the carbon dioxide increases, resulting in hypoxemia and hypercapnia. Secondly, prone position is prone to airway obstruction. Thirdly, infants may have less sleep arousal response and require more stimulation to wake them up, especially external stimuli. Fourthly, their cardiovascular capacity will change and blood flow to the brain will decrease. Finally, elevated infant body temperature can cause death, and splinting of the diaphragm is another related mechanism [32]. In contrast, supine position can reduce the risk of SICDS in premature infants. In the past, some people worried that supine position of premature infants might reduce oxygen cooperation, but two small research projects didn't support this theory, at least in babies over 32 weeks after menstruation [33, 34]. Even some studies have shown that compared with prone sleep posture, brain oxygenation is improved when lying on your back [35]. Based on the above evidence, the American Academy of Pediatrics (AAP) suggests a supine position for the premature babies. They had better start supine position from 32 weeks or earlier after menstruation [36]. In addition, side sleep may also lead to increased risk of SICDS, as side sleep can easily be converted to prone position sleep.

10.2.2.6 Sibling of SICDS Victim

Siblings of SICDS victims increased the risk of SICDS by five- to sixfold [31, 37]. However, there is no need to worry too much, because the incidence rate of SICDS is extremely low (0.06%), and the SICDS incidence rate in subsequent siblings for most families after the risk increases is less than 1%.

10.2.2.7 History of Apnea

Apnea or other respiratory dysfunction may be the final common pathway of many possible mechanisms of SICDS. Although apnea history, obvious life-threatening events (ALTE), or other respiratory pattern abnormalities cannot effectively predict the risk of SICDS, even the timely detection of apnea by a standard cardiopulmonary monitor cannot achieve a decreased SICDS risk. According to a case-control study, the history of apnea or cyanosis did not particularly increase among SICDS victims [38]. Another prospective study found no respiratory problems that could explain the elevated risk of SICDS [39].

10.2.2.8 Sleep Environment

The environment around a sleeping baby may also be related to SICDS. Pay attention to the softness of the bed surface when the baby sleeps, the pajamas, the items on the bed, the ambient temperature, and whether the baby shares a bed or a room with his or her parents. Several studies have shown that the use of soft sleep surfaces can significantly increase the risk of SICDS [40, 41]. SICDS risk can also elevate by up to five times when infants use loose bedding, especially for older infants. This association is unrelated to sleep posture and appears to be caused by soft objects covering the head or blocking airflow. In addition, studies have shown that crib bumper pads are also associated with infant deaths due to “asphyxia” [42, 43]. Therefore, the AAP and Canadian Academy of Pediatrics both advise infants not to use crib bumper pads [36, 44]. Several studies have reported that the risk of SICDS was related to bed sharing, especially for babies in the first 3 months of life [41, 45–47]. A meta-analysis used 11 case-control studies on SICDS and bed sharing and calculated an odds ratio value of 2.89, with 95% confidence interval, 1.99–4.18 [47]. Combined with other factors leading to SICDS, such as bottle feeding, parental tobacco smoke, and alcohol use, the SICDS risk of bed-sharing infants will be 15-fold higher [48]. AAP suggests infants sleeping in the same room as their parents, but not in the same bed [36]. It is a good choice to put a crib or cradle beside the parents’ bed.

10.2.2.9 Overheating

Too high room temperature, or too much wrapped clothing, can cause the baby to overheat, thus increasing the SICDS risk. A study by North American Plains Indians found a significant correlation between the incidence of SICDS and wearing at least two layers of clothing (adjusted odds ratio 6.2, 95% confidence interval 1.4–26.5) [14].

10.3 Prevention of Sudden Death in Infant and Children

The sudden infant death rates significantly declined from the late 1980s to early 1990s, after prevention campaigns were introduced across many countries, which has been marked as one of the great public health success stories in the twentieth century [49, 50]. SICDS rates in many countries during the time of introduction of the prevention programs could be seen with falls of between 42% and 92%. These campaigns appear to have effectively changed some segments of our population in behavior, this is not universal. We will summarize the principles of public health approaches to prevention and the evidence base for different strategies in this chapter. We will consider the evidence for current approaches for reducing the risk of SICDS.

10.3.1 Principle of Preventive Strategies

SICDS is a complicated phenomenon caused by multiple, interacting risk factors. As such, any single preventive approach will hardly achieve universal success. Rather, more complex and multifaceted community-based approaches may lead to further reductions in SICDS mortality. From public health approaches to injury prevention, much can be learned [50–52]. Injury prevention approaches include three domains: education, environmental modification, and enforcement of legislation or regulations [51]. Now, there can be a fourth aspect, named empowerment.

The SICDS prevention approaches can be educational most commonly. If the public are clear what are the health-promoting behaviors, they are willing to follow them. When there has some effect, they tend to be limited in their impact. It is inconsistent between changes in knowledge and actual behavior [51]. This can be demonstrated in the persistence of unsafe sleeping and parental smoking (both risk factors of SICDS), especially in some of the most vulnerable groups [53].

In SICDS prevention, approaches must be based on environmental or product modification and on enforcing legislation, which have typically been shown to have a greater improvement on outcomes [51]. Many examples exist within the published literature of successful interventions which have resulted in reductions in mortality and morbidity. As has been seen in the impact of seat belt and motorcycle helmet legislation in many countries, environmental measures are most successful when combined with legislation.

Compared with educational approaches, environmental modification or legislation are more passive approaches: once implemented, individuals do not need to repeat their behavioral changes [51]. In contrast, educational approaches depend on individuals learning the lessons, and they will consistently be implemented when they are on every occasion of potential risk.

10.3.2 Educational Approaches

10.3.2.1 Public Health Campaigns

In the late 1980s and early 1990s, the first mass public health campaigns for promoting supine sleeping were conducted after two decades of high sudden infant death rates in some countries, a period that has been described as “SICDS pandemic” [54]. At that time to avoid aspiration of vomit, prone position sleep was recommended for infants. However, observational studies found a growing evidence that SICDS was linked to prone sleeping at mid-1980s. By 1991 in the UK, Australia, and New Zealand, campaigns were conducted to encourage parents to sleep infants supine, following earlier examples in the Netherlands and Norway [49].

10.3.2.2 Sleep Supine

The prevention campaign became known as “Back to Sleep” and was conducted by the Department of Health in conjunction with the Foundation for the Study of Infant Deaths in the UK (FSID, now known as The Lullaby Trust). In this campaign, professionals can receive mass mail-outs of information and public service announcements on the radio and television [55]. This campaign successfully brought a rapid and marked decline in rate of SICDS. The successful reasons are as follows. Primarily, the intervention of sleeping infant’s supine is powerful. The SICDS rates declined dramatically with more and more parents sleeping their infant prone, which suggested the intervention was effective and responsible for the drop of SICDS deaths [56]. Second, the key message of campaigns was easy to understand and implement: avoid sleeping your infant prone. Third, the media and influential spokespeople participated in these campaigns, and public awareness and emotive case studies were heightened, which effectively conveyed that no infants are “immune” and all are at risk of SICDS [57].

SICDS is now most likely to occur in low-income families, with infants often sleeping supine and in bed-sharing situations with hazardous circumstances [54]. Recently in one study, 10–21% of infants were found to sleep on a non-recommended surface, 14–33% slept non-supine, and 87–93% were placed on loose or soft bed or other items nearby [58]. Safer sleep education and guidance are a continuous work. Noting the incidence of “sleep-related deaths” and moving away from focusing only on SICDS to a safe sleep environment, the risk of all sleep-related infant deaths can be reduced, including SICDS [59].

10.3.3 Safer Sleep Week

In 2015 a campaign named “Back to Sleep” was renewed campaigning efforts to Safer Sleep Week, which was a weeklong campaign held every March. The aim was to promote safer sleep advice for parents, professionals, and any babysitter. Activities were held during the Safer Sleep Week, which include press releases nationally and locally. Displaying the toolkits for disseminations at health and

children's centers and educational talks were also included in the activities. Safer Sleep Week successfully raised awareness of ways to reduce the risk of SICDS.

10.3.3.1 Support for High-Risk Families

There is a debate of universal provision versus targeted services in SICDS prevention. Maternal and child health service provision is significantly different among high-income countries, with models ranging from universal services free at the point of access to insurance-based provision requiring a financial contribution. The identification of populations who need to be labeled "at risk" or "in need" of service interventions is controversial. At best, targeted service provision focuses resources and supports on the more vulnerable populations to a great extent; at worst these populations are labeled and look down upon.

Young and disadvantaged mothers are usually considered a potential high-risk group, as they require targeted intervention and monitoring for lacking in parenting skills. Mothers in these groups are often supposed to be resistant to changing their behavior or infant care practices. Hence professionals have difficulty in understanding and identifying what motivates individuals in different groups, what affects behavioral change in different populations, and what kind of interventions are relevant and acceptable to different population groups. As the next section shows, several interventions successfully improved outcomes for groups with increasing risk for sudden unexpected death in infancy in varying degrees.

10.3.3.2 Parenting Support Interventions

Educational interventions that have been regarded as successful in modifying parental behavior include the Family-Nurse Partnership (FNP) model that originates from the USA [60]. This program is targeted for vulnerable, young, first-time parents and can provide intensive home visiting from early pregnancy to 1 year after childbirth. In an area of Sydney, Australia, a study that evaluated a parent support program through home visits and supported with young disadvantaged parents described the experiences of staff and identified important components that contributed to program success [61]. This intervention acknowledges that parenting happens within the context of other priorities for parents with low income and poor resources. Dealing with these issues promotes resilience and supports parents to raise their children in a safe and nurturing environment.

10.3.3.3 Support for Families with a Previous Unexpected Infant Death

Siblings born subsequently to an infant who died of SICDS in the family carry an increased risk of sudden infant death, both from explained and unexplained causes [62]. Families who have an infant who died of SICDS are more likely to have significant risk factors, including smoking, being a younger mother of higher parity, and low income, which likely exist persistently for subsequent children [63]. These factors, combined with strong feelings of anxiety at the prospect of a new infant [64], mean that families were a vulnerable group of SICDS and needed support and could benefit from targeted preventative interventions.

A study in 2011 confirmed an overrepresentation of risk factors: the smoke rate was twice in the parents and the unemployed rate was five times than the national averages and those families had a higher chance to give births [65]. Family support, such as intensive home visiting by professionals, has been proved to reduce prevalence of sudden infant death in high-risk families [66].

10.3.3.4 Peer Support Programs

At the beginning of the chapter, interventions that are modified for a specific populations are more likely to be effective and sustainable, if the populations engaged in design and implementation. However, very little research focuses on community involvement using peer educators in SICDS risk-reduction strategies. One community of particular interest is young parents whose ages are under 20. Particularly young mothers have been usually associated with an increased risk of SICDS in their children [67]. Peer support programs would reduce prevalence of sudden death in high-risk infants.

10.3.4 Approaches Based on Engineering, Environmental Modification, and Enforcement

All kinds of approaches including engineering and environmental modification have been developed to ensure safe sleep environments for infants. Equipment such as sleeping bags and safe cribs/bassinets are provided for infants and tools to support parents for early recognition of illness.

10.3.4.1 Safe Sleep Environments

Sleeping Bags

However, there is not any prospective studies until now that have evaluated the value of providing or using sleeping bags as a SICDS prevention measure for infant. Just like the risks related to prone sleeping and thick or loose bedding, intuitively at least, infant sleeping bags are a great potential approach for prevention. Thick bedding and bedding that can cover an infant's head have been shown to be risk factors for SICDS [68]. In a study, the risk of SICDS of infants using a duvet doubled compared to that sleeping with a sleeping bag or light cotton blanket [69]. However, evidence for using sleeping bags as a preventive measure for SICDS is limited until now. As a study from the Netherlands showed, cotton sleeping sacks have a protective effect [70]. Another UK study showed that sleeping bags were used more commonly in control group than that in SICDS group, but this was not significant on multivariate analysis [71].

10.3.4.2 Baby Boxes

Effective interventions can combine culture and tradition which will encourage parental behavioral change, such as the distribution of "baby boxes," an old tradition in Finland since 1938 [72]. A cardboard box, modified and repurposed as a bed,

contains a mattress and fitted sheet is used for neonates by every woman during her first pregnancy. Although these boxes were used before the SICDS “pandemic” of the 1970s and 1980s and were not associated with SICDS prevention, the low infant mortality rate in Finland [73] has been seen and noted by other countries. In recent years programs called copy-cat cardboard baby box schemes have been set up in other countries, including the USA, Scotland, and England. Some interventions are covered by the healthcare system, as in Finland, such as pilot schemes in two regions of Scotland [74], in London [75], and in Alaska [76].

10.3.4.3 Tools for Recognition of Illness

Many infants who die of SICDS, for either explained or unexplained reasons, show signs and symptoms of SICDS in the immediate 24 h before death [77]. They are most likely to present during doctors’ surgeries or in the weeks after admission to hospital [78]. Both parents and professionals can ignore the signs of serious illness sometimes, even the symptoms such as fever that can invoke in parents with high anxiety [79]. A system for assessing seriousness of illness accurately can support parents and help professionals to treat appropriately, which may help to prevent sudden infant death.

10.3.4.4 Combined Strategies for Behavioral Change: Smoking Cessation in Pregnancy

Following prone sleeping, maternal smoking is widely acknowledged as the next most important modifiable risk factor for SICDS [80]. While smoking rates in the common populations of developed countries have decreased steadily over the last 40 years, Lumley et al. identified what kinds of populations were more likely to continue to smoke and the characteristics of these groups which include lower socioeconomic status, lower educational achievement, poverty, younger women, and psychologically resilient or more marginalized and unsupported [81]. In Fleming and Blair’s study, the prevalence of smoking during pregnancy was found to decrease in the common population between 1984 and 2003 from 30% to 20%, but the proportion of smoking during pregnancy in SICDS mothers increased from 50% to 80% [82]. Thus, reducing maternal smoking among these most vulnerable populations should be a priority for SICDS prevention.

10.3.4.5 Telephone and Internet Support

It is unclear whether or not telephone and Internet-based support is successful for antenatal smoking cessation. A systematic review found there was no evidence that telephone support was more likely to have reduced, or stopped smoking at the end of pregnancy, or during the postnatal period. Even telephone support could not reduce the probability of smoking relapse [83]. Another systematic review evaluated the supportive effect of telephone helplines for smoking cessation, which found that telephone helplines can promote people to stop. However, these helplines only increase the likelihood of some persons who are ready and intend to stop smoking or want to resist relapse. So, they had more willingness than those who didn’t contact the helplines [84]. This review also revealed a “dose-response” effect in a

number of helpline contacts. Compared to a minimal intervention such as providing brief advice, standard self-help materials, or NRT, three or more calls increased the likelihood of stopping. Telephone counseling and helplines make support accessible for those who want to seek help, and therefore such interventions may be useful and measurable benefit compared to brief advice and self-help interventions.

10.3.4.6 Real-Time Feedback

Ultrasound monitoring provides visual evidence and measurement of fetal development in antenatal care, which presents an opportunity to discuss maternal behavior related to the health and development of the fetus. A study with 129 participants found that women who received detailed feedback about the growth and development of the fetus during their scan appointment were more likely to stop smoking and avoid alcohol during pregnancy [85]. However, a systematic review revealed that there was insufficient evidence to suggest that both low and high detailed feedback could result in behavioral change during the scan appointment [86].

10.3.4.7 Nicotine Replacement Therapy (NRT)

NRT may be a reasonable option to support smoking cessation during pregnancy [87]. Nicotine has significant deleterious effects on the developing fetus; it will metabolize much more quickly during pregnancy, which may protect the fetus through decreased toxic levels of circulating nicotine. Therefore, nicotine replacement products seem to be safer than smoking during pregnancy due to the reduction of exposure to the toxic levels of circulating nicotine [88]. The efficacy and safety of NRT for smoking cessation was confirmed by a systematic review of five randomized controlled trials (RCTs); there was no sufficient evidence of both efficacy and safety of its use in pregnancy with or without behavioral support [89]. However, the compliance of treatment was low across all studies, and most participants did not complete the recommended course of NRT. A subsequent meta-analysis by Myung et al. found that late pregnant smokers using NRT presented an abstinence rate 1.8 times higher than the control group; however, compared to the non-pregnant smoking population, pregnant smokers who used NRT had lower cessation rates [90]. The conclusion revealed that there was no significant impact on the birth outcomes of infants between the subject and control groups. There may be clinical supportive evidence for using NRT, and NRT was usually safe for use with pregnant women in Myung's conclusion.

10.3.4.8 Electronic Cigarettes

Since 2007, electronic cigarettes (EC) have been available and used by smokers as a cessation treatment. EC was also a more socially acceptable habit and substitute for cigarettes than cigarette smoking for non-smokers in public places [91]. Early studies have found some physiological benefits of using ECs over cigarette smoking, as they contain fewer of the toxic substance and less carbon monoxide than normal cigarettes [92]. Pregnant women may be advised by healthcare practitioners to move to ECs as a safer and more socially acceptable substitute that can encourage them for smoking cessation [93].

10.4 Conclusion

In this part, we have reviewed the epidemiology, etiology, and risk factors and the strategies of prevention of SICDS. In the 1990s, the great successes of the initial public health strategies demonstrate what can be achieved through concerted national efforts. However, after the initial rapid declines, the SICDS mortality has plateaued now. It seems clear that the approaches will now only have little effects, especially for the families with highest risk of SICDS. If we focus on those most at risk, we should understand behavioral change deeply and ensure that our interventions are more feasible toward empowerment of individuals. High levels of professional support may be required over prolonged time frames.

As we reduce the risks of SICDS further in our efforts, sound public health principles are more important than ever for risk reduction. The successful strategies should promote more creative thinking about how we can engage with, and empower, those at highest risk. We need a limited number of simple achievable interventions. These interventions are composed of combined programs that are appropriately resourced and long term in nature. Strong leadership is recruited and engaged and empowered the target communities in these programs. Finally, we must confirm that these programs are performed under robust monitoring and evaluation.

References

1. Centers for Disease Control and Prevention. About sudden unexpected infant death and sudden infant death syndrome. <https://www.cdc.gov/SICDS/aboutsuidandSICDS.htm>. Accessed 23 Nov 2019.
2. Shapiro-Mendoza CK, Camperlengo L, Ludvigsen R, et al. Classification system for the sudden unexpected infant death case registry and its application. *Pediatrics*. 2014;134(1):e210–9.
3. Heron M. Deaths: leading causes for 2014. *Natl Vital Stat Rep*. 2016;65(5):1–86.
4. Ostfeld BM, Esposito L, Perl H, Hegyi T. Concurrent risks in sudden infant death syndrome. *Pediatrics*. 2010;125(3):447–53.
5. Daltveit AK, Oyen N, Skjaerven R, Irgens LM. The epidemic of SICDS in Norway 1967–93: changing effects of risk factors. *Arch Dis Child*. 1997;77(1):23–7.
6. Babson SG, Clarke NG. Relationship between infant death and maternal age. Comparison of sudden infant death incidence with other causes of infant mortality. *J Pediatr*. 1983;103(3):391–3.
7. l’Hoir MP, Engelberts AC, van Well GT, et al. Case-control study of current validity of previously described risk factors for SICDS in the Netherlands. *Arch Dis Child*. 1998;79(5):386–93.
8. MacDorman MF, Cnattingius S, Hoffman HJ, Kramer MS, Haglund B. Sudden infant death syndrome and smoking in the United States and Sweden. *Am J Epidemiol*. 1997;146(3):249–57.
9. Golding J. Sudden infant death syndrome and parental smoking—a literature review. *Paediatr Perinat Epidemiol*. 1997;11(1):67–77.
10. Taylor JA, Sanderson M. A reexamination of the risk factors for the sudden infant death syndrome. *J Pediatr*. 1995;126(6):887–91.
11. Schoendorf KC, Kiely JL. Relationship of sudden infant death syndrome to maternal smoking during and after pregnancy. *Pediatrics*. 1992;90(6):905–8.
12. Wisborg K, Kesmodel U, Henriksen TB, Olsen SF, Secher NJ. A prospective study of smoking during pregnancy and SICDS. *Arch Dis Child*. 2000;83(3):203–6.
13. O’Leary CM, Jacoby PJ, Bartu A, D’Antoine H, Bower C. Maternal alcohol use and sudden infant death syndrome and infant mortality excluding SICDS. *Pediatrics*. 2013;131(3):e770–8.

14. Iyasu S, Randall LL, Welty TK, et al. Risk factors for sudden infant death syndrome among northern plains Indians. *JAMA*. 2002;288(21):2717–23.
15. Rosen TS, Johnson HL. Drug-addicted mothers, their infants, and SICDS. *Ann NY Acad Sci*. 1988;533:89–95.
16. Klonoff-Cohen HS, Srinivasan IP, Edelstein SL. Prenatal and intrapartum events and sudden infant death syndrome. *Paediatr Perinat Epidemiol*. 2002;16(1):82–9.
17. Li DK, Wi S. Maternal pre-eclampsia/eclampsia and the risk of sudden infant death syndrome in offspring. *Paediatr Perinat Epidemiol*. 2000;14(2):141–4.
18. Smith GC, Wood AM, Pell JP, White IR, Crossley JA, Dobbie R. Second-trimester maternal serum levels of alpha-fetoprotein and the subsequent risk of sudden infant death syndrome. *N Engl J Med*. 2004;351(10):978–86.
19. Malloy MH, Hoffman HJ. Prematurity, sudden infant death syndrome, and age of death. *Pediatrics*. 1995;96(3 Pt 1):464–71.
20. Thompson JM, Mitchell EA. Are the risk factors for SICDS different for preterm and term infants. *Arch Dis Child*. 2006;91(2):107–11.
21. Bigger HR, Silvestri JM, Shott S, Weese-Mayer DE. Influence of increased survival in very low birth weight, low birth weight, and normal birth weight infants on the incidence of sudden infant death syndrome in the United States: 1985-1991. *J Pediatr*. 1998;133(1):73–8.
22. Halloran DR, Alexander GR. Preterm delivery and age of SICDS death. *Ann Epidemiol*. 2006;16(8):600–6.
23. Blair PS, Platt MW, Smith IJ, Fleming PJ. Sudden infant death syndrome and sleeping position in pre-term and low birth weight infants: an opportunity for targeted intervention. *Arch Dis Child*. 2006;91(2):101–6.
24. Malloy MH. Size for gestational age at birth: impact on risk for sudden infant death and other causes of death, USA 2002. *Arch Dis Child Fetal Neonatal Ed*. 2007;92(6):F473–8.
25. Pharoah PO, Platt MJ. Sudden infant death syndrome in twins and singletons. *Twin Res Hum Genet*. 2007;10(4):644–8.
26. Malloy MH, Freeman DH. Sudden infant death syndrome among twins. *Arch Pediatr Adolesc Med*. 1999;153(7):736–40.
27. Beal S. Sudden infant death syndrome in twins. *Pediatrics*. 1989;84(6):1038–44.
28. Platt MJ, Pharoah PO. The epidemiology of sudden infant death syndrome. *Arch Dis Child*. 2003;88(1):27–9.
29. Neubauer J, Lecca MR, Russo G, et al. Post-mortem whole-exome analysis in a large sudden infant death syndrome cohort with a focus on cardiovascular and metabolic genetic diseases. *Eur J Hum Genet*. 2017;25(4):404–9.
30. Beal SM, Blundell HK. Recurrence incidence of sudden infant death syndrome. *Arch Dis Child*. 1988;63(8):924–30.
31. Oyen N, Skjaerven R, Irgens LM. Population-based recurrence risk of sudden infant death syndrome compared with other infant and fetal deaths. *Am J Epidemiol*. 1996;144(3):300–5.
32. Carpenter RG, Waite A, Coombs RC, et al. Repeat sudden unexpected and unexplained infant deaths: natural or unnatural. *Lancet*. 2005;365(9453):29–35.
33. Kassim Z, Donaldson N, Khetriwal B, et al. Sleeping position, oxygen saturation and lung volume in convalescent, prematurely born infants. *Arch Dis Child Fetal Neonatal Ed*. 2007;92(5):F347–50.
34. Elder DE, Campbell AJ, Galletly D. Effect of position on oxygen saturation and requirement in convalescent preterm infants. *Acta Paediatr*. 2011;100(5):661–5.
35. Fyfe KL, Yiallourou SR, Wong FY, Odoi A, Walker AM, Horne RS. Cerebral oxygenation in preterm infants. *Pediatrics*. 2014;134(3):435–45.
36. Moon RY. SICDS and other sleep-related infant deaths: evidence base for 2016 updated recommendations for a safe infant sleeping environment. *Pediatrics*. 2016;138(5):e20162938.
37. Guntheroth WG, Lohmann R, Spiers PS. Risk of sudden infant death syndrome in subsequent siblings. *J Pediatr*. 1990;116(4):520–4.
38. Hoffman HJ, Damus K, Hillman L, Krongrad E. Risk factors for SICDS. Results of the National Institute of Child Health and Human Development SICDS cooperative epidemiological study. *Ann NY Acad Sci*. 1988;533:13–30.

39. Hoppenbrouwers T, Hodgman JE, Ramanathan A, Dorey F. Extreme and conventional cardio-respiratory events and epidemiologic risk factors for SICDS. *J Pediatr.* 2008;152(5):636–41.
40. Mitchell EA, Scragg L, Clements M. Soft cot mattresses and the sudden infant death syndrome. *N Z Med J.* 1996;109(1023):206–7.
41. Erck LAB, Parks SE, Cottengim C, Faulkner M, Hauck FR, Shapiro-Mendoza CK. Sleep-related infant suffocation deaths attributable to soft bedding, overlay, and wedging. *Pediatrics.* 2019;143(5):e20183408.
42. Thach BT, Rutherford GW, Harris K. Deaths and injuries attributed to infant crib bumper pads. *J Pediatr.* 2007;151(3):271–4, 274.e1–3.
43. Scheers NJ, Woodard DW, Thach BT. Crib bumpers continue to cause infant deaths: a need for a new preventive approach. *J Pediatr.* 2016;169:93–7.e1.
44. Moon RY. SICDS and other sleep-related infant deaths: updated 2016 recommendations for a safe infant sleeping environment. *Pediatrics.* 2016;138(5):e20162940.
45. Trachtenberg FL, Haas EA, Kinney HC, Stanley C, Krous HF. Risk factor changes for sudden infant death syndrome after initiation of Back-to-sleep campaign. *Pediatrics.* 2012;129(4):630–8.
46. Blair PS, Fleming PJ, Smith IJ, et al. Babies sleeping with parents: case-control study of factors influencing the risk of the sudden infant death syndrome. CESDI SUDI Research Group. *BMJ.* 1999;319(7223):1457–61.
47. Vennemann MM, Hense HW, Bajanowski T, et al. Bed sharing and the risk of sudden infant death syndrome: can we resolve the debate. *J Pediatr.* 2012;160(1):44–8.e2.
48. Carpenter R, McGarvey C, Mitchell EA, et al. Bed sharing when parents do not smoke: is there a risk of SICDS? An individual level analysis of five major case-control studies. *BMJ Open.* 2013;3(5):e002299.
49. Ponsonby AL, Dwyer T, Cochrane J. Population trends in sudden infant death syndrome. *Semin Perinatol.* 2002;26(4):296–305. <https://doi.org/10.1053/sper.2002.34774>.
50. Rivara FP, Johnston B. Effective primary prevention programs in public health and their applicability to the prevention of child maltreatment. *Child Welfare.* 2013;92(2):119–39.
51. Deal LW, Gomby DS, Zippiroli L, Behrman RE. Unintentional injuries in childhood: analysis and recommendations. *Future Child.* 2000;10(1):4–22. <https://doi.org/10.2307/1602823>.
52. Towner E, Dowswell T. Community-based childhood injury prevention interventions: What works? *Health Promot Int.* 2002;17(3):273–84. <https://doi.org/10.1093/heapro/17.3.273>.
53. Blair PS, Sidebotham P, Berry PJ, Evans M, Fleming PJ. Major epidemiological changes in sudden infant death syndrome: a 20-year population-based study in the UK. *Lancet.* 2006;367(9507):314–9. [https://doi.org/10.1016/S0140-6736\(06\)67968-3](https://doi.org/10.1016/S0140-6736(06)67968-3).
54. Van Wouwe JP, Hirasings RA. Prevention of sudden unexpected infant death. *Lancet.* 2006;367(9507):277–8. [https://doi.org/10.1016/S0140-6736\(06\)67969-5](https://doi.org/10.1016/S0140-6736(06)67969-5).
55. NIH. Safe to Sleep public education campaign. <https://www.nichd.nih.gov/sts/Pages/default.aspx>. Accessed 5 May 2017.
56. Blair P. Sudden infant death syndrome. In: Sidebotham P, Fleming P, editors. *Unexpected death in childhood: a handbook for practitioners*. Chichester: Wiley; 2007. p. 41–60. <https://doi.org/10.1002/9780470988176.ch4>.
57. Moon RY, Hauck FR, Colson ER. Safe infant sleep interventions: what is the evidence for successful behavior change? *Curr Pediatr Rev.* 2016;12(1):67–75. <https://doi.org/10.2174/1573396311666151026110148>.
58. Batra EK, Teti DM, Schaefer EW, Neumann BA, Meek EA, Paul IM. Nocturnal video assessment of infant sleep environments. *Pediatrics.* 2016;138(3):e20161533. <https://doi.org/10.1542/peds.2016-1533>.
59. Task Force On Sudden Infant Death Syndrome. SICDS and other sleep-related infant deaths: updated 2016 recommendations for a safe infant sleeping environment. *Pediatrics.* 2016;138(5).
60. Olds DL, Henderson CR Jr, Tatelbaum R, Chamberlin R. Improving the delivery of prenatal care and outcomes of pregnancy: a randomized trial of nurse home visitation. *Pediatrics.* 1986;77(1):16–28.

61. Mills A, Schmied V, Taylor C, Dahlen H, Schuiringa W, Hudson ME. Connecting, learning, leaving: supporting young parents in the community. *Health Soc Care Community*. 2012;20(6):663–72. <https://doi.org/10.1111/j.1365-2524.2012.01084.x>.
62. Hunt CE. Sudden infant death syndrome and other causes of infant mortality: diagnosis, mechanisms, and risk for recurrence in siblings. *Am J Respir Crit Care Med*. 2001;164(3):346–57. <https://doi.org/10.1164/ajrccm.164.3.9910045>.
63. Campbell MJ, Hall D, Stephenson T, Bacon C, Madan J. Recurrence rates for sudden infant death syndrome (SICDS): the importance of risk stratification. *Arch Dis Child*. 2008;93(11):936–9. <https://doi.org/10.1136/adc.2007.121350>.
64. Brooten D, Youngblut JM, Hannan J, Caicedo C, Roche R, Malkawi F. Infant and child deaths: parent concerns about subsequent pregnancies. *J Am Assoc Nurse Pract*. 2015;27(12):690–7. <https://doi.org/10.1002/2327-6924.12243>.
65. Waite A, McKenzie A, Daman-Willems C. CONI: confirmation of continuing relevance after 20 years. *Community Pract*. 2011;84(1):25–9.
66. Taylor EM, Spencer NJ, Carpenter RG. Evaluation of attempted prevention of unexpected infant death in very high-risk infants by planned health care. *Acta Paediatr*. 1993;82(1):83–6. <https://doi.org/10.1111/j.1651-2227.1993.tb12522.x>.
67. Caraballo M, Shimasaki S, Johnston K, Tung G, Albright K, Halbower AC. Knowledge, attitudes, and risk for sudden unexpected infant death in children of adolescent mothers: a qualitative study. *J Pediatr*. 2016;174:78–83.e2.
68. Carpenter RG, Irgens LM, Blair PS, England PD, Fleming P, Huber J, et al. Sudden unexplained infant death in 20 regions in Europe: case control study. *Lancet*. 2004;363(9404):185–91. [https://doi.org/10.1016/S0140-6736\(03\)15323-8](https://doi.org/10.1016/S0140-6736(03)15323-8).
69. Vennemann MM, Bajanowski T, Brinkmann B, Jorch G, Sauerland C, Mitchell EA, et al. Sleep environment risk factors for sudden infant death syndrome: the German sudden infant death syndrome study. *Pediatrics*. 2009;123(4):1162–70. <https://doi.org/10.1542/peds.2008-0505>.
70. L'Hoir MP, Engelberts AC, van Well GT, McClelland S, Westers P, Dandachli T, et al. Risk and preventive factors for cot death in the Netherlands, a low-incidence country. *Eur J Pediatr*. 1998;157(8):681–8. <https://doi.org/10.1007/s004310050911>.
71. Blair PS, Sidebotham P, Evason-Coombe C, Edmonds M, Heckstall-Smith EM, Fleming P. Hazardous cosleeping environments and risk factors amenable to change: case-control study of SICDS in south West England. *BMJ*. 2009;339:b3666. <https://doi.org/10.1136/bmj.b3666>.
72. Moon RY, Task Force On Sudden Infant Deaths. SICDS and other sleep-related infant deaths: evidence base for 2016 updated recommendations for a safe infant sleeping environment. *Pediatrics*. 2016;138(5):e20162940. <https://doi.org/10.1542/peds.2016-2940>.
73. Statistics Finland. Causes of death: Appendix table 3. Mortality during infant and perinatal period 1987–2015. http://www.stat.fi/til/ksyyt/2015/ksyyt_2015_2016-12-30_tau_005_en.html. Accessed 11 Oct 2017.
74. Ross L. Baby boxes: improving wellbeing outcomes in Scotland. *J Health Visiting*. 2017;5(4):172–5. <https://doi.org/10.12968/johv.2017.5.4.172>.
75. Murphy M. UK's first Baby Box programme launched at London NHS Trust. *Br J Midwifery*. 2016;24(7):521–2. <https://doi.org/10.12968/bjom.2016.24.7.521>.
76. Demer L. Rash of sleep-related infant deaths troubles health officials. *Alaska Dispatch News* [internet]. 2015. <https://www.adn.com/health/article/rash-infant-deaths-related-sleeping-alarms-health-officials/2015/02/15/>. Accessed 1 Jun 2017.
77. Ward Platt M, Blair PS, Fleming PJ, Smith IJ, Cole TJ, Leach CE, et al. A clinical comparison of SICDS and explained sudden infant deaths: How healthy and how normal? CESDI SUDI Research Group. Confidential Inquiry into Stillbirths and Deaths in Infancy study. *Arch Dis Child*. 2000;82(2):98–106. <https://doi.org/10.1136/adc.82.2.98>.
78. Ford RP, Mitchell EA, Stewart AW, Scragg R, Taylor BJ. SICDS, illness, and acute medical care. New Zealand cot death study group. *Arch Dis Child*. 1997;77(1):54–5. <https://doi.org/10.1136/adc.77.1.54>.
79. Wilkinson A. Pre-hospital assessment of a child under one year old with fever. *Emerg Nurse*. 2017;24(10):28–33. <https://doi.org/10.7748/en.2017.e1663>.

80. Mitchell EA, Milerad J. Smoking and the sudden infant death syndrome. *Rev Environ Health*. 2006;21(2):81–103. <https://doi.org/10.1515/REVEH.2006.21.2.81>.
81. Lumley J, Chamberlain C, Dowswell T, Oliver S, Oakley L, Watson L. Interventions for promoting smoking cessation during pregnancy. *Cochrane Database Syst Rev*. 2009;8(3):CD001055. <https://doi.org/10.1002/14651858.CD001055.pub3>.
82. Fleming P, Blair PS. Sudden infant death syndrome and parental smoking. *Early Hum Dev*. 2007;83(11):721–5. <https://doi.org/10.1016/j.earlhumdev.2007.07.011>.
83. Lavender T, Richens Y, Milan SJ, Smyth RM, Dowswell T. Telephone support for women during pregnancy and the first six weeks postpartum. *Cochrane Database Syst Rev*. 2013;7:CD009338. <https://doi.org/10.1002/14651858.CD009338.pub2>.
84. Stead LF, Hartmann-Boyce J, Perera R, Lancaster T. Telephone counselling for smoking cessation. *Cochrane Database Syst Rev*. 2013;8:CD002850. <https://doi.org/10.1002/14651858.CD002850.pub3>.
85. Reading AE, Campbell S, Cox DN, Sledmere CM. Health beliefs and health care behavior in pregnancy. *Psychol Med*. 1982;12(2):379–83. <https://doi.org/10.1017/S0033291700046717>.
86. Nabhan AF, Faris MA. High feedback versus low feedback of prenatal ultrasound for reducing maternal anxiety and improving maternal health behavior in pregnancy. *Cochrane Database Syst Rev*. 2010;4:CD007208.
87. Dempsey D, Jacob P 3rd, Benowitz NL. Accelerated metabolism of nicotine and cotinine in pregnant smokers. *J Pharmacol Exp Ther*. 2002;301(2):594–8. <https://doi.org/10.1124/jpet.301.2.594>.
88. Benowitz N, Dempsey D. Pharmacotherapy for smoking cessation during pregnancy. *Nicotine Tob Res*. 2004;6(Suppl 2):S189–202. <https://doi.org/10.1080/14622200410001669169>.
89. Coleman T, Chamberlain C, Cooper S, Leonardi-Bee J. Efficacy and safety of nicotine replacement therapy for smoking cessation in pregnancy: systematic review and meta-analysis. *Addiction*. 2011;106(1):52–61. <https://doi.org/10.1111/j.1360-0443.2010.03179.x>.
90. Myung SK, Ju W, Jung HS, Park CH, Oh SW, Seo H, et al. Efficacy and safety of pharmacotherapy for smoking cessation among pregnant smokers: a meta-analysis. *BJOG*. 2012;119(9):1029–39. <https://doi.org/10.1111/j.1471-0528.2012.03408.x>.
91. Etter JF. Electronic cigarettes: a survey of users. *BMC Public Health*. 2010;10:231. <https://doi.org/10.1186/1471-2458-10-231>.
92. Breland A, Soule E, Lopez A, Ramoa C, El-Hellani A, Eissenberg T. Electronic cigarettes: what are they and what do they do? *Ann N Y Acad Sci*. 2017;1394(1):5–30. <https://doi.org/10.1111/nyas.12977>.
93. England LJ, Anderson BL, Tong VT, Mahoney J, Coleman-Cowger VH, Melstrom P, et al. Screening practices and attitudes of obstetricians-gynecologists toward new and emerging tobacco products. *Am J Obstet Gynecol*. 2014;211(6):695 e1–7.