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Risk Factors for Sudden Infant Death Syndrome: An Updated Narrative Review

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Abstract

Background: Sudden Infant Death Syndrome (SIDS) is defined as the sudden and unexplained death of an infant younger than one year of age that remains unexplained after a comprehensive investigation, including autopsy, examination of the death scene, and review of clinical and family history. Despite a substantial reduction in incidence following the implementation of safe sleep recommendations, SIDS remains an important public health concern. Multiple maternal, infant-related, environmental, and socioeconomic factors contribute to SIDS risk.

Objective: The aim of this narrative review was to summarize current evidence regarding modifiable and non-modifiable risk factors associated with Sudden Infant Death Syndrome and to discuss evidence-based preventive strategies.

Methods: A narrative review of literature from PubMed/MEDLINE, Scopus, Web of Science, and Embase was conducted, including reviews, meta-analyses, observational studies, and clinical guidelines.

Results: Current evidence indicates that SIDS is a multifactorial condition resulting from interactions between intrinsic infant vulnerability, developmental factors, and external environmental stressors. The strongest modifiable risk factors include prenatal and postnatal tobacco exposure, prone sleeping position, unsafe sleep environments, overheating, and bed-sharing in high-risk circumstances. Prematurity, low birth weight, male sex, and genetic susceptibility contribute to biological vulnerability. Breastfeeding and pacifier use during sleep are associated with reduced SIDS risk.

Conclusions: SIDS prevention requires a multifactorial approach focused on reducing modifiable environmental risks, promoting safe sleep practices, eliminating tobacco exposure, and improving parental education. Further research is needed to better understand genetic and biological mechanisms contributing to individual susceptibility.

Keywords: Sudden Infant Death Syndrome; SIDS; sudden unexpected infant death; risk factors; safe sleep; prone sleeping; smoking; breastfeeding; prematurity; Triple Risk Model; narrative review.

Introduction

Sudden infant death syndrome (SIDS) is defined as the sudden and unexpected death of an infant younger than one year of age that remains unexplained after a complete investigation, including autopsy, examination of the death scene, and review of the clinical and family history. The term **SIDS** was first introduced in **1969** to describe a distinct subgroup of unexpected infant deaths occurring during the post-neonatal period and characterized by relatively consistent clinical, epidemiological, and pathological features. [3,4]

According to the most recent data published by the Centers for Disease Control and Prevention (CDC), approximately 3,700 sudden unexpected infant deaths (SUID) occurred in the United States in 2022. Among these cases, 41.3% (1,529 deaths) were classified as SIDS, 30.6% (1,131 deaths) were attributed to unknown causes, and 28.1% (1,040 deaths) were classified as accidental suffocation and strangulation in bed (ASSB). These data indicate that although SIDS remains the largest single category within SUID, the majority of sudden unexpected infant deaths are either unexplained or associated with sleep-related accidental asphyxia, emphasizing the importance of standardized death investigations and adherence to evidence-based safe sleep recommendations. [5]

In Europe, SIDS remains an important contributor to post-neonatal infant mortality despite a substantial reduction in incidence following the introduction of safe sleep campaigns. According to Eurostat, overall infant mortality in the European Union remains low, with approximately 3.3–3.5 deaths per 1,000 live births in recent years, reflecting improvements in prenatal care, neonatal medicine, and preventive strategies. However, SIDS continues to represent a significant public health challenge because many cases remain unexplained despite comprehensive postmortem investigations and differences in diagnostic classification between European countries. [6]

The aim of this narrative review is to summarize current evidence regarding modifiable and non-modifiable risk factors associated with Sudden Infant Death Syndrome and to discuss current prevention strategies.

Etiology and Pathophysiology of SIDS

Despite extensive research, the exact mechanisms underlying Sudden Infant Death Syndrome remain incompletely understood. Current evidence suggests that SIDS is a multifactorial disorder resulting from the interaction between intrinsic infant vulnerability, a critical developmental period, and external environmental stressors.[7] The most widely accepted explanation is the Triple Risk Model, originally proposed by Filiano and Kinney, which describes SIDS as the consequence of the simultaneous presence of three components: (1) an intrinsically vulnerable infant, (2) a critical period of development, and (3) an external stressor that may trigger a fatal event.[8]

The first component involves intrinsic biological susceptibility, including abnormalities in autonomic nervous system regulation, impaired cardiorespiratory control, altered arousal mechanisms, and genetic factors affecting respiratory and metabolic pathways.[9] Neuropathological investigations have identified abnormalities in the brainstem, particularly within serotonergic pathways, which may contribute to impaired responses to hypoxia, hypercapnia, and other physiological challenges during sleep.[10,11]

The second component refers to a vulnerable developmental period, with the highest incidence of SIDS occurring between 2 and 4 months of age, a stage characterized by rapid maturation of respiratory control, sleep regulation, and autonomic nervous system function.[9] The third component includes external environmental stressors that may increase the likelihood of a fatal event, including prone sleeping position, prenatal and postnatal tobacco exposure, overheating, soft bedding, and unsafe sleep environments.[12]

Although the Triple Risk Model does not fully explain all cases of SIDS, it provides an important framework for understanding how biological vulnerability interacts with modifiable environmental exposures. Therefore, identification and modification of preventable risk factors remain essential components of current SIDS prevention strategies and represent the most effective approach to reducing SIDS incidence.[8]

Risk Factors Associated with Sudden Infant Death Syndrome

Sudden Infant Death Syndrome is recognized as a multifactorial condition in which genetic susceptibility, developmental vulnerability, and environmental exposures interact to increase the risk of a fatal event. Although the underlying mechanisms remain incompletely understood, numerous epidemiological studies have identified several modifiable and non-modifiable factors associated with increased SIDS risk. These factors can be broadly categorized into maternal and prenatal factors, infant-related characteristics, and environmental and sleep-related factors[13].

Maternal and Prenatal Factors

Maternal and prenatal exposures play an important role in determining infant vulnerability to SIDS. Maternal smoking during pregnancy is one of the most consistently identified and strongest modifiable risk factors. Prenatal exposure to tobacco smoke has been associated with impaired fetal development, altered autonomic regulation, reduced arousal responses, and increased susceptibility to hypoxic events after birth. Postnatal exposure to environmental tobacco smoke further increases the risk, demonstrating a dose-dependent association between nicotine exposure and SIDS. [14]

Other maternal substance exposures, including alcohol and illicit drug use during pregnancy, have also been associated with an increased risk of SIDS. These exposures may contribute through mechanisms involving impaired fetal growth, premature birth, neurological development abnormalities, and altered respiratory control.[15]

Inadequate prenatal care and limited access to maternal healthcare have been associated with increased SIDS risk, potentially due to reduced identification and management of pregnancy complications. Maternal demographic characteristics, including young maternal age, have also been associated with increased risk, although this relationship is partly influenced by socioeconomic conditions and health behaviors.[16]

Socioeconomic disadvantage has consistently been associated with an increased risk of SIDS. Low household income, limited parental education, and social deprivation may reduce access to prenatal and postnatal healthcare, limit adherence to safe sleep recommendations, and increase exposure to environmental risk factors. These factors are therefore considered indirect determinants of SIDS, acting through multiple interacting biological, behavioral, and environmental pathways rather than as independent causes.[2,17]

Infant-Related Factors

Several infant characteristics are associated with increased susceptibility to SIDS. Prematurity and low birth weight are among the most consistently reported non-modifiable risk factors. Preterm infants may have immature respiratory control systems, impaired autonomic regulation, and reduced arousal responses, which may increase vulnerability during sleep.[9]

Male sex is consistently associated with a higher incidence of SIDS, with males accounting for approximately 60% of cases in many populations. Although the biological basis of this sex difference remains uncertain, several hypotheses have been proposed, including an X-linked genetic mechanism that may contribute to increased male susceptibility during early infancy. [18]

Increasing evidence suggests that genetic susceptibility may contribute to SIDS risk. Genetic variants affecting cardiac conduction, metabolic pathways, respiratory regulation, and autonomic nervous system function have been identified in a subset of SIDS cases. Nevertheless, genetic factors are currently considered contributors to biological vulnerability rather than independent causes of SIDS, and their interaction with developmental and environmental factors is likely critical in the pathogenesis of the syndrome.[19]

A history of previous apparent life-threatening events (ALTE) or brief resolved unexplained events (BRUE) has also been investigated as a potential risk factor. Although most infants experiencing BRUE do not subsequently die from SIDS, recurrent or severe episodes may indicate underlying abnormalities requiring clinical evaluation.[20]

Environmental and Sleep-Related Factors

Environmental and sleep-related factors represent the largest group of modifiable risk factors for SIDS. Numerous epidemiological studies have identified several characteristics of the infant sleep environment that are associated with an increased risk of SIDS. Extrinsic factors associated with increased SIDS risk include prone and side sleep position, soft sleep surface and soft bedding over or under the infant, head or face covered during sleep, excess thermal insulation, bed sharing, and postnatal smoke exposure.[21]

The prone sleeping position is one of the strongest established risk factors. Infants placed to sleep in the prone position have a significantly higher risk of SIDS than those placed in the supine position.[2,12]

The use of soft bedding, pillows, blankets, and other loose materials in the sleep environment increases the risk of airway obstruction, overheating, and accidental suffocation. Similarly, excessive thermal insulation and overheating have been associated with increased risk, highlighting the importance of maintaining an appropriate infant sleep environment and avoiding excessive clothing or room temperature.[12]

Protective factors include breastfeeding and pacifier use during sleep. Breastfeeding has consistently been associated with a reduced risk of SIDS, possibly through its beneficial effects on immune function, autonomic regulation, sleep architecture, and infant arousal mechanisms. Pacifier use during sleep has also been associated with a lower risk of SIDS, although the underlying biological mechanisms remain incompletely understood. [2,22]

Transition to the Aim of the Review

Despite substantial progress in identifying SIDS risk factors, the syndrome remains incompletely understood due to its multifactorial nature and complex interaction between biological vulnerability and environmental exposures. Therefore, an updated narrative review

of current evidence is essential to evaluate established and emerging risk factors and to identify effective strategies for further reducing SIDS incidence.

Methods

A narrative literature review was performed to summarize current evidence regarding risk factors associated with Sudden Infant Death Syndrome (SIDS). Relevant publications were identified through searches of electronic databases including PubMed/MEDLINE, Scopus, Web of Science, and Embase. The search focused on studies addressing maternal, prenatal, infant-related, environmental, and protective factors associated with SIDS. Selected systematic reviews, meta-analyses, cohort studies, case-control studies, and clinical recommendations published in English were considered.

Evidence Summary

Maternal and Prenatal Risk Factors

Maternal and prenatal factors represent an important group of determinants associated with increased susceptibility to Sudden Infant Death Syndrome (SIDS). Among these factors, maternal smoking during pregnancy is one of the most consistently identified and strongest modifiable risk factors. Numerous epidemiological studies have demonstrated a significant association between prenatal tobacco exposure and increased risk of SIDS. The relationship appears to be dose-dependent, with higher levels of exposure associated with greater risk. Proposed mechanisms include impaired fetal growth, altered autonomic nervous system development, impaired cardiorespiratory regulation, and reduced infant arousal responses to hypoxic or hypercapnic challenges. [14]

Postnatal exposure to environmental tobacco smoke has also been associated with increased SIDS risk. Exposure to nicotine and other tobacco-related toxins after birth may further impair respiratory regulation and increase vulnerability during sleep. Therefore, complete avoidance of tobacco exposure during pregnancy and infancy remains one of the most important preventive strategies. [2,14,21]

Maternal alcohol consumption and exposure to illicit substances during pregnancy have also been associated with increased risk of SIDS. These exposures may influence fetal neurological development, increase the risk of prematurity and low birth weight, and affect respiratory control mechanisms during early infancy. [15]

Socioeconomic disadvantage has been repeatedly associated with increased SIDS incidence. Low maternal education, reduced household income, inadequate prenatal care, and limited access to healthcare may contribute through multiple pathways, including reduced implementation of safe sleep recommendations, increased exposure to environmental risk

factors, and differences in healthcare utilization. Socioeconomic factors should therefore be considered indirect determinants that interact with biological and behavioral factors rather than independent causes of SIDS. [16,17]

Infant-Related Risk Factors

Several infant characteristics contribute to increased vulnerability to SIDS. Prematurity and low birth weight are among the most consistently reported non-modifiable risk factors. Preterm infants may have immature respiratory control systems, impaired autonomic regulation, and reduced ability to respond adequately to hypoxia and hypercapnia during sleep. These physiological limitations may increase susceptibility when additional environmental stressors occur. [9,13]

Male sex is another consistently observed risk factor. In many populations, male infants represent approximately 60% of SIDS cases. Although the biological explanation remains uncertain, proposed mechanisms include differences in neurological maturation, hormonal regulation, and genetic susceptibility affecting early infant adaptation. [18]

Increasing evidence indicates that genetic susceptibility may contribute to SIDS pathogenesis. Genetic variants affecting cardiac conduction pathways, metabolic processes, serotonin signaling, and autonomic regulation have been identified in a subset of infants who died suddenly and unexpectedly. However, genetic factors are currently considered contributors to biological vulnerability rather than independent causes of SIDS. Their effects are likely mediated through interactions with developmental and environmental factors. [19]

Previous episodes classified as apparent life-threatening events (ALTE) or brief resolved unexplained events (BRUE) have also been investigated as potential markers of increased risk. Although most infants with BRUE do not subsequently die from SIDS, recurrent or high-risk events may indicate underlying abnormalities requiring further evaluation. [20]

Sleep Environment and Modifiable Risk Factors

Environmental and sleep-related factors represent the largest group of modifiable risk factors associated with SIDS. Among these, prone sleeping position is one of the strongest and most consistently demonstrated risk factors. Infants placed to sleep in the prone position have a significantly higher risk of SIDS compared with infants placed in the supine position. The implementation of national safe sleep campaigns recommending supine sleeping has been associated with substantial reductions in SIDS rates. [2,12,21]

Side sleeping has also been associated with increased risk because infants placed on their side may unintentionally move into the prone position during sleep. Current recommendations therefore emphasize that infants should be placed supine for every sleep period, including nighttime sleep and naps. [7,12]

The characteristics of the sleep environment strongly influence SIDS risk. The use of soft bedding, pillows, blankets, and other loose materials may increase the risk of airway obstruction, rebreathing of exhaled carbon dioxide, and impaired thermal regulation. Excessive thermal insulation and overheating have also been associated with increased risk, supporting recommendations to avoid excessive clothing and maintain an appropriate sleep environment. [2,12,21]

Bed-sharing remains a complex risk factor. Room-sharing without bed-sharing is recommended as a protective strategy, whereas sharing a sleep surface may increase SIDS risk, particularly among infants exposed to additional hazards such as parental smoking, alcohol use, prematurity, or very young age. [2,12]

Protective Factors

Several factors have been associated with reduced SIDS risk. Breastfeeding is one of the most consistently reported protective factors. A meta-analysis demonstrated that breastfeeding is associated with a significant reduction in SIDS risk. Possible mechanisms include improved immune protection, reduced infection risk, enhanced autonomic regulation, and improved infant arousal responses. [22]

Pacifier use during sleep has also been associated with reduced SIDS risk. Although the exact mechanism remains uncertain, possible explanations include improved airway patency, modification of sleep architecture, and increased frequency of partial arousals during sleep. Current recommendations consider pacifier use as a potential additional protective measure when introduced appropriately. [2,7]

Interaction Between Biological Vulnerability and Environmental Exposures

Current evidence supports the concept that SIDS is a multifactorial disorder caused by the interaction between intrinsic infant vulnerability, a critical developmental period, and external environmental stressors. The Triple Risk Model, proposed by Filiano and Kinney, remains the most widely accepted framework explaining SIDS pathogenesis. According to this model, death occurs when a vulnerable infant experiences an external stressor during a period of immature physiological development. [8]

Intrinsic vulnerability may include abnormalities in brainstem serotonergic pathways, impaired autonomic regulation, and altered cardiorespiratory control mechanisms. These abnormalities may reduce the infant's ability to respond appropriately to environmental challenges such as hypoxia, airway obstruction, or overheating. [10,11]

Therefore, SIDS prevention strategies should focus primarily on reducing modifiable environmental exposures, especially unsafe sleep practices and tobacco exposure, while recognizing that biological susceptibility contributes to individual differences in risk. [7,12]

Table 1. Summary of major risk factors associated with Sudden Infant Death Syndrome (SIDS)

Risk factor	Category	Effect on SIDS risk	Proposed mechanism	Level of evidence	Key references
Prone sleeping position	Modifiable	↑↑ Strong increase	Airway obstruction, rebreathing of CO_2, impaired arousal	High (systematic reviews, guidelines)	Moon et al. 2022 [7,12]; Moon et al. 2011 [2,21]
Side sleeping position	Modifiable	↑ Moderate increase	Increased probability of rolling into prone position	High	Moon et al. 2022 [12]
Maternal smoking during pregnancy	Modifiable	↑↑ Strong increase	Impaired autonomic regulation, altered fetal brain development	High	Anderson et al. [14]; Kim et al. [13]
Postnatal tobacco smoke exposure	Modifiable	↑ Increase	Impaired respiratory control and arousal	High	Anderson et al. [14]; Moon et al. [2]
Bed-sharing (high-risk settings)	Modifiable	↑ Increase	Airway obstruction, accidental suffocation	High	Moon et al. 2022 [12]; Moon et al. 2011 [21]
Soft bedding/soft sleep surface	Modifiable	↑ Increase	Airway obstruction and CO_2 rebreathing	High	Moon et al. [12]

Overheating/excessive bedding	Modifiable	↑ Increase	Impaired thermoregulation	Moderate–High	Moon et al. [2,12]
Prematurity	Non-modifiable	↑ Increase	Immature autonomic and respiratory control	High	Kinney & Thach [9]; Kim et al. [13]
Low birth weight	Non-modifiable	↑ Increase	Reduced physiological reserve	High	Kinney & Thach [9]
Male sex	Non-modifiable	↑ Moderate increase	Possible genetic and hormonal mechanisms	Moderate	Mage & Donner [18]
Genetic susceptibility	Non-modifiable	↑ Potential increase	Cardiac channelopathies, serotonin pathway abnormalities	Moderate	Goldstein et al. [19]
Alcohol/drug exposure during pregnancy	Modifiable	↑ Increase	Abnormal neurological development	Moderate	O'Leary et al. [15]
Low socioeconomic status	Indirect	↑ Increase	Reduced healthcare access and adherence to safe sleep recommendations	Moderate	Demissie et al. [16]; Hauck & Tanabe [17]
Breastfeeding	Protective	↓ Reduced risk	Improved immunity, enhanced arousal, fewer infections	High (meta-analysis)	Hauck et al. [23]
Pacifier use during sleep	Protective	↓ Reduced risk	Improved airway patency and sleep regulation	Moderate–High	Moon et al. [2,7]

Abbreviations: ↑ increased risk; ↓ reduced risk; CO_2 – carbon dioxide.

Discussion

Sudden Infant Death Syndrome (SIDS) remains a complex and multifactorial condition despite substantial progress in understanding its epidemiology, pathophysiology, and prevention. The evidence summarized in this narrative review supports the concept that SIDS does not result from a single causative factor but rather from the interaction between intrinsic infant

vulnerability, a critical developmental period, and external environmental stressors. This concept is consistent with the Triple Risk Model proposed by Filiano and Kinney, which remains the most widely accepted framework for explaining the mechanisms underlying SIDS. According to this model, death occurs when a vulnerable infant encounters an external stressor during a period of immature physiological development. [8]

One of the most important findings from current evidence is the continuing influence of maternal and environmental tobacco exposure as a major modifiable risk factor for SIDS. Prenatal smoking has been consistently associated with increased SIDS risk, with biological mechanisms including impaired fetal growth, altered development of autonomic regulation, reduced arousal responses, and impaired respiratory control. Although public health interventions have reduced smoking prevalence in many populations, tobacco exposure remains a significant contributor to preventable infant deaths. Therefore, smoking cessation programs targeted at women before and during pregnancy, as well as elimination of infant exposure to second-hand smoke, remain essential components of SIDS prevention strategies. [14]

The findings also emphasize the importance of safe sleep practices as the most effective preventive approach. The introduction of recommendations promoting supine sleeping has resulted in substantial reductions in SIDS incidence worldwide. However, unsafe sleep practices, including prone positioning, exposure to soft bedding, overheating, and inappropriate sleep environments, continue to occur. Current recommendations emphasize that infants should be placed on their backs for every sleep period, placed on a firm and separate sleep surface, and protected from loose bedding and excessive thermal insulation. [2,7,12]

The role of bed-sharing remains a particularly complex issue. While room-sharing without bed-sharing has been associated with reduced risk and is recommended by current guidelines, sharing a sleep surface may increase SIDS risk under specific circumstances. The risk appears to be particularly elevated when bed-sharing occurs together with additional hazards, including parental smoking, alcohol consumption, prematurity, low birth weight, or very young infant age. These findings highlight the importance of individualized parental counselling rather than focusing solely on isolated behaviors. [2,12,21]

Non-modifiable infant characteristics, including prematurity, low birth weight, male sex, and genetic susceptibility, also contribute to SIDS vulnerability. Preterm infants may have immature respiratory control systems, impaired autonomic regulation, and reduced arousal capacity, which may limit their ability to respond effectively to environmental challenges during sleep. Although genetic abnormalities affecting cardiac conduction, metabolism, serotonin pathways, and autonomic regulation have been identified in some cases, current evidence indicates that genetic factors represent contributors to susceptibility rather than direct causes of SIDS. Further research is needed to clarify how genetic predisposition interacts with environmental exposures. [9,13,19]

The evidence regarding protective factors supports the role of breastfeeding and pacifier use as components of SIDS risk reduction strategies. Breastfeeding has been associated with lower

SIDS risk, potentially through immunological benefits, reduced infection frequency, and improved autonomic regulation. Similarly, pacifier use during sleep has demonstrated a protective association, although the biological mechanisms remain incompletely understood. Importantly, these protective factors should be considered complementary strategies and should not replace established safe sleep recommendations. [7,22]

Socioeconomic determinants represent another important aspect of SIDS prevention. Higher SIDS rates among socioeconomically disadvantaged populations likely reflect the combined effects of limited healthcare access, differences in prenatal care, housing conditions, parental education, and reduced exposure to preventive health messages. Consequently, effective prevention requires not only individual-level education but also broader public health interventions addressing social inequalities. [16,17]

Despite significant advances in prevention, several challenges remain. First, SIDS diagnosis continues to depend on detailed death investigations, including autopsy and evaluation of the death scene, and differences in classification practices between countries may influence reported incidence rates. [1,3,4] Second, the absence of reliable biomarkers makes early identification of infants with increased biological susceptibility difficult. Future research should focus on integrating genetic, neurological, metabolic, and epidemiological data to better characterize infants at highest risk.

This review has several limitations. As a narrative review, it does not follow a systematic search strategy with predefined inclusion criteria, and therefore publication bias and selection bias cannot be excluded. Additionally, differences in study designs, diagnostic criteria, and population characteristics limit direct comparison between studies. Nevertheless, this approach allows integration of evidence from epidemiological studies, biological investigations, and clinical recommendations to provide a comprehensive overview of current knowledge regarding SIDS risk factors.

Conclusions

Sudden Infant Death Syndrome remains a multifactorial disorder resulting from complex interactions between infant vulnerability, developmental processes, and environmental exposures. The strongest evidence supports the importance of reducing modifiable risk factors, particularly prenatal and postnatal tobacco exposure, unsafe sleep practices, prone sleeping position, and overheating.

Implementation of evidence-based safe sleep recommendations, promotion of breastfeeding, appropriate parental education, and reduction of socioeconomic barriers remain essential strategies for further decreasing SIDS incidence. Future studies should integrate genetic, neurological, metabolic, and epidemiological approaches to better identify infants with increased biological susceptibility.

Disclosure

Author's contribution

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