



Evidence Base for 2022 Updated Recommendations for a Safe Infant Sleeping Environment to Reduce the Risk of Sleep-Related Infant Deaths

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Every year in the United States, approximately 3500 infants die of sleep-related infant deaths, including sudden infant death syndrome (SIDS) (*International Statistical Classification of Diseases and Related Health Problems 10th Revision* [ICD-10] R95), ill-defined deaths (ICD-10 R99), and accidental suffocation and strangulation in bed (ICD-10 W75). After a substantial decline in sleep-related deaths in the 1990s, the overall death rate attributable to sleep-related infant deaths have remained stagnant since 2000, and disparities persist. The triple risk model proposes that SIDS occurs when an infant with intrinsic vulnerability (often manifested by impaired arousal, cardiorespiratory, and/or autonomic responses) undergoes an exogenous trigger event (eg, exposure to an unsafe sleeping environment) during a critical developmental period. The American Academy of Pediatrics recommends a safe sleep environment to reduce the risk of all sleep-related deaths. This includes supine positioning; use of a firm, noninclined sleep surface; room sharing without bed sharing; and avoidance of soft bedding and overheating. Additional recommendations for SIDS risk reduction include human milk feeding; avoidance of exposure to nicotine, alcohol, marijuana, opioids, and illicit drugs; routine immunization; and use of a pacifier. New recommendations are presented regarding noninclined sleep surfaces, short-term emergency sleep locations, use of cardboard boxes as a sleep location, bed sharing, substance use, home cardiorespiratory monitors, and tummy time. In addition, additional information to assist parents, physicians, and nonphysician clinicians in assessing the risk of specific bed-sharing situations is included. The recommendations and strength of evidence for each recommendation are published in the accompanying policy statement, which is included in this issue.

abstract

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SEARCH STRATEGY AND METHODOLOGY

Literature searches using PubMed were conducted for each of the topics in the technical report, concentrating on papers published since 2015 (to avoid omitting papers that were published between the time when the last technical report¹ and policy² statement were submitted for review and published). All iterations of the search terms were used for each topic area. For example, the pacifier topic search combined either “SIDS,” “SUID,” “sudden death,” “cot death,” “suffocation,” “asphyxia,” “overlay,” “obstruction,” or “airway” with “pacifier,” “dummy,” “soother,” and “sucking.” A total of 159 new studies were judged to be of sufficiently high quality to be included in this technical report. Strength of evidence for recommendations, using the Strength-of-Recommendation Taxonomy (SORT),³ was determined by the task force members. Draft versions of the policy statement and technical report were submitted to relevant committees and sections of the American Academy of Pediatrics (AAP) for review and comment. After the appropriate revisions were made, a final version was submitted to the AAP Executive Committee for final approval.

SUDDEN INFANT DEATH: DEFINITIONS AND DIAGNOSTIC ISSUES

Sudden unexpected infant death (SUID) is a term used to describe any sudden and unexpected death, whether explained or unexplained, occurring during infancy. After case investigation, it may be determined that an unexpected death was caused by a specific unnatural or natural etiology, such as suffocation, mechanical asphyxia, entrapment, infection, ingestions, metabolic diseases, or trauma (unintentional or nonaccidental). Unexpected deaths that cannot be explained are referred to as either

sudden unexplained infant death, sudden infant death syndrome (SIDS), or deaths of undetermined cause. In actual usage, the acronyms and “U” terms (variably unexpected, unexplained, undetermined, unascertained) are frequently confused, and this has undermined consistent communication and surveillance.⁴ Two large, multidisciplinary teams of experts have recently recommended adoption of the term unexplained sudden death in infancy or SIDS for deaths of infants younger than 1 year of age that remain unexplained following investigation, autopsy, medical history review, and appropriate laboratory testing.^{5,6} This terminology takes into consideration difficulties created by acronyms, adheres to current criteria for SIDS, and is inclusive of deaths with combinations of extrinsic factors and/or intrinsic vulnerabilities or abnormalities that do not reach a diagnostic threshold for a specific cause of death. Unexplained sudden death in infancy, and not SIDS, is the terminology preferred by the National Association of Medical Examiners.^{4,5} Because nearly all of the deaths discussed here occur during infant sleep or in a sleep environment, this technical report uses the term sleep-related death (infants implied) to encompass unexplained sudden death in infancy or SIDS and accidental deaths explained by a physical hazard in the sleep environment, except where reference is made to published data that used a specific terminology and definition (Table 1).

National tools for conducting thorough case investigations for sleep-related deaths in infants have been developed.^{5,7,8} Case investigations are not uniform across the more than 2000 US

medical examiner and coroner jurisdictions for a multitude of reasons, ranging from inadequate resources to varied policies and diverse background and training of investigators.^{9,10} In 2014, about two-thirds of medical examiners and coroners used the Centers for Disease Control and Prevention (CDC)’s reporting form or an equivalent (>85% use in medium and large district offices, but only 54% in small district offices).¹¹ In addition, there are varied opinions and preferences regarding diagnostic criteria for cause of death and wording of certification statements. Recently, much attention has focused on reporting differences among death certifiers¹² and the impact on health statistics—that is, the so-called “diagnostic shift” in SIDS data.^{9,13} At 1 extreme, some certifiers have abandoned using SIDS as a cause of death.^{5,6,9,14} On the other extreme, some certifiers will continue to use SIDS even when there is strong evidence from the scene investigation of an unintentional suffocation. Difficulties in differentiating deaths truly caused by mechanical asphyxia from unexplained sleep-related death in an unsafe environment (ie, unexplained sudden death with the possibility of mechanical asphyxia) have resulted in imprecise classification. There is hope that recently developed criteria for certification of infant deaths as being caused by asphyxia will have a positive impact.⁶

United States Trends in Sleep-Related Deaths and Postneonatal Mortality

To monitor trends in causes of death, the United States classifies diseases and injuries according to the *International Statistical Classification of Diseases and Related Health Problems 10th Revision* (ICD-

TABLE 1 Definitions of Terms

Term	Definition
ASSB, accidental strangulation or suffocation in bed	An explained sudden and unexpected infant death in a sleep environment (bed, crib, couch, chair, etc) in which the infant's nose and mouth are obstructed, or the neck or chest is compressed from soft or loose bedding, an overlay, or wedging causing asphyxia. Corresponds to ICD-10 W75.
Bed sharing	Parent(s) and infant sleeping together on any surface (bed, couch, chair). Medical examiners prefer the term "surface sharing."
Caregivers	Throughout the document, "parents" are used, but this term is meant to indicate any infant caregivers.
Cosleeping	This term is commonly used in other publications, is not recommended because it lacks clarity, being variably used for sleeping in close proximity (eg, room sharing) and/or sleep surface or bed sharing.
Room sharing	Parent(s) and infant sleeping in the same room on separate surfaces.
SIDS (sudden infant death syndrome)	Cause assigned to infant deaths that cannot be explained after a thorough case investigation, including a death scene investigation, autopsy, and review of the clinical history.
Sleep-related infant death	A sudden unexpected infant death that occurs during an observed or unobserved sleep period, or in a sleep environment.
Sudden unexpected infant death (SUID)	A sudden and unexpected death, whether explained or unexplained (including SIDS), occurring during infancy. Defined by the National Center for Health Statistics to mean deaths with an underlying cause code of ICD-10 R95, R99, or W75. ²⁴
Unexplained sudden death in infancy or sudden infant death syndrome (SIDS)	Surface sharing: Parent(s) and infant sleeping together on any surface. Medical examiners prefer "surface sharing" over "bed sharing." The sudden unexpected death of an apparently healthy infant under 1 y of age, in which investigation, autopsy, medical history review, and appropriate laboratory testing fails to identify a specific cause, including cases that meet the definition of sudden infant death syndrome. ⁶ The panel of experts representing the National Association of Medical Examiners recommends the use of unexplained sudden death in infancy and not sudden infant death syndrome. ⁵
Wedging or entrapment	A form of suffocation or mechanical asphyxia in which the nose and mouth or thorax is compressed or obstructed because of the infant being trapped or confined between inanimate objects, preventing respiration. ^{5,37} A common wedging scenario is an infant stuck between a mattress and a wall (or a bedframe) in an adult bed.

10) diagnostic codes. In the United States, the National Center for Health Statistics assigns a diagnostic code for SIDS (ICD-10 R95) if the cause of death listed on the death certificate is SIDS (including presumed, probable, or consistent with SIDS), sudden unexplained infant death, or other similar phrases that include "sudden" and "death."^{15,16} A death will be coded "other ill-defined and unspecified causes of mortality" (ICD-10 R99) if the cause of death is certified as unknown, unascertained, or undetermined.¹⁵ A death is coded "accidental suffocation and strangulation in bed" (ICD-10 W75) when the terms asphyxia, asphyxiated, asphyxiation, strangled, strangulated, strangulation, suffocated, or suffocation are used in the cause of death, along with the terms bed, crib, or other surfaces

such as couches and armchairs. ICD-10 W75 will be applied to both explained and unexplained deaths depending on the precise wording of the death certificate. In January 2022, the *International Statistical Classification of Diseases and Related Health Problems 11th Revision* (ICD-11) officially went into effect among World Health Organization member states. An international group of experts has proposed changes to the ICD to better define diagnostic codes for unexplained infant deaths and their meanings.⁶ This proposal is currently under review.

Although the term "SIDS" was not widely used until the mid-1980s,⁴ there was minimal change in the incidence of SIDS in the United States until the early 1990s. In 1992, in response to epidemiologic reports from Europe and Australia,

the AAP recommended that infants be placed for sleep in a nonprone position as a strategy to reduce the risk of SIDS.¹⁷ The "Safe to Sleep" campaign (formerly known as the "Back to Sleep" campaign) was launched in 1994 and spearheaded by the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD). Under the NICHD's continued leadership, this national public education effort is undertaken by several entities, including the AAP, the American College of Obstetricians and Gynecologists, the Division of Reproductive Health of the CDC, First Candle, the Maternal and Child Health Bureau of the Health Resources and Services Administration, and the United States Consumer Product Safety Commission (CPSC).¹⁸ Between 1992 and 2001, the SIDS rate

declined, with the most dramatic declines in the years immediately after the release of the first nonprone sleep position recommendations, and this decline was consistent with the steady increase in the prevalence of supine sleeping.¹⁹ The United States SIDS rate decreased from 120 deaths per 100 000 live births in 1992 to 56 deaths per 100 000 live births in 2001, representing a reduction of 53% over 10 years. From 2001 to 2008, the rate remained constant (Fig 1) and then declined from 54 per 100 000 live births in 2009 to 33 per 100 000 live births in 2019 (the latest year for which data are available). In 2019, 1248 infants died of SIDS.^{18,20} Overall, SIDS rates have declined by almost 75% since the early 1990s. However, in 2019, SIDS, unknown or unexplained cause, and accidental suffocation and strangulation in bed were the second, third, and fourth most common causes of overall infant mortality.²⁰ SIDS remains the

leading cause of postneonatal (28 days to 1 year of age) mortality.

As mentioned earlier, several studies have observed that some deaths previously classified as SIDS (ICD-10 R95) are now being classified as other causes of sleep-related infant death (eg, accidental suffocation and strangulation in bed [ASSB, ICD-10 W75] or other ill-defined or unspecified causes [ICD-10 R99])^{14,21,22} and that at least some of the decline in SIDS rates may be explained by increasing rates of these other assigned causes of death.^{21,23} To account for variations in certification and classification and to more consistently track unexplained sudden death and sleep-related infant deaths, the National Center for Health Statistics has created the special cause-of-death category, SUID (defined in this context as sudden unexpected infant death). This SUID category captures deaths with an underlying cause coded as

ICD-10 R95, R99, and W75.²⁴ In 2019, SIDS accounted for 37% of the 3376 SUIDs in the United States.²⁰

Similar to the SIDS rate, the SUID rate also declined in the late 2000s, from 99 per 100 000 live births in 2009 to 90.1 in 2019.²⁰

SUID rates vary dramatically by state.²⁵ From 2015 to 2019, there were 28 states with rates above the US average of 91.7 per 100 000 live births. Among the 50 states and the District of Columbia, Vermont had the lowest SUID rate (46 per 100 000 live births) and Mississippi had the highest SUID rate (185 per 100 000 live births).²⁰

Racial and Ethnic Disparities

SIDS and SUID mortality rates, like other causes of infant mortality, have notable and persistent racial and ethnic disparities, reflecting broader racial and ethnic societal inequities.²⁰ Despite the decline in SIDS and SUIDs in all races and

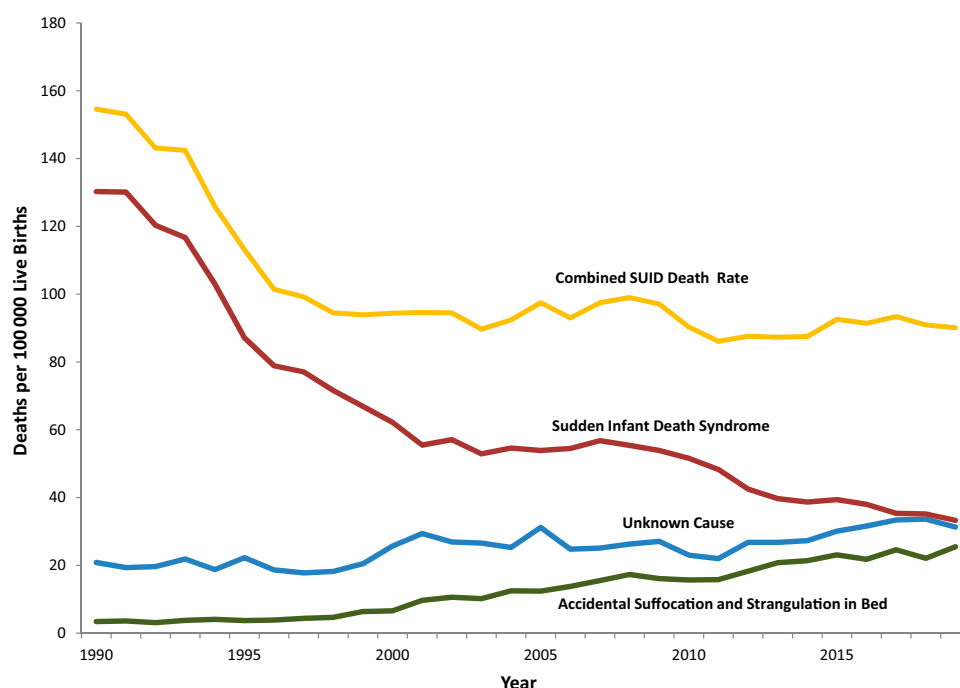


FIGURE 1

Trends in sleep-related infant deaths by cause from 1990 to 2019 from the Centers for Disease Control and Prevention and National Center for Health Statistics, National Vital Statistics System, Compressed Mortality File. Figure duplicated from <http://www.cdc.gov/sids/data.htm>.

ethnicities, the rate of SUIDs among non-Hispanic Black (187 per 100 000 live births) and American Indian and Alaska Native (212 per 100 000 live births) infants was more than double and almost triple, respectively, that of non-Hispanic White infants (85 per 100 000 live births) between 2010 and 2013 (Fig 2). SUID rates for Asian and Pacific Islander and Hispanic infants (54 and 34 per 100 000 live births, respectively) were much lower than the rate for non-Hispanic White infants. These racial and ethnic disparities are observed with deaths attributed to SIDS, ASSB, and ill-defined or unspecified deaths (Fig 2). Furthermore, racial and ethnic disparities have worsened. Compared with non-Hispanic White infants, SUID rates for non-Hispanic Black and American Indian and Alaska Native infants decreased more slowly, and rates for Asian and Pacific Islander and Hispanic infants have decreased more rapidly.²⁶

Differences in the prevalence of supine positioning and other sleep environment conditions among different racial and ethnic populations may contribute to these

disparities.²⁷ The factors underlying these disparities are likely multidimensional. Studies have indicated that factors, such as low socioeconomic status (SES) or low socioeconomic position,²⁸ unemployment, housing instability, and domestic violence, which leave families with infants socially vulnerable, are associated with increased prevalence of known risk factors for sudden unexpected death in infancy.²⁹ These factors are also highly correlated with race and ethnicity in the United States.³⁰ Low SES has consistently been associated with higher risk of SIDS and SUID.³¹ The risk of low SES has been demonstrated across a wide range of socioeconomic characteristics, including income, social status, maternal education, and employment.³¹ On the basis of data from 29 states participating in the Pregnancy Risk Assessment and Monitoring System (PRAMS),³² the prevalence of usual supine positioning in 2016 among non-Hispanic White infants was 84%, compared with 62%, 74%, and 76% among non-Hispanic Black, Hispanic, and non-Hispanic Asian and Pacific Islander infants, respectively.²⁷

Parent-infant bed sharing^{33–35} and use of soft bedding are also more common among Black families than among other racial and ethnic groups.²⁷ Addressing the potential impact of structural racism; recognizing the lack of access to economic, social, and educational resources as a risk factor for sleep-related deaths; working closely with communities to identify possible unknown risk factors; and engaging health care and public health professionals in thoughtful and respectful conversation with families about safe infant sleep will be important in improving understanding of the most effective strategies to promote adoption of safe infant sleep practices among various populations.

Age at Death

Sudden unexpected infant death rates differ by age at death. In general, SUID occurs more frequently in younger infants.³⁶ For example, during 2011 to 2013, 76% to 86% of SUID cases in the United States occurred from 0 through 4 months of age, with a peak at 1 to 2 months.²⁶ With regard to SIDS specifically, 90% of cases occur

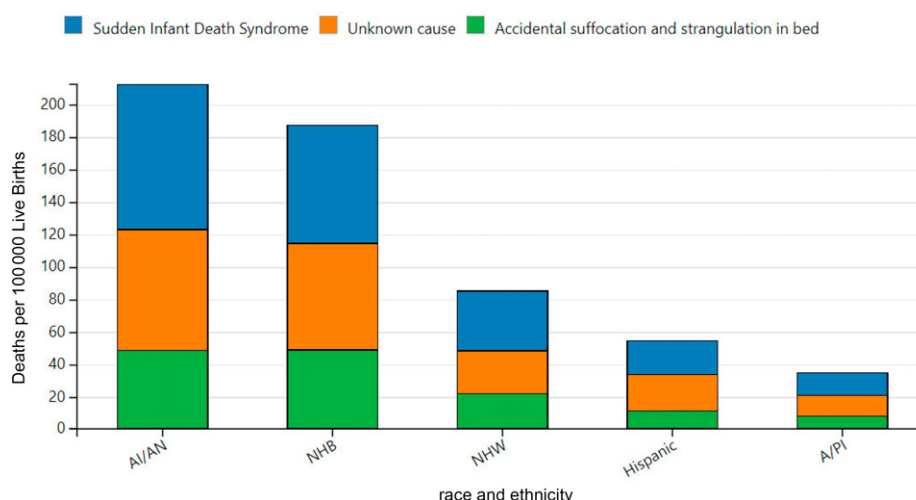


FIGURE 2

Sudden unexpected infant death by race and ethnicity from 2014 to 2018 from the Centers for Disease Control and Prevention and National Center for Health Statistics, National Vital Statistics System, Compressed Mortality File. Figure duplicated from <http://www.cdc.gov/sids/data.htm>. AI and AN, American Indian and Alaska Native; NHB, Non-Hispanic Black; NHW, Non-Hispanic White; A and PI, Asian and Pacific Islander.

before an infant reaches the age of 6 months.¹³ SIDS peaks between 1 and 4 months of age and is uncommon after 8 months of age.¹³ Although a similar age distribution is seen for ASSB,³⁷ there are distinct patterns in age at death within different mechanisms of ASSB. The median age at death for suffocations attributable to soft bedding is 3 months, and the median age at death for suffocations attributable to overlay and wedging are 2 and 6 months, respectively.³⁷

In recent years, there has been increasing attention to sudden unexpected deaths occurring in the neonatal period, namely sudden unexpected postnatal collapse and sudden unexpected early neonatal deaths.^{38,39} In 2019, SUID accounted for 129 deaths at 0 to 6 days and 288 deaths at 7 to 27 days. Similar to postneonatal SUID, the cause of many of these deaths remains unexplained; however, the risk factors and mechanisms may be different. Ongoing surveillance of SUID rates by age at death is important to evaluate the impact of infant care interventions, identify new risk factors, and track progress toward reducing SUID mortality.²²

PATHOPHYSIOLOGY AND GENETICS OF SUDDEN INFANT DEATH

The pathophysiology of sudden death in infants is complex and incompletely understood because of the expanse and heterogeneity of factors and mechanisms involved. The most widely held conceptual framework of SIDS pathogenesis is the triple risk model, which describes convergence of exogenous factors or stressors (eg, prone or side sleep position, overbundling, airway obstruction), a critical period of development (the highest risk being from 1 to 4 months of age), and intrinsic vulnerability (eg, dysfunctional and/or immature cardiorespiratory and/or arousal

systems) leading to death (Fig 3).⁴⁰ The exogenous stressor initiates a fatal sequence of mechanisms, made possible by the pre-existing milieu of immaturity and intrinsic vulnerabilities or actual abnormalities. Thus, each fatality results from interaction of multiple factors, which vary from case to case, making identification of a single cause or universal sequence of mechanisms for sudden death extremely challenging. However, common themes have emerged. Recognition of external stressors, most often potentially asphyxiating and/or overheating sleep environments, has substantially increased because of improved death investigation and systematic review of case series. Progressive asphyxia, bradycardia, hypotension, metabolic acidosis, and ineffectual gasping or arousal are among the more common lethal mechanisms hypothesized.⁴¹ Research on intrinsic vulnerabilities has uncovered compelling anatomic, genetic, and physiologic developmental factors or anomalies in many cases, particularly with respect to dysfunctional cardiorespiratory and/or arousal systems. Although the triple risk model proposes that these deaths will necessarily have a contribution from each of the 3 model components (external stressor,

critical developmental period, and intrinsic vulnerability),⁴² each is not demonstrable in all sudden infant deaths at the individual case level.

The most common intrinsic vulnerabilities recognized to date include in utero environmental conditions, maldevelopment, or delay in maturation,^{43,44} and genetically determined conditions. Infants who die suddenly and unexpectedly are more likely to have been born preterm and/or were growth restricted, which suggests a suboptimal intrauterine environment.^{45,46} Other adverse in utero environmental conditions include exposure to nicotine or other components of cigarette smoke and alcohol.⁴⁷

Numerous studies have explored how prenatal exposure to cigarette smoke may result in an increased risk for SIDS. The physiologic consequence of in utero nicotine exposure have been recently reviewed.⁴⁸ In animal models, exposure to cigarette smoke or nicotine during brain development alters the expression of the nicotinic acetylcholine receptors in areas of the brainstem important for autonomic function and alters the numbers of orexin receptors in piglets^{49–51}; reduces the number

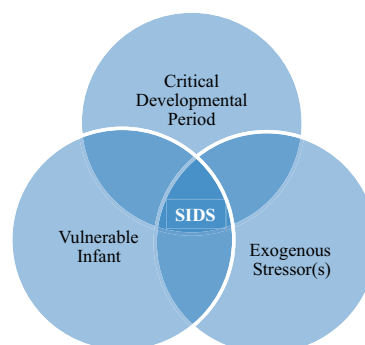


FIGURE 3

The Triple Risk Model proposes that SIDS occurs when an infant with intrinsic vulnerability (often manifested by impaired arousal, cardiorespiratory, and/or autonomic responses) undergoes an exogenous trigger event (eg, exposure to an unsafe sleeping environment) during a critical developmental period.⁴⁰

and activity^{49,50} of medullary serotonergic (serotonin or 5-hydroxytryptamine [5-HT]) neurons in the raphe obscurus in mice⁵²; increases 5-HT and 5-HT turnover in baboons⁵³; alters neuronal excitability of neurons in the nucleus tractus solitarius (a brainstem region important for sensory integration) in guinea pigs⁵⁴; and alters fetal autonomic activity and medullary neurotransmitter receptors, including nicotinic receptors, in baboons.^{55–57} From a functional perspective, prenatal exposure to nicotine causes hypoventilation and increased apnea,^{57–61} reduces hypercarbia and hypoxia-induced ventilator chemoreflexes in rodents^{52,58,59,62} and lambs,⁶³ and blunts arousal in response to hypoxia in rats⁶² and lambs.⁶³

In human infants, there are strong associations between nicotinic acetylcholine receptors and serotonergic (5-HT) receptors in the brainstem during development,⁶⁴ and there is important recent evidence of epigenetic changes in the placentas of infants with prenatal tobacco smoke exposure.⁶⁵ In some infants who have died of SIDS, brainstem alterations of acetylcholine receptor subtype distribution and expression have been identified,⁶⁶ and increased programmed cell death in the hippocampus and brainstem⁶⁷ and altered expression of brain-derived neurotrophic factor, a growth factor with crucial roles in neuronal differentiation, survival and synaptic transmission,⁶⁸ have been associated with gestational cigarette smoke exposure. Prenatal exposure to tobacco smoke attenuates recovery from hypoxia in preterm infants,⁶⁹ decreases heart rate variability in preterm⁷⁰ and term⁷¹ infants, and abolishes the normal relationship between heart rate and gestational age at birth.⁷⁰ Infants

born to substance misusing and smoking mothers have an impaired ventilatory response to hypoxic challenges during quiet sleep and in the prone position^{72,73} and impaired arousal patterns to trigeminal stimulation in proportion to urinary cotinine concentrations.⁷⁴ It is important to note also that prenatal exposure to tobacco smoke alters the normal programming of cardiovascular reflexes, such that the increase in blood pressure and heart rate in response to breathing 4% carbon dioxide (CO₂) or a 60° head-up tilt is greater than expected.⁷⁵ These changes in autonomic function, arousal, and cardiovascular reflexes may all increase an infant's vulnerability to a sleep-related death.

The brainstem plays a key role in coordinating many respiratory, arousal, and autonomic functions, and when dysfunctional, might prevent normal protective responses to stressors that commonly occur during sleep. A large systematic review of the neuropathological features of unexplained sudden infant death, including only studies that met strict criteria, concluded that "... the most consistent findings, and most likely to be pathophysiologically significant, are abnormalities of serotonergic neurotransmission in the caudal brain stem."⁷⁶ Brainstem abnormalities that involve the 5-HT (serotonin) system in up to 70% of infants who die of SIDS have now been confirmed in several independent data sets and laboratories.^{47,77–79} These include decreased serotonin 1A (5-HT1A) receptor binding, a relative decreased binding to the 5-HT transporter, abnormalities of 5-HT neuron number, density and morphology, and decreased tissue levels of 5-HT and the rate-limiting enzyme for 5-HT synthesis, tryptophan hydroxylase.^{80,81}

Moreover, 5-HT deficiency is attributable to impaired synthesis, rather than excessive serotonin degradation, as assessed by levels of 5-hydroxyindoleacetic acid (the main metabolite of serotonin) or ratios of 5-hydroxyindoleacetic acid to serotonin.⁵⁵ The brainstem 5-HT system is involved in termination of apneas,^{82–84} and even partial dysfunction of the raphe serotonergic system has been shown to impair autoresuscitation and increase mortality in mice.⁸⁵

There are significant associations between brainstem 5-HT1A receptor binding abnormalities and specific SIDS risk factors, including tobacco smoking.⁷⁹ These data confirm results from earlier studies in humans^{47,80} and are also consistent with studies in piglets that reveal that postnatal exposure to nicotine decreases medullary 5-HT1A receptor immunoreactivity.⁸⁶ Serotonergic neurons located in the medullary raphe and adjacent paragigantocellularis lateralis play important roles in many autonomic functions, including the control of respiration, blood pressure, heart rate, thermoregulation, sleep and arousal, and upper airway patency. Engineered mice with decreased numbers of 5-HT neurons and rats or piglets with decreased activity secondary to 5-HT1A autoreceptor stimulation have diminished ventilator responses to CO₂, dysfunctional heat production and heat loss mechanisms, and altered sleep architecture.⁸⁷ The aberrant thermoregulation in these models provides evidence for a biological substrate for the risk of SIDS associated with potentially overheating environments. In addition, mice pups with a constitutive reduction in 5-HT-producing neurons (PET1 knockout) or rat pups in which a large fraction of medullary 5-HT neurons have been destroyed with

locally applied neurotoxins have a decreased ability to autoresuscitate in response to asphyxia.^{88,89} Moreover, animals with 5-HT neuron deficiency caused by direct injection of a 5-HT selective neurotoxin have impaired arousal in response to hypoxia.⁹⁰

Potentially relevant findings are not confined to serotonergic nuclei but also include projection sites and other brainstem structures. For example, abnormalities of Phox2B immune-reactive neurons have been reported in the human retrotrapezoid nucleus, a region of the brainstem that receives important 5-HT projections and is critical to CO₂ chemoreception and implicated in congenital central hypoventilation syndrome.⁹¹ Through continued in-depth examination of the brainstem of unexplained and explained infant deaths, hypoplasia of nuclei and neuronal abnormalities are being recognized in an expanding list of brainstem structures involved in regulation of homeostasis and vital functions.⁹¹

The brainstem has important reciprocal connections to the limbic system comprising both cortical and subcortical components, including the limbic cortex, hypothalamus, amygdala, and hippocampus. These areas of the brain are also important in regulation of autonomic function, particularly in response to emotional stimuli. Thus, the brainstem and limbic system constitute a key network in controlling many aspects of autonomic function. Morphologic changes of the dentate gyrus (a component of the hippocampal formation) and hippocampal gliosis have been identified in a portion of unexplained infant deaths and more frequently in sudden unexplained death of older children and persons with epilepsy.⁹² However, the occurrence of such findings in the hippocampal formation of controls

suggest further studies are needed to explore the specificity and significance of these findings and the implication that SIDS may share mechanisms with sudden death in people with epilepsy and children with febrile seizures.

Abnormalities of other systems involved in cardiorespiratory control and arousal have been demonstrated in SIDS, including the noradrenergic system,⁹³ glutamatergic and GABAergic systems, central and peripheral chemoreceptors (reviewed⁹⁴), orexin-producing neurons,^{95,96} and hypothalamus (reviewed⁹⁷), spurring continued refinement and expansion of hypotheses for mechanisms for increased vulnerability and death. Structural and neurochemical abnormalities of the systems thus far described are not typically demonstrable by routine postmortem examination of tissues without the use of special research techniques and preparations. However, identification of elevated serum 5-HT levels in a subset of SIDS not only presents the possibility of a relevant biomarker for the future but also indicates a potential association with peripheral serotonin abnormalities that will require further study.⁹⁸

Some cases of unexpected infant death have a genetic cause. Genetic variation can take the form of common base changes (polymorphisms) that alter gene function or rare base changes (mutations) that often have highly deleterious effects.⁹⁹ (For a comprehensive review, see Opdal and Rognum¹⁰⁰) To date, genetic studies have shown that the basis for the pattern of genetic variations associated with SIDS is heterogeneous. Mutations in genes controlling metabolic functions or cardiac ion channels are represented by diseases such as medium-chain

acyl-coenzyme A dehydrogenase deficiency and long QT syndrome (LQTS).¹⁰¹ A recent California study showed that the frequency of mutations for undiagnosed inborn errors of metabolism was similar in SIDS and controls and that newborn screening was effective in detecting medium-chain and very long-chain acyl-coenzyme A dehydrogenase deficiencies that could potentially lead to sudden death.¹⁰² In the instance of LQTS, 700 mutations identified in 12 genes are the predominant variations detected.¹⁰³ Although the manifestation of LQTS resulting in sudden infant death may differ, the primary mechanism results in a cardiac arrhythmia attributable to dysfunctional sodium or potassium cardiac receptor channels. It has been estimated that 5% to 10% of infants who die suddenly and unexpectedly have novel mutations in the cardiac sodium or potassium channel genes resulting in LQTS as well as in other genes that regulate channel function.¹⁰⁴ Some of these mutations may represent an actual cause of death, but others may contribute to causing death when combined with environmental factors, such as acidosis.¹⁰⁵ There is molecular and functional evidence that implicates specific SCN5A (sodium channel gene) β subunits in SIDS pathogenesis.¹⁰⁶ In addition, 2 rare mutations in connexin 43, a major gap junction protein, have been found in SIDS cases and not in ethnically matched controls.¹⁰⁷ In vitro assays of 1 mutation showed a lack of gap junction function, which could lead to ventricular arrhythmogenesis. The other mutation did not appear to have functional consequences. A recent study also adds weight to the need to perform functional assays and morphologic studies of the altered gene products. Several of the missense variants in genes encoding cardiac channels that have been

found in SIDS cases had high prevalence in the National Heart, Lung, and Blood Institute GO Exome Sequencing Project Database.¹⁰⁸ A large study of a nonreferred nationwide Danish cohort estimates that up to 7.5% of SIDS cases may be explained by genetic variants in the sodium channel complex.¹⁰⁹ These estimates are in the range of those previously reported. However, it is important that for each channelopathy variant discovered, the biological plausibility for pathogenicity is investigated to consider it as a cause of or contributor in SIDS.^{110,111}

Several categories of physiologic functions relevant to SIDS have been examined for altered genetic makeup. Genes related to the serotonin transporter, cardiac channelopathies, and the development of the autonomic nervous system are the subject of current investigation.¹⁰⁴ The serotonin transporter recovers serotonin from the extracellular space and largely serves to regulate overall serotonin neuronal activity. There are reports that polymorphisms in the promoter region that enhance the efficacy of the transporter (L) allele seem to be more prevalent in infants who die of SIDS compared with polymorphisms that reduce efficacy (S)¹⁰⁰; however, at least 1 study did not confirm this association.¹¹² It has also been reported that a polymorphism (12-repeat intron 2) of the promoter region of the serotonin transporter, which also enhances serotonin transporter efficiency, was increased in Black infants who died of SIDS¹⁰⁴ but not in a Norwegian population.¹⁰⁰

An impaired ability for an infant to mount an immune response to infections may also create an intrinsic vulnerability for SIDS. The immunomodulatory genes identified in 251 cases of SIDS by Hafke et al

provide insight into the potential role and contribution of the immune system.¹¹³ Two variants in interferon γ and 1 variant in interferon α 8 were shown to have statistically significant associations with the occurrence of SIDS when single nucleotide polymorphisms were analyzed. Fard et al were unable to replicate this finding through genotyping of 40 single nucleotide polymorphisms from 15 candidate genes but did show minimal evidence of associations with variants in interleukin 6 and interleukin 10, supporting the potential role of infection and inflammation in SIDS.^{113,114}

The identification of polymorphisms in genes pertinent to the embryologic origin of the autonomic nervous system in SIDS cases also lends support to the hypothesis that a genetic predisposition contributes to the etiology of SIDS. The pituitary adenylate cyclase-activation polypeptide (PACAP) gene and the gene of 1 of its receptors (PAC1) have received recent attention because of a possible association of SIDS cases with specific alleles.¹¹⁵ This association between variants in the PAC1 gene and SIDS was not found in another study, but a number of potential associations between genetic variants and SIDS were identified; these warrant further study.¹¹⁶ Variant mutations in the brain aquaporins AQP1 and AQP9 have been found more frequently in SIDS cases, supporting the theory of a genetic predisposition of regulatory brainstem function as a mechanism for death.

Previous studies of racial differences in the genetics of SIDS have largely been limited to differences between Black and White infants. Race is a social construct¹¹⁷ and can be a proxy for aspects of one's lived experiences (educational,

economic, housing, etc) that can affect health outcomes.¹¹⁸ Adverse childhood experiences are associated with epigenetic changes that may help to explain disparities.^{119,120} As we continue to research the polymorphisms or mutations in genes regulating inflammation,^{121–123} energy production,^{124–127} and hypoglycemia^{127,128} in infants who died of SIDS, the associations between these polymorphisms and epigenetic changes require more study to determine their importance. The role of epigenetics on any observed racial and ethnic differences should be prioritized in future research.

RECOMMENDATIONS TO REDUCE THE RISK OF SLEEP-RELATED INFANT DEATHS

The recommendations outlined herein were developed to reduce the risk of sleep-related infant deaths, including SIDS and sleep-related suffocation, asphyxia, and entrapment. As defined by epidemiologists, risk refers to the probability that an outcome will occur given the presence of a particular factor or set of factors. Although all 19 recommendations are intended for everyone who cares for infants, the last 4 recommendations are directed specifically toward health policy makers, researchers, and professionals who care for or work on behalf of infants. In addition, because certain behaviors, such as smoking, can increase risk for the infant, some recommendations are directed toward people who are pregnant or may become pregnant in the near future.

The guidance in this technical report is intended to be inclusive of all families. Gendered language, such as “mothers” and “breastfeeding,” is occasionally used, particularly when discussing or quoting published

articles that used these definitions.¹²⁹ However, the authors acknowledge that parents may be of any gender and that transgender men and nonbinary-gendered individuals may also give birth and/or may want to breastfeed or feed at the chest.

The recommendations, along with the strength of recommendation, are summarized in the accompanying policy statement, “Sleep-Related Infant Deaths: Updated 2022 Recommendations for Reducing Infant Deaths in the Sleep Environment.”¹³⁰ It should be noted that because there are no randomized controlled trials related to SIDS and other sleep-related deaths, case-control studies are the best evidence available.

The recommendations are based on studies that include infants up to 1 year of age. Therefore, recommendations for sleep position and the sleep environment, unless otherwise specified, are for the first year after birth. The evidence-based recommendations that follow are provided to guide pediatricians, other physicians, and nonphysician clinicians in conversations with parents and others who care for infants. Physicians and nonphysician clinicians are encouraged to have open and nonjudgmental conversations with families about their sleep practices. Individual medical conditions may warrant that a clinician recommend otherwise after weighing the relative risks and benefits.

INFANT SLEEP POSITION

To reduce the risk of sleep-related death, it is recommended that infants be placed for sleep in the supine (back) position for every sleep by every caregiver until the child

reaches 1 year of age. Side sleeping is not safe and is not advised.

The prone or side sleep position can increase the risk of rebreathing expired gases, resulting in hypercapnia and hypoxia.^{131–134} The prone position also increases the risk of overheating by decreasing the rate of heat loss and increasing body temperature more than the supine position.^{135,136} Evidence suggests that prone sleeping alters the autonomic control of the infant cardiovascular system during sleep, particularly at 2 to 3 months of age,¹³⁷ and may result in decreased cerebral oxygenation.¹³⁸ The prone position places infants at high risk for SIDS (odds ratio [OR], 2.3–13.1).^{139–143} In 1 US study, SIDS risk associated with side position was similar in magnitude to that associated with prone position (OR, 2.0 and 2.6, respectively),¹⁴⁰ and a higher population-attributable risk has been reported for side sleep position than for prone position.^{142,144} Furthermore, the risk of SIDS is exceptionally high for infants who are placed on the side and found on the stomach (OR, 8.7).¹⁴⁰ The side sleep position is inherently unstable, and the probability of an infant rolling to the prone position from the side sleep position is significantly greater than rolling prone from the back.^{142,145} Infants who are unaccustomed to the prone position and are placed prone for sleep are also at greater risk than those usually placed prone (adjusted OR [aOR], 8.7–45.4).^{140,146,147} It is, therefore, critically important that every caregiver place the infant in the supine sleep position for every sleep. This is particularly relevant in situations in which a new caregiver is introduced—for example, when an infant is placed in foster care or an adoptive home, or when an infant enters child care for the first

time or has a change in child care providers.

Despite these recommendations, the prevalence of supine positioning has remained stagnant for the last decade.^{27,148} One reason often cited by parents for not using the supine sleep position is the perception that the infant is uncomfortable or does not sleep well.^{149–157} However, an infant who wakes frequently is typical and should not be perceived as a poor sleeper. Physiologic studies demonstrate that infants are less likely to arouse when they are sleeping in the prone position.^{158–166} The ability to arouse from sleep is an important protective physiologic response to stressors during sleep,^{167–171} and the infant’s ability to sleep for sustained periods may not be physiologically advantageous.

The supine sleep position on a firm, flat, noninclined surface does not increase the risk of choking and aspiration in infants and is recommended for every sleep, even for infants with gastroesophageal reflux.

Parents and caregivers continue to be concerned that an infant will choke or aspirate while supine.^{149–157} Parents often misconstrue coughing or gagging, which is evidence of a normal protective gag reflex, for choking or aspiration. Multiple studies in different countries have not demonstrated an increased incidence of aspiration since the change to supine sleeping.^{172–174} Parents and caregivers are often concerned about aspiration when the infant has been diagnosed with gastroesophageal reflux (GER). The AAP concurs with the North American Society for Pediatric Gastroenterology and Nutrition that “... no position other than supine position is recommended for infants because of the risk of sudden infant

death syndrome (SIDS).¹⁷⁵ Further, “the working group recommends not to use positional therapy (ie, head elevation, lateral and prone positioning) to treat symptoms of GERD (gastroesophageal reflux disease) in sleeping infants.”¹⁷⁵ There is no evidence to show that infants receiving nasogastric or orogastric feeds are at increased risk for aspiration if placed in the supine position. Elevating the head of the infant’s crib while the infant is supine is ineffective in reducing gastroesophageal reflux^{176,177} and is not recommended. Additionally, a recent biomechanical analysis found that infants cannot be placed at a 30 degree incline without sliding down.¹⁷⁸ This raises concern that the infant could slide into a position that may compromise respiration. This analysis also found that infants sleeping at lesser inclines can more easily flex their trunk and lift their head, facilitating rolling onto the side or prone, at which point they are at higher risk for muscle fatigue and potential suffocation.¹⁷⁸

Place hospitalized preterm infants supine as soon as clinical status has stabilized and they have achieved positional stability (ie, when therapeutic or nonsupine positioning is no longer medically indicated).

Infants born preterm (<37 weeks’ gestational age) have an increased risk of SIDS.^{46,179,180} Additionally, the association between prone position and SIDS among low birth weight and preterm infants is equal to, or perhaps even stronger than, the association among those born at term.¹⁴⁶ Therefore, preterm infants should be placed supine for sleep as soon as clinical status has stabilized and they have achieved positional stability—in other words, when therapeutic or nonsupine positioning is no longer medically indicated. This is usually achieved by 32 weeks’ gestational age as the infant’s flexion tone and strength

develops.^{181,182} The AAP reiterates its previous recommendation that (1) “preterm infants should be placed supine for sleeping, just as term infants should, and the parents of preterm infants should be counseled about the importance of supine sleeping in preventing SIDS. Hospitalized preterm infants should be kept predominantly in the supine position, at least from the postmenstrual age of 32 weeks onward, so that they become acclimated to supine sleeping before discharge,”¹⁸³ and (2) even among preterm infants with GER, “safe sleep approaches, including supine positioning on a flat and firm surface and avoidance of commercial devices designed to maintain head elevation in the crib, should be paramount as a model for parents of infants approaching discharge (ie, infants greater than 32 weeks’ postmenstrual age) from the hospital.”¹⁸⁴ Further, the AAP believes that neonatologists, neonatal nurses, and other clinicians responsible for organizing the hospital discharge of infants from NICUs should be vigilant about endorsing recommendations to reduce the risk of sleep-related death from birth. They should model these recommendations as soon as the infant is medically stable and significantly before the infant’s anticipated discharge from the hospital. In addition, NICUs are encouraged to develop and implement policies to ensure that supine sleeping and other safe sleep practices are modeled for parents before discharge from the hospital.^{185,186} See “Transition to a Safe Home Sleep Environment for the NICU Patient” for additional details.¹⁸⁷

During the birth hospitalization, place healthy newborn infants supine and on a flat, noninclined surface for every sleep when they are not

engaged in skin-to-skin care or in the arms of an awake or alert individual.

As stated in the AAP clinical report on safe sleep and skin-to-skin care, “skin-to-skin care is recommended for all mothers and newborns, regardless of feeding or delivery method, immediately following birth (as soon as the mother is medically stable, awake, and able to respond to her newborn), and to continue for at least an hour.”¹⁸⁸ Thereafter, or when the parent needs to sleep or take care of other needs, infants should be placed supine in a noninclined bassinet.

Placement of infants on the side after birth by physicians, nurses, or other clinicians continues to be a concern. The practice likely occurs because of a belief among hospital staff that newborn infants need to clear their airways of amniotic fluid and may be less likely to aspirate while on the side. No evidence that such fluid will be cleared more readily while in the side position exists. Perhaps most importantly, if parents observe physicians, nurses, or other clinicians placing infants in the side or prone position, they are likely to infer that supine positioning is not important¹⁸⁹ and may, thus, be more likely to copy this practice and use the side or prone position at home.^{154,157,190} Infants who are rooming in with their parents or cared for in a separate newborn nursery should be placed in the supine position as soon as they are ready to be placed in the bassinet. To promote breastfeeding, placing the infant skin-to-skin with parent after delivery, with appropriate observation and/or monitoring, is the best approach. When the parent needs to sleep or take care of other needs, the infant should be placed supine in a bassinet.

Infants who can roll from supine to prone and from prone to supine can

be allowed to remain in the sleep position that they assume.

Parents and caregivers are frequently concerned about the appropriate strategy for infants who have learned to roll over, which generally occurs at 4 to 6 months of age. As infants mature, it is more likely that they will roll. In 1 study, 6% and 12% of 16- and 23-week-old infants placed on their backs or sides, respectively, were found in the prone position; among infants ≥ 24 weeks of age, 14% of those placed on their backs and 18% of those placed on their sides were found in the prone position.¹⁹¹ Because data to make specific recommendations as to when it is safe for infants to sleep in the prone position are lacking, the AAP recommends that all infants continue to be placed supine until 1 year of age. Infants who can roll from supine to prone and from prone to supine can be allowed to remain in the sleep position that they assume. One study analyzing sleep-related deaths reported to state child death review teams found that the predominant risk factor for sleep-related deaths in infants 4 to 12 months of age was rolling into objects in the sleep area.¹⁹² Thus, parents and caregivers should continue to keep the infant's sleep environment clear of everything but a fitted sheet. Parents may be reassured in being advised that the incidence of SIDS begins to decline after 4 months of age.²²

SLEEP SURFACES

Use a firm, flat, noninclined sleep surface (eg, tightly fitting mattress in a safety-approved crib) covered by a fitted sheet with no other bedding or soft objects to reduce the risk of suffocation or wedging or entrapment.

Place infants on a firm, flat, noninclined sleep surface (eg, tightly fitting mattress in a safety-approved crib) covered by a fitted sheet with

no other bedding or soft objects. A firm surface maintains its shape and does not indent or conform to the shape of the infant's head when the infant is placed on the surface. The surface does not change its shape when the fitted sheet designated for that model is used, such that there are no gaps between the mattress and the wall of the crib, bassinet, portable crib, or play yard. Soft mattresses, including those with adjustable firmness or those made from memory foam, could create a pocket (or indentation) and increase the chance of rebreathing or suffocation if the infant is placed in or rolls over to the prone position.^{133,193} Many mattresses intended for use by older children or adults contain memory foam or have adjustable firmness. The use of mattresses that are soft, adjustable, or with memory foam is dangerous for infants.

A flat, noninclined surface is safest for infants. An independent expert hired by the CPSC conducted infant testing to evaluate inclined sleep products and demonstrated that none of these tested products were safe for infant sleep. Infants on an inclined surface can more easily flex their trunk and lift their head, facilitating rolling onto the side or prone, at which point they are at higher risk for muscle fatigue and potential suffocation. This report concluded that products with inclines of more than 10 degrees are unsafe for infant sleep.¹⁷⁸

A crib, bassinet, portable crib, or play yard that conforms to the safety standards of the CPSC is recommended.

A crib that is safety-approved is 1 that meets the safety standards of the CPSC, including those for slat spacing, snugly fitting and firm mattresses, and no drop sides.¹⁹⁴ The AAP recommends the use of new cribs, because used cribs may

no longer meet current safety standards, may have missing parts, or may be incorrectly assembled. In addition, parents and providers should check the CPSC Web site (www.cpsc.gov) to ensure that the product has not been recalled. This is particularly important for used cribs. If a used crib is to be used, care must be taken to ensure that there have been no recalls on the crib model, that all of the hardware is intact, and that the assembly instructions are available. Cribs with missing hardware or missing instructions should not be used, nor should parents or providers attempt to fix broken components of a crib, because many deaths have occurred in cribs that were broken or with missing parts (including those that had presumably been fixed).

For some families, use of a crib may not be possible for financial or space considerations. In addition, parents may be reluctant to place the infant in the crib because of concerns that the crib is too large for the infant or that "crib death" (ie, SIDS) only occurs in cribs, a common misunderstanding of the evidence. These concerns should be assessed and addressed by physicians and nonphysician clinicians and include a conversation with the parents about the importance of safe sleep environments to reduce the risk of sleep-related death. Smaller sleep surfaces, such as portable cribs, play yards, and bassinets that meet safety standards of the CPSC^{195,196} can be used and may be more acceptable for some families because they are smaller, more portable, and typically more affordable.

Ensure that mattresses are firm, flat, and maintain their shape even when the fitted sheet designated for that model is used and that there are no gaps between the mattress and the wall of the bassinet, playpen, portable crib, play yard, or bedside

sleeper. Only use mattresses designed for the specific product. Do not use pillows or cushions as substitutes for mattresses or in addition to a mattress. It is not safe to place soft materials or objects, such as pillows (including semi-circular or other nursing pillows), quilts, comforters, or fur-like materials, even if covered by a sheet, under a sleeping infant. Mattress toppers, designed to make the sleep surface softer, are not safe for infants younger than 1 year. Any fabric on the crib walls or a canopy could create a suffocation risk for the infant and is not recommended.

Do not place infants for sleep on adult-sized beds or mattresses because of the risk of entrapment and suffocation.¹⁹⁷ Portable bed rails (railings installed on the side of the bed that are intended to prevent an older child from falling off of the bed) should not be used with infants because of the risk of entrapment and strangulation.¹⁹⁸ Keep the infant sleep area free of hazards, including dangling cords, electric wires, and window covering cords, because these may present a strangulation risk.

There are commercially available special crib mattresses and sleep surfaces that claim to reduce the chance of rebreathing CO₂ when the infant is in the prone position that have been introduced. Although there are no apparent disadvantages of using these mattresses if they meet the safety standards as described previously, no studies have demonstrated decreased risk of death. (See section on Commercial Devices for further discussion of special mattresses.)

Bedside sleepers are attached to the side of the parental bed. The CPSC has published safety standards for bedside sleepers,¹⁹⁹ and they may be considered by some parents as an option.

There is inadequate published evidence to recommend for or against the use of alternative sleep surfaces. At a minimum, to be considered a safe option, any alternative sleep surface (such as inclined sleep products, hammocks, cardboard boxes, in-bed sleepers [including pepi-pods or wahakuras], baby nests and pods, compact bassinets without a stand or legs, travel bassinets, and baby tents) should adhere to the June 2021 CPSC rule that any infant sleep product must meet existing federal safety standards for cribs, bassinets, play yards, and bedside sleepers.

In June 2021, the CPSC passed a rule that any sleep products for infants 5 months and younger (defined as any product with packaging, marketing, or instructions indicating that the product is for sleep or naps or with any images of sleeping infants) must meet the existing federal safety standards for cribs, bassinets, play yards, and bedside sleepers.²⁰⁰ This includes inclined sleep products, hammocks, cardboard boxes, in-bed sleepers, baby nests and pods, compact bassinets without a stand or legs, travel bassinets, and baby tents. The AAP does not recommend use of any products that do not meet the federal safety standard, as they are likely not safe for infant sleep.

There are a variety of in-bed sleepers, many commercially available, and others mostly used for research purposes.^{201–203} Studies in New Zealand have compared overnight vital signs for infants using 2 in-bed sleepers (wahakura, a flax-woven sleeper for the Maori population, and the pepi-pod, a plastic version of the wahakura) with historical bassinet controls and found no differences in oxygen saturations or skin temperature; however, infants in the pepi-pod had a higher average heart rate (146 $\pm$ 8.8 vs 138 $\pm$ 10.1; $P < .001$).²⁰² A similarly designed study evaluating the wahakura

compared with a bassinet found no differences in oxygen saturations, desaturation events, heart rate, or temperature.²⁰³ Additionally, studies comparing these 2 devices to bassinets have shown no differences in prone or side sleep positioning, head covering, or direct bed sharing, although 1 trial found poorer maternal sleep quality with the wahakura at 1 month of age.^{201, 203} Although these small studies are encouraging, there is wide variation in the design of in-bed sleepers. In-bed sleepers that do not meet the federal safety standard²⁰⁰ are likely not safe for infant sleep and should not be used. In a retrospective review of CPSC hazard reports associated with bedside and in-bed sleepers, there were 6 deaths and 20 injuries.²⁰⁴ Among the 6 deaths (mean age 3.1 months), 5 of the deaths were attributable to asphyxia and 1 was attributable to SIDS. Half of the deaths were associated with the same model of in-bed cosleeper, and the other half involved bedside sleepers from 1 manufacturer. Four cases had additional environmental risk factors. Of the 20 reported injuries (mean age 4.8 months), 70% occurred with bedside sleepers. The most common injury hazards were entrapment and suffocation, with mechanism of injury involving the infant becoming trapped in gaps and spaces created by the bedside sleeper or with improper use or assembly of the unit.

Cardboard boxes have been distributed as sleep surfaces in Finland since the 1930s, when few households could afford cribs and as an incentive for early prenatal care. This program continues today, primarily because families want to continue to receive the baby products in the box rather than use the box for their infant sleep. Although Finland's SIDS rates are very low, they are equally low in other countries in the region that do not routinely provide boxes.²⁰⁵ One US study evaluated a program

including standardized safe sleep education and provision of a cardboard box distributed to birth families at hospital discharge.²⁰⁶ Of 1429 mothers receiving the box, 47.9% (685) responded to a questionnaire administered within 72 hours after birth hospital discharge. Only 51% of respondents reported using the box as a sleeping space, with 12% using it as the primary infant sleeping space. Bed-sharing rates at 1 week after hospital discharge among those receiving the box, compared with those who did not receive a box, were significantly lower for exclusively breastfed infants (rate ratio: 2.0 [1.01–3.15]).²⁰⁶ It is not clear whether the decrease in bed sharing at 1-week post hospital discharge was attributable to the box or the accompanying safe sleep education, and no studies have assessed use rates in infants older than 1 week of age. Two qualitative studies have also described that mothers have mixed feelings about using a box as an infant sleep surface.^{207,208}

Although boxes are viewed positively for being portable, compact, affordable, and decorative, mothers do not like that the boxes are low to the ground, with inadequate structural integrity or design and stability. Mothers also describe that they might feel social stigma if they used the box for their infant to sleep in. Some international experts have raised safety concerns, including lids on the boxes, hazards with use on a floor, fall risk with use at a height, durability (especially if the box becomes wet or dirty), and outgrowing the box at an age at which risk of sleep-related death is at its peak.²⁰⁹ Cardboard boxes that do not meet the federal safety standard²⁰⁰ are likely not safe for infant sleep and should not be used.

Some American Indian and Alaska Native communities have promoted the use of cradleboards as an infant

sleep surface. There are no data regarding the safety of cradleboards for sleep, but the Eunice Kennedy Shriver National Institutes of Health and Human Development (NICHD)-led Healthy Native Babies Project suggests cradleboards as a culturally appropriate infant sleep surface.²¹⁰ Care should be taken so that infants do not overheat (because of overbundling) in the cradleboard.

Parents and caregivers should adhere to the manufacturer's guidelines regarding maximum weight of infants using alternative products.^{211,212} Regardless of sleep surface, the AAP recommends supine positioning, use of a firm, noninclined sleep surface without padded sides, and avoidance of soft objects and loose bedding.

Sitting devices, such as car seats, strollers, swings, infant carriers, and infant slings, are not recommended for routine sleep in the hospital or at home, particularly for infants younger than 4 months.

Some parents choose to allow their infants to sleep in a car seat or other sitting device. Sitting devices include but are not restricted to car seats, strollers, swings, infant carriers, and infant slings. Parents and caregivers often use these devices, even when not traveling, because they are convenient. One study found that the average young infant spends 5.7 hours per day in a car seat or similar sitting device.²¹³ However, there are multiple concerns about using sitting devices as a usual infant sleep location. Placing an infant in such devices can potentiate GER²¹⁴ and positional plagiocephaly.²¹⁵ Because they still have poor head control and often experience flexion of the head while in a sitting position, infants younger than 4 months in sitting devices may be at increased risk for upper airway obstruction and oxygen desaturation.^{216–220} In 2019, major

manufacturers voluntarily recalled inclined sleepers after a series of deaths were reported to the CPSC, and additional deaths were discovered.²²¹

A retrospective study reviewed deaths involving sitting and carrying devices (car seats, bouncers, swings, strollers, and slings) reported to the CPSC between 2004 and 2008. Of the 47 deaths analyzed, 31 occurred in car seats, 5 occurred in slings, 4 each occurred in swings and bouncers, and 3 occurred in strollers. Fifty-two percent of deaths in car seats were attributed to strangulation from straps; the others were attributed to positional asphyxia.²²² In addition, analyses of CPSC data report injuries from falls when car seats are placed on elevated surfaces,^{223–227} from strangulation on unbuckled or partially buckled car seat straps,²²² and from suffocation when car seats overturn after being placed on a bed, mattress, or couch.²²⁶ A more recent review of National Center for Fatality Review and Prevention data from 2004 to 2014 evaluated 348 (3%) sleep-related deaths occurring in sitting devices.²²⁸ There was at least 1 risk factor (eg, prematurity, tobacco exposure, and sleeping caregiver) in 81.9% of the deaths in sitting devices and at least 2 risk factors in 54.9%. The car seat was used properly in <10% of the cases. Compared with other sleep-related deaths, deaths in sitting devices had higher odds of occurring under the supervision of a child care provider (aOR 2.8; 95% confidence interval [CI], 1.5 to 5.2) or babysitter (aOR 2.0; 95% CI, 1.3 to 3.2) compared with a parent. Therefore, when infants fall asleep in a sitting device, they should be removed from the product and moved to a crib or other appropriate firm, flat surface

as soon as is safe and practical. Car seats and similar products are not stable on a crib mattress or other elevated surface.^{223–227} Infants should not be left unattended in car seats and similar products and should not be placed or left in car seats and similar products with the straps unbuckled or partially buckled.²²² Additionally, parents should give specific instruction to child care or other providers to remove the baby from the car seat as soon as they are dropped off for care.

A recent biomechanics study demonstrated that infants could more easily roll from supine to prone in an inclined sleeper, and once in the prone position, they would fatigue faster than they would on a stable, flat surface because of the high musculoskeletal demands necessary to maintain safe posture to prevent suffocation. The study also found that prone positioning on an inclined (>10 degrees from horizontal) sleep surface places the infant at higher risk of airway obstruction or suffocation, as evidenced by oxygen saturation results.¹⁷⁸ These results may provide a mechanism to some of the deaths related to car seats and other sitting and carrying devices.

There are also reports of suffocation in infants, particularly those who are younger than 4 months, who are carried in infant sling carriers.^{222,229–231} When infant slings are used for carrying, it is important to ensure that the infant's head is up and above the fabric, the face is visible, and the nose and mouth are clear of obstructions. After nursing, reposition the infant in the sling so that the head is up and is clear of fabric and the airway is not obstructed by the adult's body.²²²

FEEDING OF HUMAN MILK

Feeding of human milk is recommended, as it is associated with a reduced risk of SIDS, unless it is contraindicated or the parent is unable to do so, it is recommended that infants be fed with human milk (ie, not offered any formula or other nonhuman milk-based supplements) exclusively for approximately 6 months, with continuation of human milk feeding for 1 year or longer as mutually desired by parent and infant, in alignment with recommendations of the AAP.

The risk-reducing role of human milk feeding on SIDS is enhanced when it is exclusive and without formula introduction.^{232–234} Studies do not distinguish between feeding at the breast and providing expressed human milk. In the Agency for Healthcare Research and Quality's "Evidence Report on Breastfeeding in Developed Countries," 6 studies were included in the SIDS-breastfeeding meta-analysis, and ever having breastfed was associated with a lower risk of SIDS (adjusted summary OR, 0.64; 95% CI, 0.51 to 0.81).²³² Another meta-analysis of 18 case control studies found an unadjusted summary OR for any breastfeeding of 0.40 (95% CI, 0.35 to 0.44) and a pooled adjusted OR of 0.55 (95% CI, 0.44 to 0.69).²³⁴ The protective effect of breastfeeding increased with exclusivity, with an unadjusted summary OR of 0.27 (95% CI, 0.24 to 0.31) for exclusive breastfeeding of any duration.²³⁴ A subsequent meta-analysis using individual level data from 8 case-control studies (2267 SIDS cases and 6837 control infants) found in multivariable pooled analysis that any breastfeeding for under 2 months was not protective (aOR, 0.91; 95% CI, 0.68 to 1.22).²³⁵ However, any breastfeeding for 2 to 4 months, 4 to 6 months, and >6 months was strongly protective (aOR, 0.60 and

95% CI, 0.44 to 0.82; aOR, 0.40 and 95% CI, 0.26 to 0.63; aOR, 0.36 and 95% CI, 0.22 to 0.61, respectively). Results were similar for exclusive breastfeeding for durations of 2 to 4 months and 4 to 6 months.²³⁵ Therefore, breastfeeding of at least 2 months, either exclusive or any, was associated with a decrease in the risk of SIDS by approximately half.

Initiation and duration of human milk feeding are lower in preterm infants compared with term infants.²³⁶ Because preterm and low birth weight infants are at higher risk of dying of SIDS,²³⁷ it is particularly important to emphasize the benefits of human milk, engage with families to understand the barriers and facilitators to provision of human milk, and provide more intensive assistance during prolonged NICU hospitalization for these groups.

Physiologic sleep studies showed that breastfed infants are more easily aroused from sleep than their formula-fed counterparts.^{238,239} In addition, breastfeeding results in a decreased incidence of diarrhea, upper and lower respiratory infections, and other infectious diseases²⁴⁰ that are associated with an increased vulnerability to SIDS and provides overall immune system benefits attributable to maternal antibodies and micronutrients in human milk.^{241,242} Exclusive breastfeeding for 6 months has been found to be more protective against infectious diseases, compared with exclusive breastfeeding to 4 months of age and partial breastfeeding thereafter.²⁴⁰ Furthermore, exclusive breastfeeding results in a gut microbiome that supports a normally functioning immune system and protection from infectious disease, and this commensal microbiome has been proposed as another possible

mechanism or marker for protection against SIDS.²⁴³

Some parents are unable to or choose not to feed human milk. When discussing feeding practices, culturally appropriate, respectful, and nonjudgmental communication between health care professionals and parents is recommended. These families should still be counseled on the importance of following the other safe sleep recommendations.

INFANT SLEEP LOCATION

It is recommended that infants sleep in the parents' room, close to the parents' bed, but on a separate surface designed for infants, ideally for at least the first 6 months.

The terms bed sharing and cosleeping are often used interchangeably, but they are not synonymous. Cosleeping is when parent and infant sleep in close proximity (on the same surface or different surfaces) so as to be able to see, hear, and/or touch each other.^{244,245} Cosleeping arrangements can include bed sharing or sleeping in the same room in close proximity.^{245,246} Bed sharing refers to a specific type of cosleeping when the infant is sleeping on the same surface with another person.²⁴⁵ The shared surface can include a bed, sofa, or chair. Because the term cosleeping can be misconstrued and does not precisely describe sleep arrangements, the AAP recommends use of the terms bed sharing or surface sharing and room sharing (when the infant sleeps in the parents' room but on a separate sleep surface [crib or similar surface] close to the parents' bed) (Table 1).

The AAP recommends room sharing, because this arrangement decreases the risk of SIDS by as much as 50%^{141,143,247–249} and is safer than

bed sharing^{141,143,247,248} or solitary sleeping (when the infant is in a separate room).^{141,247,249} In addition, this arrangement is most likely to prevent suffocation, strangulation, and entrapment that may occur when the infant is sleeping in the adult bed. Furthermore, this arrangement allows close proximity to the infant, which will facilitate feeding, comforting, and monitoring of the infant.

The AAP recommends that the infant's crib, portable crib, play yard, or bassinet be placed in the parents' bedroom, ideally for at least the first 6 months. Room sharing without bed sharing is protective for the first year of life, and there is no specific evidence for when it might be safe to moving an infant to a separate room before 1 year of age. However, the rates of sleep-related deaths are highest in the first 6 months, so room sharing during this vulnerable period is especially important. Placing the crib close to the parents' bed so that the infant is within view and within arms' reach can facilitate feeding, comforting, and monitoring of the infant to give parents peace of mind about their infant's safety. This arrangement reduces SIDS risk and removes the possibility of suffocation, strangulation, and entrapment that may occur when the infant is sleeping in the adult bed.

Parent-infant bed sharing for all or part of sleep duration is common. In 2015 PRAMS data collected in 14 states, 61.4% of mothers reported any bed sharing.¹⁴⁸ Similarly, 2016 PRAMS data collected in 29 states found that only 41.1% of parents reported exclusively room sharing without bed sharing.²⁷ The rate of routine bed sharing is higher among some racial and ethnic groups, including Black, Hispanic, and American Indian and Alaska Native parents and infants.¹⁴⁸ There are

often cultural and personal reasons why parents choose to bed share, including convenience for feeding (human milk or formula), comforting a fussy or sick infant, helping the infant and/or parent sleep better, bonding and attachment, and because it is a family tradition.^{250,251} In addition, many parents may believe that their own vigilance is the only way that they can keep their infant safe and that the close proximity of bed sharing allows them to maintain vigilance, even while sleeping.²⁵² Some parents will use bed sharing specifically as a safety strategy if the infant sleeps in the prone position^{252,253} or there is concern about environmental dangers, such as vermin or stray gunfire.²⁵²

There is an increasing body of research on the effects of room sharing on both infant and parent sleep. Several studies indicate that mothers who room share have increased awakenings^{254,255} and poorer quality of sleep than mothers who sleep in a separate room. In a recent study, Paul looked at differences in infant sleep in early (<4 months) versus later (between 4 and 9 months) independent sleepers (ie, sleeping in a separate room from parents) compared with room sharers and found that at 4 months, early independent sleepers had longer stretches of sleep indicating earlier sleep consolidation, but no increase in total sleep. At 9 months, room-sharing infants were sleeping 14 to 40 minutes less than independent sleepers, but there was no significant difference in night time awakenings. At 12 months, the differences in sleep duration were no longer significant.²⁵⁶ Another study looking at sleep characteristics found that parental presence and room sharing were associated with increased nighttime awakenings, but not total sleep time

at 1 year of age.²⁵⁷ Early sleep consolidation and fewer awakenings may be appealing to tired parents; however, decreased arousals likely contribute to an increased risk for sleep-related death.¹⁶⁷⁻¹⁷¹ Therefore, the AAP continues to recommend room sharing until at least 6 months of age.

Parent-infant bed sharing continues to be highly controversial. Electrophysiologic and behavioral studies offer a strong case for its effect in facilitating breastfeeding,^{258,259} there is some physiologic evidence that bed sharing increases infant calming,²⁶⁰ and many parents believe that they can maintain vigilance of the infant while they are asleep and bed sharing.²⁵²

The effect of bed sharing on childhood attachment and psychological outcomes for children are also now being looked at more closely, with varied results and significant limitations. Some studies indicated that bed sharing in infancy was associated with increased reliance on security objects and sleep aids later on, and small but significant positive effects on cognitive competence in childhood and psychosexual adjustment in adulthood.^{261,262} More recently, a small study found that infants who fully or partially bed share at 3 months had greater self-regulatory behavior at 6 months and that fully bed-sharing infants had less negativity at 6 months.²⁶³ A 2016 study by Mileva-Seitz et al looking specifically at bed sharing at 2 months and secure attachment at 14 months found that solitary sleeping was associated with insecure, and more specifically resistant, attachment.²⁶⁴ However, the study was limited by only asking about bed sharing at a single time point. Additionally, there was no dose-response association, leading to the conclusion that further study was

needed. More recently, a study compared mother-infant dyads who bed shared or did not bed share in the infant's first 6 months of life and found no differences in infant-mother attachment, infant behavior, bonding, or sensitive parenting at 18 months.²⁶⁵ A recent study from Brazil found increased odds of psychiatric diagnoses and internalizing problems at age 6 years among both early-only bed sharers (bed shared until 2 years) and persistent bed sharers (bed shared consistently until 6 years) when compared with solitary sleepers, but there were also sociodemographic differences in the 2 groups.²⁶⁶ In 1 study, cosleeping (defined as room sharing with or without bed sharing) was associated with increased social criticism of mothers' choice of sleep arrangement, maternal depression and concerns about infant sleep.²⁶⁷ The only recent study to look specifically at room sharing without bed sharing found that this sleep arrangement for the first 6 months was not associated with any sleep or behavior problems at ages 6 to 8 years.²⁶⁸ Likely complicating these findings further is the fact that all of these results are expected to be confounded by parental behavior, and 1 recent study demonstrated that parental response was different for bed sharers and solitary sleepers.²⁶⁹

However, epidemiologic studies have shown that bed sharing is associated with a number of conditions, including soft bedding,²⁷⁰⁻²⁷³ head covering,²⁷⁴⁻²⁷⁷ and, for infants of smokers, increased exposure to tobacco smoke,²⁷⁸ which are risk factors for SIDS. In addition, bed sharing itself is associated with an increased risk of SIDS; a meta-analysis of 11 studies investigating the association of bed sharing and SIDS showed a summary OR of 2.88 (95% CI, 1.99

to 4.18) with bed sharing.²⁷⁹ Furthermore, bed sharing in an adult bed not designed for infant safety, especially when associated with other risk factors, exposes the infant to additional risks for unintentional injury and death, such as suffocation, asphyxia, entrapment, falls, and strangulation.^{280,281} Infants younger than 4 months²⁸² and those born preterm and/or with low birth weight²³⁷ are at highest risk, possibly because immature motor skills and muscle strength make it difficult to escape potential threats.²⁷⁹ In recent years, the concern among public health officials about bed sharing has increased, because there have been more reports of infant deaths occurring in high-risk sleep environments, particularly bed sharing and/or sleeping on a couch or armchair.²⁸³⁻²⁸⁵ The Supplemental Table 2 outlines the added risk of common hazards associated with bed sharing. It should be noted that the presence of separate risk factors can lead to a marked increased risk beyond the baseline risk of bed sharing. Given the high rates of bed sharing, these risk factors should be thoughtfully discussed with all parents of neonates and infants, not just those who indicate during health care visits that they are bed sharing.

On the other hand, some breastfeeding advocacy groups encourage bed sharing to promote breastfeeding,²⁸⁶ and debate continues as to the safety of this sleep arrangement for low-risk, breastfed infants. As described in detail in the 2016 AAP technical report, Blair and Carpenter each analyzed data from multiple case-controlled studies regarding the risk of bed sharing and they came to conflicting conclusions about the risk of SIDS in otherwise low-risk infants. Both studies lacked power and given the controversial nature

of this recommendation, the task force requested an independent review of the studies by Dr. Robert Platt, a biostatistician with expertise in perinatal epidemiology from McGill University. Dr. Platt had no connection to the task force nor a vested interest in the recommendations. He concluded that both studies should be interpreted with a degree of caution, but that, “Clearly, these data do not support a definitive conclusion that bed sharing in the youngest age group is safe, even under less hazardous circumstances.”^{1,2} Given this and the absence of additional, more recent data to the contrary, the AAP continues to recommend room sharing without bed sharing and recommends that all families be counseled on the risks of additional hazards that make bed sharing more dangerous.

There is insufficient evidence to recommend for or against the use of devices promoted to make bed sharing “safe.”

There is no evidence that devices marketed to make bed sharing “safe” reduce the risk of SIDS or suffocation or are safe. There are no peer-reviewed published data demonstrating the safety of products designed for in-bed use. Bedside sleepers, which attach to the side of the parental bed and for which the CPSC published standards in 2013,¹⁹⁹ may be considered by some parents as an option. At a minimum, to be considered a safe option, any of these devices should adhere to the June 2021 CPSC rule that any infant sleep product must meet existing federal safety standards for cribs, bassinets, play yards, and bedside sleepers.²⁰⁰ (See section on Sleep Surfaces for further discussion of sleepers.)

Return infants who are brought into the bed for feeding or comforting to

their own crib or bassinet when the parent is ready to return to sleep.

Studies have found an association between bed sharing and longer duration of breastfeeding,^{258,259,287,288} but most of these were cross-sectional studies, which do not enable determination of a temporal relationship—ie, whether bed sharing promotes breastfeeding or whether breastfeeding promotes bed sharing, and whether women who prefer 1 practice are also likely to prefer the other.^{288,289} However, a more recent longitudinal study provides strong evidence that bed sharing promotes breastfeeding duration, with the greatest effect among frequent bed sharers.²⁹⁰ Another recent study has shown that compared with mothers who room shared without bed sharing, mothers who bed shared were more likely to report exclusive breastfeeding (aOR, 2.46; 95% CI, 1.76 to 3.45) or partial breastfeeding (aOR, 1.75; 95% CI, 1.33 to 2.31).²⁹¹ A recent study evaluating sleep location in women with strong breastfeeding outcomes again found that women who bed shared with their infants were more likely to be exclusively breastfeeding at 6 months and had a longer duration of breastfeeding. In addition, the authors found that bed sharing in mothers who continued to breastfeed increased when the infants were 6 to 12 months of age.²⁸⁷ However, although bed sharing may facilitate breastfeeding,²⁵¹ there are other factors, such as intent, that influence successful breastfeeding.²⁹² Furthermore, 1 case-control study found that the risk of SIDS while bed sharing was similar among infants in the first 4 months of life, regardless of breastfeeding status, implying that the benefits of breastfeeding do not outweigh the increased risk associated with bed sharing for younger infants.²⁸² The risk of bed sharing is higher the longer the duration of bed sharing during the

night,¹⁴³ especially when associated with other risks.^{141,142,293,294} Returning the infant to the crib after bringing the infant into the bed for a short period of time is not associated with increased risk.^{142,294} Therefore, after the infant is brought into the bed for feeding, comforting, and bonding, the infant should be returned to the crib when the parent is ready for sleep.

Couches and armchairs are extremely dangerous places for infants and should never be used for infant sleep.

Sleeping on couches and armchairs places infants at extraordinarily high risk (with 22- to 67-fold increased risk) for infant death, including SIDS,^{139,141,142,248,294,295} suffocation through entrapment or wedging between seat cushions, or overlay if another person is also sharing this surface.²⁸⁴ Therefore, parents and other caregivers need to be especially vigilant as to their wakefulness when feeding infants or lying with infants on these surfaces. It is important to emphasize this point to those who are breastfeeding, as 25% of mothers in 1 study reported falling asleep during the night when breastfeeding their infant on 1 of these surfaces.²⁹⁶ Infants should never be placed on a couch or armchair for sleep.

The safest place for a baby to sleep is on a separate sleep surface designed for infants close to the parents’ bed.

Infants sleeping in a separate room are 2.75 to 11.5 times more likely to die suddenly and unexpectedly than infants who are room sharing without bed sharing.^{141,247,249} When all bed-sharing or surface-sharing circumstances are included in meta-analyses, the risk of dying suddenly and unexpectedly is almost 3 times higher than room sharing without bed sharing.²⁷⁹

The AAP understands and respects that many parents choose to routinely bed share for a variety of reasons, including facilitation of breastfeeding, cultural preferences, and belief that it is better and safer for their infant. However, on the basis of the evidence,²⁹⁷ the AAP is unable to recommend bed sharing under any circumstances. Having the infant close by their bedside in a crib or bassinet will allow parents to feed, comfort, and respond to their infant's needs. It is also important for parents, pediatricians, other physicians, and nonphysician clinicians to know that the following factors increase the magnitude of risk when bed sharing or surface sharing:

More than 10 times the baseline risk of parent-infant bed sharing:

- Bed sharing with someone who is impaired in their alertness or ability to arouse because of fatigue or use of sedating medications (eg, certain antidepressants, pain medications) or substances (eg, alcohol, illicit drugs).^{143,283,295,297}
- Bed sharing with a current smoker (even if the smoker does not smoke in bed) or if the pregnant parent smoked during pregnancy.^{141,142,279,293,298}
- Bed sharing on a soft surface, such as a waterbed, old mattress, sofa, couch, or armchair.^{139,141,142,248,294}

5 to 10 times the baseline risk of parent-infant bed sharing:

- Term, normal-weight infant younger than 4 months, even if neither parent smokes and even if the infant is breastfed.^{141,143,248,279,294,297,299} This is a particularly vulnerable time, so parents who choose to feed their infants younger than 4

months in bed need to be especially vigilant to avoid falling asleep.

- Bed sharing with anyone who is not the infant's parent, including nonparental caregivers and other children.¹³⁹

2 to 5 times the baseline risk of parent-infant bed sharing:

- Preterm or low birth weight infant, even if neither parent smokes.²³⁷
- Bed sharing with soft bedding accessories, such as pillows or blankets.^{139,300}

Pediatricians, other physicians, and nonphysician clinicians are encouraged to counsel all families on these factors that can substantially increase the risk of sleep-related death while bed sharing.

A retrospective series of SIDS cases reported that mean maternal body weight was higher for bed-sharing mothers than for nonbed-sharing mothers.³⁰¹ The only case-control study to investigate the relationship between maternal body weight and bed sharing did not find an increased risk of bed sharing with increased maternal weight.³⁰²

Guidance for parents who fall asleep while feeding the infant

Bed sharing can occur unintentionally if parents fall asleep while feeding their infant or at times when parents are particularly tired, or infants are fussy. Evidence suggests that it is relatively less hazardous (but still not recommended) to fall asleep with the infant in the adult bed than on a sofa or armchair, should the parent fall asleep. It is important to note that a large percentage of infants who die of SIDS are found with their head covered by bedding.²⁷⁴ Therefore, it is advised that no pillows, sheets, blankets, pets, or

any soft or loose items that could obstruct infant breathing^{139,270} or cause overheating be in the bed.^{303–306} Parents should follow safe sleep recommendations outlined elsewhere in this statement. Because there is evidence that the risk of bed sharing is higher with longer duration, if the parent falls asleep while feeding the infant in bed, the parent is advised to return the infant to a separate sleep surface as soon as the parent awakens.^{141,142,293,294}

Any potential benefits of cobedding twins and higher-order multiples are outweighed by the risk of cobedding. It is prudent to provide separate sleep areas and avoid cobedding (sleeping on the same sleep surface) for twins and higher-order multiples in the hospital and at home.

Cobedding of twins and other infants of multiple gestation is a frequent practice, both in the hospital setting and at home.³⁰⁷ However, the benefits of cobedding twins and higher-order multiples have not been established.^{308–310} Twins and higher-order multiples are often born preterm and with low birth weights, so they are at increased risk for SIDS.^{46,179} Furthermore, cobedding increases the potential for overheating and rebreathing, and size discordance between multiples may increase the risk of unintentional suffocation.³⁰⁹ Most cobedded twins are placed on the side rather than supine.³⁰⁷ Finally, cobedding of twins and higher-order multiples in the hospital setting may encourage parents to continue this practice at home.³⁰⁹ Because the evidence for the benefits of cobedding twins and higher-order multiples is not compelling and because of the increased risk of SIDS and suffocation, the AAP believes that it is prudent to provide separate sleep areas for these infants to decrease

the risk of SIDS and unintentional suffocation.

USE OF BEDDING

Keep soft objects, such as pillows, pillow-like toys, quilts, comforters, mattress toppers, fur-like materials, and loose bedding, such as blankets and nonfitted sheets, away from the infant's sleep area to reduce the risk of SIDS, suffocation, entrapment or wedging, and strangulation.

Soft objects, such as pillows and pillow-like toys, quilts, comforters, fur-like materials, and loose bedding, such as blankets and nonfitted sheets, can obstruct an infant's airway and increase risk for SIDS,^{139,270} suffocation, and rebreathing.^{131,133,134,193,311–313} In the United States, more than 40% of infants are placed to sleep underneath or on top of bedding such as thick blankets, quilts, and pillows.^{27,314} The prevalence of bedding use is highest among infants whose mothers are teenagers, from minority racial and ethnic groups, and among those without a 4-year college degree.²⁷

Pillows, quilts, comforters, fur-like materials, and other soft bedding can be hazardous when placed under the infant^{37,139,270,305,315–320} or left loose in the infant's sleep area.^{37,142,270,300,313,318–325} Bedding in the sleeping environment increases SIDS risk fivefold independent of sleep position,^{139,270} and this risk increases to 21-fold when the infant is placed prone.^{139,270} Many infants who die of SIDS are found in the supine position but with their heads covered by loose bedding.^{142,315,316,321} Additionally, infants who bed share have a higher SIDS risk when sleeping on a soft as opposed to firm surface.³⁰⁰

In addition to SIDS risk, soft objects and loose bedding in the sleeping

environment may lead to unintentional suffocation.^{192,313,326} Airway obstruction from soft objects or loose bedding is the most common way accidental infant suffocation occurs.³⁷ A review of 66 SUID case investigations in 2011 showed that soft bedding was the most frequently reported factor among deaths classified as possible and explained unintentional suffocation deaths.³¹³ In addition, a CPSC report of sleep-related infant deaths from 2009 to 2011 found that most deaths attributed to suffocation (regardless of whether the infant was sleeping in a crib, on a mattress, or in a play yard) involved extra bedding, such as pillows or blankets.³²⁶ A more recent report found that among 250 accidental suffocations during 2011 to 2014, 69% were attributed to soft bedding occluding the infant's airway.³⁷ Soft bedding (eg, blankets and stuffed animals) may also be a stronger risk factor for sleep-related deaths among infants older than 3 months than it is for their younger counterparts, especially when infants are placed in or roll to the prone position.^{37,192} Another study restricted to accidental infant suffocations, found younger infants (≤ 4 months) were more often suffocated by soft bedding or overlay than older infants (5–11 months). Among suffocations attributed to soft bedding, older infants (5–11 months) were more likely to have their airways obstructed by blankets (as opposed to pillows or cushions).³⁷

It is recommended that weighted blankets, weighted sleepers, or other weights not be placed on or near the sleeping infant. A single crossover randomized nonblinded trial of 16 infants with neonatal abstinence syndrome found no adverse events when a 1-pound weighted blanket was placed on each infant for 30 minute observed

episodes.³²⁷ However, no studies have documented the safety of weights for infants in an unobserved, nonclinical sleep environment.

Parents and caregivers are likely motivated by good intentions and perceived cultural norms when they opt to use bedding for infant sleep. Qualitative studies show that parents who use bedding want to provide a comfortable and safe environment for their infant.^{328,329} For comfort, parents may use blankets to provide warmth or to soften the sleep surface. For safety, parents may use pillows as barriers to prevent falls from adult beds or sofas or as a prop to keep their infant on the side.^{328,329} Images of babies sleeping with blankets, pillows, and other soft objects are widespread in popular magazines targeted to families with newborn infants.^{330,331} Parents and caregivers who see these images may perceive the use of these items as the norm, both favorable and the ideal, for infant sleep.

Dressing the infant with layers of clothing is preferable to blankets and other coverings to keep the infant warm while reducing the possibility of head covering or entrapment that could result from blanket use. However, care must be taken to select appropriately sized sleep clothing and to avoid overheating. Wearable blankets can also be used. Nursing and hospital staff should model safe sleep arrangements to new parents after delivery.

Bumper pads or similar products that attach to crib slats or sides are not recommended, because they have been implicated in deaths attributable to suffocation, entrapment or wedging, and strangulation. With current safety standards for crib slats, bumper pads and similar products are not

necessary for safety against head entrapment or to prevent other injury.

Bumper pads and similar products attaching to crib slats or sides are frequently used with the thought of protecting infants from injury. Bumper pads were originally developed to prevent head entrapment between crib slats.³³² However, newer crib standards requiring crib slat spacing to be less than 2 3/8 inches have obviated the need for crib bumpers. In addition, infant deaths have occurred because of bumper pads. A case series by Thach using 1985 to 2005 CPSC data found that deaths attributed to bumper pads occurred as a result of 3 mechanisms: (1) suffocation against soft, pillow-like bumper pads; (2) entrapment between the mattress or crib and firm bumper pads; and (3) strangulation from bumper pad ties.³³³ However, a 2010 CPSC white paper that reviewed the same cases concluded that there were other confounding factors, such as the presence of pillows and/or blankets, that may have contributed to many of the deaths in this report.³³⁴ The white paper pointed out that available data from the scene investigations, autopsies, law enforcement records, and death certificates often lacked sufficiently detailed information to conclude how or whether bumper pads contributed to deaths. Two more recent analyses of CPSC data have also come to different conclusions. The CPSC review concluded again that there was insufficient evidence to support that bumper pads were primarily responsible for infant deaths when bumper pads were used per manufacturer instructions and in the absence of other unsafe sleep risk factors.³³⁵ Scheers et al, in their reanalysis,³³⁶ concluded that the rate of bumper pad-related deaths has increased, recognizing that

changes in reporting may account for the increase, and that 67% of the deaths could have been prevented if the bumper pads had not been present. Limitations of CPSC data collection processes contribute to the difficulty in determining the risk of bumper pad use.

However, other investigators^{333,337} have concluded that use of bumper pads only prevents minor injuries and that the potential benefits of preventing minor injury with bumper pad use are far outweighed by the risk of serious injury, such as suffocation or strangulation. Additionally, most bumper pads obscure infant and parent visibility, which may increase parental anxiety.^{328,332} Other products exist that attach to crib sides or crib slats and claim to protect infants from injury; however, there are no published data that support these claims.

Because of the potential for suffocation, entrapment, and strangulation and lack of evidence to support that bumper pads or similar products that attach to crib slats or sides prevent injury in young infants, the AAP does not recommend their use.

PACIFIER USE

Offering a pacifier at nap time and bedtime is recommended to reduce the risk of SIDS.

Multiple case-control studies^{139,143,294,338–344} and 2 meta-analyses^{345,346} have reported a protective effect of pacifiers on the incidence of SIDS, with decreased risk of SIDS ranging from 50% to 90%. Further, 1 study found that pacifier use favorably modified the risk profile of infants who sleep in the prone or side position, bed share, or use soft bedding.³⁴⁷ The mechanism for this apparent strong protective effect is still unclear, but

favorable modification of autonomic control during sleep in term and preterm infants^{348–350} and maintaining airway patency during sleep³⁵¹ have been proposed. Physiologic studies of the effect of pacifier use on arousal are conflicting; 1 study found that pacifier use decreased arousal thresholds,²³⁸ but others have found no effects on arousability with pacifier use.^{352,353} It is common for the pacifier to fall from the mouth soon after the infant falls asleep; even so, the protective effect persists throughout that sleep period.^{238,354} Two studies have shown that pacifier use is most protective when used for all sleep periods.^{294,344} However, these studies also showed increased risk of SIDS when the pacifier was habitually used but not during the last time the infant was placed for sleep; the significance of these findings is yet unclear.

The pacifier can be offered when the infant is placed for naps or nighttime sleep. It does not need to be reinserted once the infant falls asleep. Infants who refuse the pacifier should not be forced to take it. In those cases, parents can try to offer the pacifier again when the infant is a little older.

The AAP policy statement “Breastfeeding and the Use of Human Milk” includes a recommendation that pacifiers can be used during breastfeeding but that introduction should be delayed until breastfeeding is well established.³⁵⁵ This is defined as having sufficient maternal milk supply; consistent, comfortable, and effective latch for milk transfer; and appropriate infant weight gain as defined by established normative growth curves.³⁵⁶ The time required to establish breastfeeding is variable. Infants who are not being directly breastfed can begin pacifier use as soon as desired.

Although some SIDS experts and policy makers have endorsed pacifier use recommendations that are similar to those of the AAP,^{357,358} concerns about possible deleterious effects have prevented others from making a recommendation for pacifier use as a risk reduction strategy.³⁵⁹

Although several observational studies^{360–362} have shown a correlation between pacifiers and reduced breastfeeding duration, a recent Cochrane review comparing pacifier use and nonuse in healthy term infants who had initiated breastfeeding found that pacifier use had no effects on partial or exclusive breastfeeding rates at 3 and 4 months.³⁶³ One randomized controlled trial found that among preterm infants pacifiers supported an accelerated transition from complementary feeding to exclusive breastfeeding.³⁶⁴ Furthermore, 2 systematic reviews found that the highest level of evidence (ie, from randomized controlled clinical trials) does not support an adverse relationship between pacifier use and breastfeeding duration or exclusivity.^{365,366} The association between shortened duration of breastfeeding and pacifier use in observational studies likely reflects a number of complex factors, such as breastfeeding difficulties or intent to wean.^{365,367} However, some have also raised the concern that studies that demonstrate no effect of pacifier introduction on breastfeeding duration or exclusivity may not account for early weaning or failure to establish breastfeeding.^{368,369}

Some dental malocclusions have been found more commonly among pacifier users than nonusers, but the differences generally disappeared after pacifier cessation.³⁷⁰ A policy statement from the American Academy of Pediatric Dentistry on oral habits states that nonnutritive

sucking behaviors (ie, fingers or pacifiers) are considered normal in infants and young children and that, in general, sucking habits in children to the age of 3 years are unlikely to cause any long-term problems.³⁷¹ Pacifier use is associated with an approximate 1.2- to two-fold increased risk of otitis media, particularly between 2 and 3 years of age.^{372,373} The incidence of otitis media is generally lower in the first year after birth, especially the first 6 months, when the risk of sleep-related death is the highest.^{374–379} However, pacifier use, once established, may persist beyond 6 months, thus increasing the risk of otitis media. Gastrointestinal tract infections and oral colonization with *Candida* species were also found to be more common among pacifier users than nonusers.^{375–377}

Because of the risk of strangulation,³⁸⁰ a pacifier should never be hung around the infant's neck or attached to infant clothing when the infant is placed for sleep or sleeping. Objects such as blankets, plush or stuffed toys, and other items that may present a suffocation or choking risk should never be attached to pacifiers.

There is insufficient evidence that finger sucking is protective against SIDS.

The literature on infant finger sucking and SIDS is extremely limited. Only 2 case-control studies have reported these results.^{342,343} One study from the United States showed a protective effect of infant finger sucking (reported as “thumb sucking”) against SIDS (aOR, 0.43; 95% CI, 0.25 to 0.77), but it was less protective than pacifier use if the infant also sucked the thumb (aOR, 0.07; 95% CI, 0.01 to 0.64), or if the infant did not suck the thumb and just used the pacifier (aOR, 0.08; 95% CI, 0.03 to 0.23).³⁴³ Another study from the Netherlands did not

demonstrate an association between usual finger sucking (reported as “thumb sucking”) and SIDS risk (OR, 1.38; 95% CI, 0.35 to 1.51), but the wide confidence interval suggests that there was insufficient power to detect a significant association.³⁴²

PRENATAL AND POSTNATAL EXPOSURES (INCLUDING SMOKING AND USE OF ALCOHOL, OPIOIDS, AND MARIJUANA)

It is recommended that pregnant people obtain regular prenatal care.

There is substantial epidemiologic evidence linking a lower risk of SIDS for infants when there has been regular prenatal care.^{194,381–383} However, limited prenatal care often results from social determinants of health that are also associated with increased risk of SIDS. Pregnant people are advised to follow guidelines for frequency of prenatal visits.³⁸⁴ Prenatal care provides the opportunity for physicians and nonphysician clinicians to counsel future parents on safe sleep practices and to manage high risk behaviors, such as smoking. However, in 1 study, more than half of obstetricians reported spending only 1 to 4 minutes discussing smoking cessation and more than half stated that competing priorities, lack of time, patient resistance, and lack of training and communication resources were significant barriers to smoking cessation treatment.³⁸⁵ A history of limited receipt of prenatal care may alert pediatricians, other physicians, and nonphysician clinicians that additional attention to and education regarding modifiable risk factors for sleep-related infant death may be needed.

Avoid smoke and nicotine exposure during pregnancy and after birth.

Maternal smoking during pregnancy has been identified as a major risk factor in almost every epidemiologic study of SIDS.^{386–389} Smoke in the

infant's environment after birth has been identified as a separate major risk factor in a few studies,^{387,390} although separating this variable from maternal smoking before birth is problematic. Third-hand smoke refers to residual contamination from tobacco smoke after the cigarette has been extinguished³⁹¹; there is no research to date on the significance of third-hand smoke with regard to SIDS risk. Smoke exposure adversely affects infant arousal^{392–398}; in addition, smoke exposure increases risk for preterm birth and low birth weight, both risk factors for these deaths. The effect of tobacco smoke exposure is dose dependent. The risk for a sudden unexpected infant death doubles with even 1 cigarette per day (aOR, 1.98; 95% CI, 1.73 to 2.28).³⁹⁹ The adjusted odds increase by 0.07 for every additional cigarette per day up to 20 cigarettes per day (aOR, 0.07 × cigarettes per day + 1.91).³⁹⁹ The risk of a sleep-related death is particularly high when the infant bed shares with an adult smoker (OR, 2.3 to 32.8), even when the adult does not smoke in bed.^{141,142,279,293,295,297,298,400} It is estimated that one third of these deaths could be prevented if all smoking by pregnant people was eliminated.^{401,402}

The AAP supports the elimination of all tobacco smoke exposure, both prenatally and environmentally. Thus, pregnant parents are advised not to smoke during pregnancy or after the infant's birth.^{194,381–383} It is also advised that no one smoke near pregnant people or infants. Although there is no evidence on the relationship of vaping or electronic cigarette use and sleep-related deaths, electronic cigarettes contain nicotine, which has been implicated in these deaths. Encourage families to set strict rules for smoke-free homes and cars and to eliminate secondhand tobacco smoke from all places

where children and other nonsmokers spend time.⁴⁰³

Avoid alcohol, marijuana, opioids, and illicit drug use during pregnancy and after birth.

Several studies have specifically investigated the association of SIDS with prenatal and postnatal exposure to alcohol, marijuana, opioids, or illicit drug use, although substance abuse often involves more than 1 substance, and it is often difficult to separate out these variables from each other and from smoking. A retrospective study from western Australia found that a maternal alcoholism diagnosis recorded during pregnancy (adjusted hazard ratio, 6.92; 95% CI, 4.02 to 11.90) or within 1 year postpregnancy (adjusted hazard ratio, 8.61; 95% CI, 5.04 to 14.69) was associated with increased SIDS risk, and the authors estimated that at least 16.41% of SIDS deaths were attributable to maternal alcohol use disorder.⁴⁰⁴ Another study from Denmark, based on prospective data about maternal alcohol use, has also shown a significant relationship between maternal binge drinking and postneonatal infant mortality, including SIDS.⁴⁰⁵

The concomitant use of alcohol and smoking after the first trimester may pose an especially high risk. A multicenter prospective study of approximately 11 500 infants followed until their first birthday found that infants of mothers who drank alcohol and smoked beyond the first trimester had approximately 12 times higher relative risk of SIDS (adjusted relative risk 11.8; 95% CI, 2.6 to 53.7), and smoking alone (without alcohol use) after the first trimester had an elevated, but low relative risk (adjusted relative risk, 4.9; 95% CI, 0.97 to 24.3).⁴⁰⁶ Another study found that periconceptional maternal alcohol use (aOR, 6.2; 95%

CI, 1.6 to 23.3) and maternal first-trimester binge drinking (aOR, 8.2; 95% CI, 1.9 to 35.3) were associated with increased SIDS risk, independent of prenatal cigarette smoking exposure.³⁰⁶

Parental alcohol and/or illicit drug use in combination with bed sharing places the infant at particularly high risk for SIDS and unintentional suffocation.^{143,283}

Rat models have demonstrated increased arousal latency to hypoxia in rat pups exposed to prenatal alcohol.⁴⁰⁷ Further, 1 postmortem study demonstrated that prenatal cigarette smoking was significantly associated with decreased serotonin receptor binding in the brainstem. In this study, the association of maternal alcohol drinking in the 3 months before or during pregnancy was of borderline significance in univariate analysis but was not significant when prenatal smoking and case versus control status was in the model.⁴⁷ However, this study had limited power for multivariate analysis because of small sample size. One study found an association of SIDS with heavy maternal alcohol consumption in the 2 days before the death.⁴⁰⁸ Several studies have found a particularly strong association when alcohol consumption or illicit drug use occurs in combination with bed sharing.^{141–143,409}

Studies investigating the relationship of marijuana or other substance use and SIDS have focused on specific drugs or illicit substance use in general. One study found maternal cannabis use to be associated with an increased risk of SIDS (aOR, 2.35; 95% CI, 1.36 to 4.05) at night but not during the day.⁴¹⁰ In utero exposure to opioids (primarily methadone and heroin) has been shown in retrospective studies to be associated with an increased risk of SIDS.^{411,412} With

the exception of 1 study that did not show increased risk,⁴¹³ population-based studies have generally shown an increased risk with in utero cocaine exposure.^{414–416} However, these studies did not control for confounding factors. A prospective cohort study found the SIDS rate to be significantly increased for infants exposed in utero to methadone (OR, 3.6; 95% CI, 2.5 to 5.1), heroin (OR, 2.3; 95% CI, 1.3 to 4.0), methadone and heroin (OR, 3.2; 95% CI, 1.2 to 8.6), and cocaine (OR, 1.6; 95% CI, 1.2 to 2.2), even after controlling for race and ethnicity, maternal age, parity, birth weight, year of birth, and maternal smoking.⁴¹⁷ In addition, a meta-analysis of studies investigating an association between in utero cocaine exposure and SIDS found an increased risk of SIDS to be associated with prenatal exposure to cocaine and illicit substances in general.⁴¹⁸

OVERHEATING, FANS, AND ROOM VENTILATION

Avoid overheating and head covering in infants.

Excessive clothing or blankets covering an infant and the room temperature are associated with an increased SIDS risk.^{303–306} Infants who sleep in the prone position also have a higher risk of overheating than supine sleeping infants.³⁰⁵ However, the definition of overheating in the studies finding an increased risk of SIDS varies. Therefore, it is difficult to provide specific room temperature guidelines for avoiding overheating. The AAP recommends that parents and caregivers consider the ambient temperature when dressing or bundling the infant. In general, dress infants appropriately for the environment, with no greater than one layer more than an adult would wear to be comfortable in that environment. Evaluate the

infant for signs of overheating, such as sweating, flushed skin, or the infant's chest feeling hot to the touch.

Avoid overbundling and covering of the face and head.²⁷⁴ Given the questionable benefit of hat use for the prevention of hypothermia⁴¹⁹ and the risk of overheating, it is advised not to place hats on infants when indoors.

With concerns of climate change and the increasing incidence of extreme weather, a number of studies have explored the possible relationship between meteorologic temperature, heat stress, and SIDS.^{420–427} Several older studies found an association between colder temperatures and increased SIDS risk.^{423,424,428}

However the seasonal variation of SIDS has diminished significantly over time.²⁶ Recent studies of the association between meteorologic temperature and SIDS have demonstrated inconsistent results. A Canadian (Montreal) case-crossover study found that compared with a temperature of 20°C (68°F), maximum daily temperatures of >29°C (84.2°F) on the day of death was associated with an almost threefold increase in the odds of SIDS (OR, 2.78; 95% CI, 1.64 to 4.70).⁴²⁵ The odds of SIDS increased with higher temperature and the association was stronger for infants 3 to 12 months of age compared with those 1 to 2 months of age. However, a study of vital statistics records from SIDS cases in Vienna, Austria, was unable to replicate the results of the Canadian study.⁴²⁰ Using the same statistical approach and a similar population to that of the Montreal study, the investigators found no relationship between temperature elevation and increased SIDS risk.

A case-crossover study of 210 US cities found a 5.6°C (10°F) higher daily temperature was associated

with an increased SIDS risk of 8.6% (95% CI, 3.6% to 13.8%) in the summer, compared with a 3.1% decrease (95% CI, –5.0% to –1.3%) in the winter.⁴²⁶ During the summer, the excess risk was greater among Black infants (18.5%; 95% CI, 9.3% to 28.5%) than White infants (3.6%; 95% CI, –2.3% to 9.9%), and among infants 3 to 11 months of age (16.9%; 95% CI, 8.9% to 25.5%) than infants 0 to 2 months of age (2.7%, 95% CI –3.5% to 9.2%). The temperature-SIDS association was stronger in the Midwest and surrounding northern regions. A separate study in California focusing on the warm season found increased all cause infant mortality risk of 4.4% but no increase in risk of SIDS.⁴²¹

Rather than examining environmental temperature elevation as an acute event, Korean researchers found an association between cumulative temperature elevation over 2 weeks and 1 month before death.⁴²⁷ For every temperature increase of 1°C 1 month before death, the hazard ratio for all-cause infant mortality was 1.52 (95% CI, 1.46 to 1.57) and 1.50 (95% CI, 1.35 to 1.66) for SIDS.

These environmental studies have significant limitations, including reliance on ecological data rather than on individual monitoring to assign exposure, lack of data on infant clothing and air conditioning at the time of death, infant activity patterns, amount of time spent indoors versus outdoors, socioeconomic status, and other individual potential confounders.

It is unclear whether the relationship to overheating is an independent factor or merely a reflection of the increased risk of SIDS and suffocation with blankets and other potentially asphyxiating objects in the sleeping environment. Head covering during sleep is of

particular concern. In 1 systematic review, the pooled mean prevalence of head covering among SIDS victims was 24.6%, compared with 3.2% among control infants.²⁷⁴ Although head covering usually refers to bedding or bed clothes, 1 study found significantly more SIDS cases in infants wearing hats compared with controls.³²¹ It is not known whether the risk related to head covering is attributable to overheating, hypoxia, or rebreathing. A study on the aerodynamics of rebreathing exhaled gases demonstrated that with higher temperature and humidity, the exhaled gas is denser and does not escape the vicinity of the nostrils.⁴²⁹ In this in vitro model, the result was increased rebreathing of CO₂-rich gas, suggesting that both overheating and rebreathing are important components in the association between head covering and SIDS.

Some have suggested that room ventilation may be important. One study has found that bedroom heating, compared with no bedroom heating, increases SIDS risk (OR, 4.5),⁴³⁰ and another study has also demonstrated a decreased risk of SIDS in a well-ventilated bedroom (windows and doors open) (OR, 0.4).⁴³¹ In 1 study, the use of a fan appeared to reduce the risk of SIDS (aOR, 0.28; 95% CI, 0.10 to 0.77).⁴³² However, because of the possibility of recall bias, the small sample size of controls using fans ($n = 36$), a lack of detail about the location and types of fans used, and the weak link to a mechanism, this study should be interpreted with caution. Based on available data, the AAP cannot make a recommendation on the use of a fan as a SIDS risk-reduction strategy.

IMMUNIZATIONS

It is recommended that infants be immunized in accordance with guidelines from the AAP and CDC.

The incidence of sleep-related death peaks at a time when infants are receiving numerous immunizations. Case reports of a cluster of deaths shortly after immunization with diphtheria-tetanus-pertussis (DTP) vaccine in the late 1970s created concern of a possible causal relationship between vaccinations and SIDS.^{433–436} Case-control studies were performed to evaluate this temporal association. Four of the 6 studies showed no relationship between DTP vaccination and subsequent SIDS^{437–440}; the other 2 suggested a temporal relationship, but only in specific subgroup analysis.^{441,442} In 2003, the Institute of Medicine reviewed available data and concluded: “The evidence favors rejection of a causal relationship between exposure to multiple vaccinations and SIDS.”⁴⁴³ Multiple analyses of the US Vaccine Adverse Event Reporting System (VAERS) database have demonstrated no relationship between vaccines and SIDS.^{444–447} Additionally, several large population case-control trials consistently have found vaccines to be protective against SIDS,^{448–451} although this protective effect may have been attributable to confounding factors (social, maternal, birth, and infant medical history).⁴⁵² It also has been theorized that the decreased SIDS rate immediately after vaccination was attributable to infants being healthier at the time of immunization (“healthy vaccinee effect”).⁴⁵³ Recent illness would both place infants at higher risk for SIDS and make them more likely to have immunizations deferred.⁴⁵³

More recent studies have attempted to control for confounding by social, maternal, birth, and infant medical history.^{448,450,454} A meta-analysis of 4 studies found a multivariate summary odds ratio for immunizations and SIDS to be 0.54 (95% CI, 0.39 to 0.76), indicating

that the risk of SIDS is halved by immunization.⁴⁵⁴ The evidence continues to show no causal relationship between immunizations and SIDS and suggests that vaccination may have a protective effect against SIDS.

COMMERCIAL DEVICES

Avoid the use of commercial devices that are inconsistent with safe sleep recommendations.

Risk-reduction strategies are based on the best available evidence in large epidemiologic studies. Thus, claims that sleep devices, mattresses, or special sleep surfaces reduce the risk of SIDS must, therefore, be supported by epidemiologic evidence. At a minimum, any devices used should meet safety standards of the CPSC, the Juvenile Product Manufacturers Association, and the ASTM.

The AAP recommends that parents and caregivers be particularly wary of devices that claim to reduce the risk of SIDS or other sleep-related deaths. There is no evidence that any of these devices reduce the risk of these deaths. Importantly, the use of products claiming to increase sleep safety may provide a false sense of security and complacency for caregivers. It is important to understand that use of such products does not diminish the importance of following recommended safe sleep practices. The AAP concurs with the US Food and Drug Administration (FDA) and CPSC that manufacturers should not claim that a product or device protects against sleep-related deaths unless there is scientific evidence to that effect.

Wedges and positioning devices are often used by parents to maintain the infant in the side or supine position because of claims that these products reduce the risk for SIDS,

suffocation, or gastroesophageal reflux. However, these products are frequently made with soft, compressible materials, which might increase the risk of suffocation. The CPSC has received reports of deaths attributable to suffocation and entrapment associated with wedges and positioning devices. Most of these deaths occurred when infants were placed in the prone or side position with these devices⁴⁵⁵; other incidents have occurred when infants have slipped out of the restraints or rolled into a prone position while using the device.^{334,456} Because of the lack of evidence that they are effective against SIDS, suffocation, or gastroesophageal reflux and because of potential for suffocation and entrapment risk, the AAP concurs with the CPSC and the FDA in warning against the use of these products. If positioning devices are used in the hospital as part of physical therapy, they should be removed from the infant sleep area well before discharge from the hospital.

Certain crib mattresses have been designed with air-permeable materials to reduce rebreathing of expired gases, in the event that an infant ends up in the prone position during sleep, and these may be preferable to those with air-impermeable materials. Using a head box model, Bar-Yishay et al found that a permeable sleeping surface exhibited significantly better aeration properties in dispersing CO₂ and in preventing its accumulation.⁴⁵⁷ They also found the measured temperature within the head box to be substantially lower with the more permeable mattress, concluding that it was due to faster heat dissipation. This could be potentially protective against overheating, which has been identified as a risk factor for SIDS. Colditz and colleagues also

performed studies both in vitro and in vivo, demonstrating better diffusion and less accumulation of CO₂ with a mesh mattress.⁴⁵⁸

However, Carolan et al found that even porous surfaces are associated with CO₂ accumulation and rebreathing thresholds, unless there is an active CO₂ dispersal system.⁴⁵⁹ In addition, although rebreathing has been hypothesized to contribute to death in SIDS, particularly if the head is covered or when the infant is face down, there is no evidence that rebreathing, per se, causes SIDS and no epidemiologic evidence that these mattresses reduce the risk of SIDS. The use of “breathable” mattresses can be an acceptable alternative as long as they meet CPSC safety standards.

HOME MONITORS, SIDS, AND BRIEF RESOLVED UNEXPLAINED EVENTS

Do not use home cardiorespiratory monitors as a strategy to reduce the risk of SIDS.

For many years, it was believed that brief resolved unexplained events (formerly known as apparent life-threatening events) were the predecessors of SIDS, and home apnea monitors were used as a strategy for preventing SIDS.⁴⁶⁰ However, use of home cardiorespiratory monitors has not been documented to decrease the incidence of SIDS.^{461–464} Home cardiorespiratory monitors are sometimes prescribed for use at home to detect apnea, bradycardia, and when pulse oximetry is used, decreases in oxyhemoglobin saturation for selected NICU patients with “unusually prolonged course of recurrent” cardiorespiratory events.⁴⁶⁵ Current evidence suggests that if such monitoring is elected, it can be discontinued in most infants after 43 weeks’ postmenstrual age unless indicated by other significant medical conditions.⁴⁶⁶ Routine in-hospital cardiorespiratory

monitoring before discharge from the hospital has not been shown to detect infants at risk for SIDS.

Direct-to-consumer heart rate and pulse oximetry monitoring devices, including wearable monitors, are sold as consumer wellness devices. A consumer wellness device is defined by the FDA as one intended “for maintaining or encouraging a healthy lifestyle and is unrelated to the diagnosis, cure, mitigation, prevention, or treatment of a disease or condition.”⁴⁶⁷ Thus, these devices are not required to meet the same regulatory requirements as medical devices and, by the nature of their FDA designation, are not to be used to prevent sleep-related deaths. One study found that, using a direct-to-consumer device, tachyarrhythmias were detected among 2.5% of the infants during home monitoring.⁴⁶⁸ However, as stated by the authors, this finding was not confirmed by electrocardiography and may represent subclinical events, the significance of which remains unclear.

With regard to the prevention of sleep-related death specifically, although use of these direct-to-consumer monitors may give parents “peace of mind,” reduced anxiety, and better sleep,⁴⁶⁹ and there is no contraindication to using these monitors, data are lacking to support their use to reduce the risk of these deaths. Furthermore, these direct-to-consumer monitors may not be as reliable or accurate in identifying significant events when compared with medical monitors.⁴⁷⁰ There is also concern that use of these monitors will lead to parent complacency and decreased adherence to safe sleep guidelines. Therefore, the AAP does not recommend using video or direct-to-consumer pulse oximetry monitors as a strategy to reduce the risk of a sleep-related death. A family’s

decision to use monitors at home should not be considered a substitute for following AAP safe sleep guidelines. The AAP recognizes, however, that technology is continually changing and improving. It is possible that in the future, direct-to-consumer monitors are reliable and affordable and may help to prevent some sudden deaths.

TUMMY TIME

Supervised, awake tummy time is recommended to facilitate infant development and to minimize development of positional plagiocephaly. Parents are encouraged to place the infant in tummy time while awake and supervised for short periods beginning soon after hospital discharge, increasing incrementally to at least 15 to 30 minutes total daily by 7 weeks of age.

Positional plagiocephaly, or plagiocephaly without synostosis, can be associated with supine sleeping position (aOR, 7.2; 95% CI, 2.98 to 16.53).²¹⁵ It is most likely to result if the infant's head position is not varied when placed for sleep, if the infant spends little or no time in awake, supervised tummy time, and if the infant is not held in the upright position when not sleeping.^{215,471,472} Children with developmental delay and/or neurologic injury have increased rates of plagiocephaly without synostosis, although a causal relationship has not been demonstrated.^{215,473–477} In healthy normal children, the incidence of positional plagiocephaly decreases spontaneously from 20% at 8 months to 3% at 24 months of age.⁴⁷¹

One study of 380 infants in the Netherlands found that those whose parents reported awake tummy time fewer than 3 times daily had more than twofold odds of developing

plagiocephaly (aOR, 2.4; 95% CI 0.90 to 6.20).⁴⁷² One US study found that among 66 2-month-old infants, spending at least 15 minutes daily in awake tummy time was associated with earlier attainment of head up 45 and 90 degrees and sitting with head steady at 2 months of age ($P < .05$), but not with earlier attainment of gross motor milestones at 4 or 6 months of age.⁴⁷⁸ Another study of 288 infants in Taiwan found that >30 minutes of parent-reported daily awake tummy time was associated with earlier acquisition of some gross motor milestones ($P < .02$).⁴⁷⁹ Thus, parents should be encouraged to place the infant in tummy time while awake and supervised for short periods of time beginning soon after hospital discharge, increasing incrementally to at least 15 to 30 minutes total daily by 7 weeks of age.^{472,478–480}

SWADDLING

There is no evidence to recommend swaddling as a strategy to reduce the risk of SIDS. There is a high risk for death if a swaddled infant is placed in or rolls to the prone position. If infants are swaddled, always place them on the back. When an infant exhibits signs of attempting to roll, swaddling should no longer be used.

Many cultures and newborn nurseries have traditionally used swaddling, or wrapping the infant in a light blanket, as a strategy to soothe infants and, in some cases, to encourage sleep in the supine position. For instance, some Native American cultures use swaddling in conjunction with cradleboards. More recently, some sleep experts have recommended swaddling, which, when done correctly, can be an effective technique to help calm infants and promote sleep.^{481,482} There is also some evidence that educational interventions about swaddling and other soothing

methods may be an effective way to educate parents about other safe sleep recommendations such as position and bed sharing risks.⁴⁸³

Some have argued that swaddling can alter certain risk factors for sleep-related death, thus reducing the risk of such deaths. For instance, it has been suggested that the physical restraint associated with swaddling may prevent infants placed supine from rolling to the prone position.⁴⁸¹ One study suggested a decrease in SIDS rate with swaddling if the infant was supine, but notably, there was increased risk of SIDS if the infant was swaddled and placed in the prone position.³⁰⁵ Although another study found a 31-fold increase in SIDS risk with swaddling, the analysis was not stratified by sleep position.²⁸³ Although it may be more likely that parents will initially place a swaddled infant supine, this protective effect may be offset by the 12-fold increased risk for SIDS if the infant is either placed or rolls to the prone position when swaddled.^{305,482} In addition, an analysis of CPSC data found that deaths associated with swaddling were most often attributed to positional asphyxia related to prone sleeping, and a large majority of sleep environments had soft bedding.⁴⁸⁴ Thus, if swaddling is used, the infant should be placed wholly supine. When an infant exhibits signs of attempting to roll (which usually occurs at 3 to 4 months but may occur earlier), swaddling is no longer appropriate, as it could increase the risk of suffocation if the swaddled infant rolls to the prone position.^{305,482,484} Commercially available swaddle sacks are an acceptable alternative, particularly if the parent or caregiver does not know how to swaddle an infant with a conventional thin blanket. Weighted swaddle clothing or weighted

objects within swaddles are not safe and, therefore, not recommended. There is no evidence with regard to SIDS risk related to the arms being swaddled in or out. Parents can decide on an individual basis whether to swaddle, and whether the arms are swaddled in or out, depending on the behavioral needs of the infant.

There is some evidence that swaddling may cause detrimental physiologic consequences. For example, it can cause an increase in respiratory rate,⁴⁸⁵ and tight swaddling can reduce the infant's functional residual lung capacity.^{481,486,487} Tight swaddling can also exacerbate hip dysplasia if the hips are kept in extension and adduction,^{488–491} which is particularly important because some have advocated that the calming effects of swaddling are related to the “tightness” of the swaddling. In contrast, “loose” or incorrectly applied swaddling could result in airway obstruction and, in some cases, strangulation if the blankets become loose in the bed. Swaddling may also possibly increase the risk of overheating in some situations, especially when the head is covered or there is infection.^{492,493} However, 1 study found no increase in abdominal skin temperature when infants were swaddled in a light cotton blanket from the shoulders down.⁴⁸⁶

Impaired arousal has often been postulated as a mechanism contributing to SIDS, and several studies have investigated the relationship between swaddling and arousal and sleep patterns in infants. Physiologic studies have demonstrated that, in general, swaddling decreases startling,⁴⁸⁵ increases sleep duration, and decreases spontaneous awakenings.⁴⁹⁴ Swaddling also decreases arousability (ie, increases cortical arousal thresholds) to a

nasal pulsatile air-jet stimulus, especially in infants who are easily arousable when not swaddled.⁴⁸⁵ One study found decreased arousability in infants at 3 months of age who were not usually swaddled and then were swaddled, but no effect on arousability in routinely swaddled infants.⁴⁸⁵ Another study found preterm infants in the NICU had longer total sleep time and quiet sleep time when swaddled.⁴⁹⁵ In contrast, another investigator has shown infants to be more easily arousable⁴⁹⁴ and to have increased autonomic (subcortical) responses to an auditory stimulus when swaddled.⁴⁹⁶ Thus, although swaddling clearly promotes sleep and decreases the number of awakenings, the effects on arousability to an external stimulus remain unclear. Accumulating evidence suggests, however, that routine swaddling has only minimal effects on arousal. In addition, there have been no studies investigating the effects of swaddling on arousal to more relevant stimuli such as hypoxia or hypercapnia.

HEARING SCREENS

Current data do not support the use of newborn hearing screens as screening tests for SIDS.

Few retrospective case-control studies have examined the use of newborn evoked otoacoustic emission hearing screening tests as a tool to identify infants at subsequent risk for SIDS.^{497,498} In a United States study, infants subsequently dying of SIDS did not fail their hearing tests, but compared with controls, showed a decreased signal-to-noise ratio score in the right ear only, at frequencies of 2000, 3000, and 4000 Hz. A United Kingdom study found slight but statistically not significant increases in otoacoustic emissions signals in the right ear

only, particularly at lower frequencies.⁴⁹⁸ A larger, but nonpeer-reviewed report of hearing screening data in Michigan⁴⁹⁹ and a peer-reviewed retrospective study in Hong Kong⁵⁰⁰ showed no relationship between hearing screening test results and SIDS cases. With regard to autopsy findings, a small case-control study found a higher incidence in histologic alterations in brainstem auditory structures in SIDS victims compared with controls.⁵⁰¹ Until additional data are available, hearing screening, particularly given that most results are reported as a simple pass or fail, should not be considered as a valid screening tool to determine which infants may be at subsequent risk for SIDS. Furthermore, an increased risk of SIDS should not be inferred from an abnormal hearing screen result.

SAFE SLEEP EDUCATION AND MODELING

It is essential that physicians, nonphysician clinicians, hospital staff, and child care providers endorse and model safe infant sleep guidelines from the beginning of pregnancy.

Caregiver receipt of safe infant sleep education is associated with increased adherence with the guidelines.²⁷ This education should be culturally appropriate, respectful, nonjudgmental, and aimed at increasing caregiver knowledge of the recommended practices, anticipating and problem solving barriers to safe sleep, addressing caregiver concerns and misconceptions that may create negative attitudes about the recommended practices, and emphasizing that these practices are prevalent, acceptable, and expected (ie, social norms). Language interpreters should be used as needed.

The Theory of Planned Behavior⁵⁰² and other behavioral theories^{503–505} suggest that one is most likely to carry out a specific practice if one has intention to do so. Intention is more likely when one has positive attitudes about the practice and perceives it to be normative behavior (ie, what most people are doing and what others expect one to do).^{506,507} Studies have found that positive attitudes and social norms are highly correlated with safe sleep practices, including breastfeeding. Additionally, interventions that have focused on improving attitudes and social norms regarding safe infant sleep have been effective.⁵⁰⁸ Given that safe sleep practices should begin immediately after birth, safe sleep education should begin in the prenatal period,^{509,510} including at the prenatal visit, so that parents have time to acquire the necessary knowledge, skills, and confidence to practice the recommendations, acquire the necessary items (eg, crib or bassinet) for a safe infant sleep environment, develop positive attitudes and social norms, and form an intention to follow safe sleep practices.

An example of improving attitudes would be to address caregiver concerns about infant comfort, choking, and aspiration while the infant is sleeping supine.^{149,150,508,511,512} Education that is integrated with other health messaging, such as discussion of the risk of falls and potential skull fractures if infants fall from an adult's arms or a sleep surface, can be helpful. Strategies to avoid inadvertent bed sharing could include setting of alarms or alternative activities (books, television shows, etc) to avoid falling asleep. Establishment of safe sleep as normative behavior begins with consistent modeling of these practices by physicians, nonphysician clinicians, and child

care providers. This is particularly important given the growing influence of family members, friends, and social media on parental practice.^{149,513,514} Studies have demonstrated that parents are most likely to use unsafe sleep practices when they have seen these unsafe sleep practices modeled by physicians, nurses, and other clinicians.^{515–517} Quality improvement initiatives to enhance physician and nonphysician clinician adherence with and messaging of safe sleep guidelines have been effective in both the inpatient^{516–521} and ambulatory settings.^{523,524}

MEDIA MESSAGES

It is advised that media and manufacturers follow safe sleep guidelines in their messaging, advertising, production, and sales to promote safe sleep practices as the social norm.

Media images often show unsafe sleep environments, and this sends confusing messages to caregivers. For example, 1 study found that, in magazines targeted toward childbearing women, more than one third of pictures of sleeping infants and two thirds of pictures of infant sleep environments portrayed unsafe sleep positions and sleep environments.³³⁰ Media exposures (including movie, television, magazines, newspapers, websites, and social media), manufacturer advertisements, and store displays affect individual behavior by influencing beliefs, attitudes, and perceived social norms.^{508,525,526} Frequent exposure to health-related media messages can affect individual health decisions,^{527,528} and media messages have been very influential in decisions regarding sleep position.^{154,157,529} Media, images, social network posts, and advertising messages contrary to safe sleep recommendations may

create misinformation about safe sleep practices and provide a false sense of security that infants are safe in unsafe sleep environments or positions.^{331,530,531}

Media and manufacturer messaging and advertising should model safe sleep guidelines in text, photos, videos, and illustrations, especially when targeting consumer groups with a disproportionate rate of sudden unexpected infant death, such as non-Hispanic Black and American Indian and Alaska Native families.⁵³⁰ Studies have shown that a “one-size-fits-all” message does not resonate equally across different racial and ethnic groups, as it fails to account for group-specific sociocultural practices and credibility or resemblance of the messenger to the intended audience.^{532,533} For some audiences, the inclusion of all parents and grandparents, as well as age, race, or gender-concordant role models and messengers, may be more appropriate.^{534–537} To address the evolving needs of the families they serve, public health departments, hospitals and birthing centers, and organizations that provide safe sleep information should review, revise, and reissue this information on an as-needed basis, but at least every 5 years, to ensure that each generation of new parents receives appropriate information.^{508,531}

RECOMMENDATIONS

The recommendations for a safe infant sleeping environment to reduce the risk of both SIDS and other sleep-related infant deaths are specified in the accompanying policy statement.¹³⁰

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ABBREVIATIONS

5-HT: serotonin or 5-hydroxytryptamine
5-HT1A: serotonin 1A
AAP: American Academy of Pediatrics
aOR: adjusted odds ratio
ASSB: accidental suffocation or strangulation in bed
CDC: Centers for Disease Control and Prevention
CI: confidence interval
CO₂: carbon dioxide
CPSC: Consumer Product Safety Commission
FDA: US Food and Drug Administration
GER: gastroesophageal reflux
GERD: gastroesophageal reflux disease
ICD-10: International Statistical Classification of Diseases and Related Health Problems 10th Revision
ICD-11: International Statistical Classification of Diseases and Related Health Problems 11th Revision
LQTS: long QT syndrome
OR: odds ratio
PRAMS: Pregnancy Risk Assessment and Monitoring System
SES: socioeconomic status
SIDS: sudden infant death syndrome
SUID: sudden unexpected infant death

REFERENCES

1. Moon RY; Task Force on Sudden Infant Death Syndrome. Task Force on Sudden Infant Death Syndrome, SIDS and other sleep-related infant deaths: evidence base for 2016 updated recommendations for a safe infant sleeping environment. *Pediatrics*. 2016;138(5):e20162940

2. Moon RY. Task Force on Sudden Infant Death Syndrome, SIDS and other sleep-related infant deaths: updated 2016 recommendations for a safe infant sleeping environment. *Pediatrics*. 2016;138(5):e20162938.
3. Ebell MH, Siwek J, Weiss BD, et al. Strength of recommendation taxonomy (SORT): a patient-centered approach to grading evidence in the medical literature. *Am Fam Physician*. 2004;69(3):548–556
4. Shapiro-Mendoza CA, Palusci VJ, Hoffman BD, Batra E, Yester M, Corey TS; American Academy of Pediatrics Task Force on Sudden Infant Death Syndrome, Council on Child Abuse and Neglect, Council on Injury, Violence and Poison Prevention, Section on Child Death Review and Prevention, National Association of Medical Examiners. Half century since SIDS: a reappraisal of terminology. *Pediatrics*. 2022;148(4):e2021053746
5. Bundock EA, Corey TS, eds. *National Association of Medical Examiners' Panel on Sudden Unexpected Death in Pediatrics, Unexplained Pediatric Deaths: Investigation, Certification, and Family Needs*. San Diego, CA: Academic Forensic Pathology International; 2019
6. Goldstein RD, Blair PS, Sens MA, et al; 3rd International Congress on Sudden Infant and Child Death. Inconsistent classification of unexplained sudden deaths in infants and children hinders surveillance, prevention and research: recommendations from The 3rd International Congress on Sudden Infant and Child Death. *Forensic Sci Med Pathol*. 2019;15(4):622–628
7. Centers for Disease Control and Prevention. Sudden unexplained infant death investigation reporting form (SUIDIRF). Available at: www.cdc.gov/SIDS/SUIDIRF.htm. Accessed June 1, 2022
8. Hanzlick RL, Jentzen JM, Clark SC. *Sudden, Unexplained Infant Death Investigation, Infant Death Investigation: Guidelines for the Scene Investigator*. Atlanta, GA: Department of Health and Human Services (US), Centers for Disease Control; 2007.
9. Camperlengo LT, Shapiro-Mendoza CK, Kim SY. Sudden infant death syndrome: diagnostic practices and investigative policies, 2004. *Am J Forensic Med Pathol*. 2012;33(3):197–201
10. Erck Lambert AB, Parks SE, Camperlengo L, et al. Death scene investigation and autopsy practices in sudden unexpected infant deaths. *J Pediatr*. 2016;174:84–90.e1
11. Cottengim C, Parks S, Rhoda D, et al. Protocols, practices, and needs for investigating sudden unexpected infant deaths. *Forensic Sci Med Pathol*. 2020;16(1):91–98
12. Byard RW, Shipstone RA, Young J. Continuing major inconsistencies in the classification of unexpected infant deaths. *J Forensic Leg Med*. 2019;64:20–22
13. Krous HF, Chadwick AE, Haas EA, Stanley C. Pulmonary intra-alveolar hemorrhage in SIDS and suffocation. *J Forensic Leg Med*. 2007;14(8):461–470
14. Shapiro-Mendoza CK, Parks SE, Brustrom J, et al. Variations in cause-of-death determination for sudden unexpected infant deaths. *Pediatrics*. 2017;140(1):e20170087
15. Kim SY, Shapiro-Mendoza CK, Chu SY, Camperlengo LT, Anderson R. Differentiating cause-of-death terminology for deaths coded as SIDS, accidental suffocation, and unknown cause: an investigation using US death certificates, 2003–2004. *Am J Forensic Sci*. 2012;57(2):364–369
16. Shapiro-Mendoza CK, Kim SY, Chu SY, Kahn E, Anderson RN. Using death certificates to characterize sudden infant death syndrome (SIDS): opportunities and limitations. *J Pediatr*. 2010;156(1):38–43
17. Kattwinkel J, Brooks J, Myerberg D. American Academy of Pediatrics AAP Task Force on Infant Positioning and SIDS: positioning and SIDS. [published correction appears in *Pediatrics* 1992;90(2 Pt 1):264] *Pediatrics*. 1992;89(6 Pt 1):1120–1126
18. NICHD, National Institutes of Health. Safe to sleep campaign. Available at: www.nichd.nih.gov/sts/Pages/default.aspx. Accessed June 1, 2022
19. NICHD, National Institutes of Health. Fast Facts about SIDS. Available at: safetosleep.nichd.nih.gov/safesleepbasics/SIDS/fastfacts. Accessed June 1, 2022
20. United States Department of Health and Human Services (US DHHS). Centers of Disease Control and Prevention (CDC), National Center for Health Statistics (NCHS), Office of Analysis and Epidemiology (OAE), Division of Vital Statistics (DVS), linked birth/infant death records on CDC WONDER online database. Available at: <http://wonder.cdc.gov/>. Accessed June 1, 2022
21. Malloy MH, MacDorman M. Changes in the classification of sudden unexpected infant deaths: United States, 1992–2001. *Pediatrics*. 2005;115(5):1247–1253
22. Shapiro-Mendoza CK, Tomashek KM, Anderson RN, Wingo J. Recent national trends in sudden, unexpected infant deaths: more evidence supporting a change in classification or reporting. *Am J Epidemiol*. 2006;163(8):762–769
23. Shapiro-Mendoza CK, Kimball M, Tomashek KM, Anderson RN, Blanding S. US infant mortality trends attributable to accidental suffocation and strangulation in bed from 1984 through 2004: are rates increasing? *Pediatrics*. 2009;123(2):533–539
24. Matthews TJ, MacDorman MF, Thoma ME. Infant mortality statistics from the 2013 period linked birth/infant death data set. *Natl Vital Stat Rep*. 2015;64(9):1–30
25. Erck Lambert AB, Parks SE, Shapiro-Mendoza CK. National and state trends in sudden unexpected infant death: 1990–2015. *Pediatrics*. 2018;141(3):e20173519
26. Parks SE, Erck Lambert AB, Shapiro-Mendoza CK. Racial and ethnic trends in sudden unexpected infant deaths: United States, 1995–2013. *Pediatrics*. 2017;139(6):e20163844
27. Hirai AH, Kortsmit K, Kaplan L, et al. Prevalence and factors associated with safe infant sleep practices. *Pediatrics*. 2019;144(5):e20191286
28. Krieger N, Williams DR, Moss NE. Measuring social class in US public health research: concepts, methodologies, and guidelines. *Annu Rev Public Health*. 1997;18:341–378
29. Shipstone RA, Young J, Kearney L, Thompson JMD. Applying a social exclusion framework to explore the relationship between sudden

- unexpected deaths in infancy (SUDI) and social vulnerability. *Front Public Health*. 2020;8:563573
30. Cutter SL, Boruff BJ, Shirley WL. Social vulnerability to environmental hazards. *Soc Sci Q*. 2003;84(2):242–261
 31. Spencer N, Logan S. Sudden unexpected death in infancy and socioeconomic status: a systematic review. *J Epidemiol Community Health*. 2004; 58(5):366–373
 32. Centers for Disease Control and Prevention. PRAMS. Available at: <https://www.cdc.gov/prams/index.htm>. Accessed June 1, 2022
 33. Lahr MB, Rosenberg KD, Lapidus JA. Maternal-infant bedsharing: risk factors for bedsharing in a population-based survey of new mothers and implications for SIDS risk reduction. *Matern Child Health J*. 2007;11(3):277–286
 34. Willinger M, Ko CW, Hoffman HJ, Kessler RC, Corwin MJ; National Infant Sleep Position study. Trends in infant bed sharing in the United States, 1993–2000: the National Infant Sleep Position study. *Arch Pediatr Adolesc Med*. 2003;157(1):43–49
 35. Fu LY, Colson ER, Corwin MJ, Moon RY. Infant sleep location: associated maternal and infant characteristics with sudden infant death syndrome prevention recommendations. *J Pediatr*. 2008;153(4):503–508
 36. Shapiro-Mendoza CK, Parks S, Lambert AE, Camperlengo L, Cottengim C, Olson C. The epidemiology of sudden infant death syndrome and sudden unexpected infant deaths: diagnostic shift and other temporal changes. In: *SIDS Sudden Infant and Early Childhood Death: The Past, the Present and the Future*, JR Duncan, RW Byard, Eds. Australia: Adelaide; 2018
 37. Erck Lambert AB, 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
 38. Bass JL, Gartley T, Lyczkowski DA, Kleinman R. Trends in the incidence of sudden unexpected infant death in the newborn: 1995–2014. *J Pediatr*. 2018;196:104–108
 39. Lavista Ferres JM, Anderson TM, Johnston R, Ramirez JM, Mitchell EA. Distinct populations of sudden unexpected infant death based on age. *Pediatrics*. 2020;145(1):e20191637
 40. Filiano JJ, Kinney HC. A perspective on neuropathologic findings in victims of the sudden infant death syndrome: the triple-risk model. *Biol Neonate*. 1994;65(3-4):194–197
 41. Kinney HC. Brainstem mechanisms underlying the sudden infant death syndrome: evidence from human pathologic studies. *Dev Psychobiol*. 2009;51(3):223–233
 42. Kinney HC, Thach BT. The sudden infant death syndrome. *N Engl J Med*. 2009;361(8):795–805
 43. Hunt NJ, Phillips L, Waters KA, Machaalani R. Proteomic MALDI-TOF/TOF-IMS examination of peptide expression in the formalin fixed brainstem and changes in sudden infant death syndrome infants. *J Proteomics*. 2016;138:48–60
 44. Lavezzi AM, Ferrero S, Lattuada D, Pisciole F, Alfonsi G, Maturri L. Pathobiological expression of the brain-derived neurotrophic factor (BDNF) in cerebellar cortex of sudden fetal and infant death victims. *Int J Dev Neurosci*. 2018;66:9–17
 45. Malloy MH. Prematurity and sudden infant death syndrome: United States 2005–2007. *J Perinatol*. 2013;33(6): 470–475
 46. Sowter B, Doyle LW, Morley CJ, Altmann A, Halliday J. Is sudden infant death syndrome still more common in very low birthweight infants in the 1990s? *Med J Aust*. 1999;171(8):411–413
 47. Kinney HC, Randall LL, Sleeper LA, et al. Serotonergic brainstem abnormalities in Northern Plains Indians with the sudden infant death syndrome. *J Neuropathol Exp Neurol*. 2003;62(11): 1178–1191
 48. Bednarczuk N, Milner A, Greenough A. The role of maternal smoking in sudden fetal and infant death pathogenesis. *Front Neurol*. 2020;11:586068
 49. Browne CJ, Sharma N, Waters KA, Machaalani R. The effects of nicotine on the alpha-7 and beta-2 nicotinic acetylcholine receptor subunits in the developing piglet brainstem. *Int J Dev Neurosci*. 2010;28(1):1–7
 50. Hunt NJ, Waters KA, Machaalani R. Orexin receptors in the developing piglet hypothalamus, and effects of nicotine and intermittent hypercapnic hypoxia exposures. *Brain Res*. 2013;1508:73–82
 51. Vivekanandarajah A, Waters KA, Machaalani R. Cigarette smoke exposure effects on the brainstem expression of nicotinic acetylcholine receptors (nAChRs), and on cardiac, respiratory and sleep physiologies. *Respir Physiol Neurobiol*. 2019;259:1–15
 52. Cerpa VJ, Aylwin ML, Beltrán-Castillo S, et al. The alteration of neonatal raphe neurons by prenatal-perinatal nicotine: meaning for sudden infant death syndrome. *Am J Respir Cell Mol Biol*. 2015;53(4):489–499
 53. Slotkin TA, Seidler FJ, Spindel ER. Prenatal nicotine exposure in rhesus monkeys compromises development of brainstem and cardiac monoamine pathways involved in perinatal adaptation and sudden infant death syndrome: amelioration by vitamin C. *Neurotoxicol Teratol*. 2011;33(3): 431–434
 54. Sekizawa S, Joad JP, Pinkerton KE, Bonham AC. Secondhand smoke exposure alters K⁺ channel function and intrinsic cell excitability in a subset of second-order airway neurons in the nucleus tractus solitarius of young guinea pigs. *Eur J Neurosci*. 2010;31(4):673–684
 55. Duncan JR, Paterson DS, Hoffman JM, et al. Brainstem serotonergic deficiency in sudden infant death syndrome. *JAMA*. 2010;303(5):430–437
 56. Duncan JR, Garland M, Myers MM, et al. Prenatal nicotine-exposure alters fetal autonomic activity and medullary neurotransmitter receptors: implications for sudden infant death syndrome. *J Appl Physiol*. 2009;107(5): 1579–1590
 57. Duncan JR, Garland M, Stark RI, et al. Prenatal nicotine exposure selectively affects nicotinic receptor expression in primary and associative visual cortices of the fetal baboon. *Brain Pathol*. 2015;25(2):171–181

58. St-John WM, Leiter JC. Maternal nicotine depresses eupneic ventilation of neonatal rats. *Neurosci Lett*. 1999;267(3):206–208
59. Eugenin J, Otárola M, Bravo E, et al. Prenatal to early postnatal nicotine exposure impairs central chemoreception and modifies breathing pattern in mouse neonates: a probable link to sudden infant death syndrome. *J Neurosci*. 2008;28(51):13907–13917
60. Lee SY, Sirieix CM, Nattie E, Li A. Pre- and early postnatal nicotine exposure exacerbates autoresuscitation failure in serotonin-deficient rat neonates. *J Physiol*. 2018;596(23):5977–5991
61. Zhao L, Zhuang J, Gao X, Ye C, Lee LY, Xu F. From the cover: prenatal nicotine exposure attenuates respiratory chemoreflexes associated with downregulation of tyrosine hydroxylase and neurokinin 1 receptor in rat pup carotid body. *Toxicol Sci*. 2016;153(1):103–111
62. Fewell JE, Smith FG, Ng VK. Prenatal exposure to nicotine impairs protective responses of rat pups to hypoxia in an age-dependent manner. *Respir Physiol*. 2001;127(1):61–73
63. Hafström O, Milerad J, Sundell HW. Prenatal nicotine exposure blunts the cardiorespiratory response to hypoxia in lambs. *Am J Respir Crit Care Med*. 2002;166(12 Pt 1):1544–1549
64. Duncan JR, Paterson DS, Kinney HC. The development of nicotinic receptors in the human medulla oblongata: interrelationship with the serotonergic system. *Auton Neurosci*. 2008;144(1–2):61–75
65. Wilhelm-Benartzi CS, Houseman EA, Maccani MA, et al. In utero exposures, infant growth, and DNA methylation of repetitive elements and developmentally related genes in human placenta. *Environ Health Perspect*. 2012;120(2):296–302
66. Aishah A, Hinton T, Waters KA, Machaalani R. The $\alpha 3$ and $\alpha 4$ nicotinic acetylcholine receptor (nAChR) subunits in the brainstem medulla of sudden infant death syndrome (SIDS). *Neurobiol Dis*. 2019;125:23–30
67. Ambrose N, Waters KA, Rodríguez ML, Bailey K, Machaalani R. Neuronal apoptosis in the brainstem medulla of sudden unexpected death in infancy (SUDI), and the importance of standardized SUDI classification. *Forensic Sci Med Pathol*. 2018;14(1):42–56
68. Machaalani R, Chen H. Brain derived neurotrophic factor (BDNF), its tyrosine kinase receptor B (TrkB) and nicotine. *Neurotoxicology*. 2018;65:186–195
69. Schneider J, Mitchell I, Singhal N, Kirk V, Hasan SU. Prenatal cigarette smoke exposure attenuates recovery from hypoxemic challenge in preterm infants. *Am J Respir Crit Care Med*. 2008;178(5):520–526
70. Thiriez G, Bouhaddi M, Mouro L, et al. Heart rate variability in preterm infants and maternal smoking during pregnancy. *Clin Auton Res*. 2009;19(3):149–156
71. Fifer WP, Fingers ST, Youngman M, Gomez-Gribben E, Myers MM. Effects of alcohol and smoking during pregnancy on infant autonomic control. *Dev Psychobiol*. 2009;51(3):234–242
72. Ali K, Rosser T, Bhat R, et al. Antenatal smoking and substance-misuse, infant and newborn response to hypoxia. *Pediatr Pulmonol*. 2017;52(5):650–655
73. Rossor T, Ali K, Bhat R, Trenear R, Rafferty G, Greenough A. The effects of sleeping position, maternal smoking and substance misuse on the ventilatory response to hypoxia in the newborn period. *Pediatr Res*. 2018;84(3):411–418
74. Richardson HL, Walker AM, Horne RS. Maternal smoking impairs arousal patterns in sleeping infants. *Sleep*. 2009;32(4):515–521
75. Cohen G, Vella S, Jeffery H, Lagercrantz H, Katz-Salamon M. Cardiovascular stress hyperreactivity in babies of smokers and in babies born preterm. *Circulation*. 2008;118(18):1848–1853
76. Paine SM, Jacques TS, Sebire NJ. Review: neuropathological features of unexplained sudden unexpected death in infancy: current evidence and controversies. *Neuropathol Appl Neurobiol*. 2014;40(4):364–384
77. Panigrahy A, Filiano J, Sleeper LA, et al. Decreased serotonergic receptor binding in rhombic lip-derived regions of the medulla oblongata in the sudden infant death syndrome. *J Neuropathol Exp Neurol*. 2000;59(5):377–384
78. Ozawa Y, Takashima S. Developmental neurotransmitter pathology in the brainstem of sudden infant death syndrome: a review and sleep position. *Forensic Sci Int*. 2002;130(Suppl):S53–S59
79. Machaalani R, Say M, Waters KA. Serotonergic receptor 1A in the sudden infant death syndrome brainstem medulla and associations with clinical risk factors. *Acta Neuropathol*. 2009;117(3):257–265
80. Paterson DS, Trachtenberg FL, Thompson EG, et al. Multiple serotonergic brainstem abnormalities in sudden infant death syndrome. *JAMA*. 2006;296(17):2124–2132
81. Bright FM, Byard RW, Vink R, Paterson DS. Medullary serotonin neuron abnormalities in an Australian cohort of sudden infant death syndrome. *J Neuropathol Exp Neurol*. 2017;76(10):864–873
82. Donnelly WT, Bartlett D Jr, Leiter JC. Serotonin in the solitary tract nucleus shortens the laryngeal chemoreflex in anaesthetized neonatal rats. *Exp Physiol*. 2016;101(7):946–961
83. Donnelly WT, Xia L, Bartlett D, Leiter JC. Activation of serotonergic neurons in the medullary caudal raphe shortens the laryngeal chemoreflex in anaesthetized neonatal rats. *Exp Physiol*. 2017;102(8):1007–1018
84. Dosumu-Johnson RT, Cocoran AE, Chang Y, Nattie E, Dymecki SM. Acute perturbation of *Pet1*-neuron activity in neonatal mice impairs cardiorespiratory homeostatic recovery. *eLife*. 2018;7:e37857
85. Barrett KT, Dosumu-Johnson RT, Dautenspeck JA, et al. Partial raphe dysfunction in neurotransmission is sufficient to increase mortality after anoxic exposures in mice at a critical period in postnatal development. *J Neurosci*. 2016;36(14):3943–3953
86. Say M, Machaalani R, Waters KA. Changes in serotonergic receptors 1A and 2A in the piglet brainstem after intermittent hypercapnic hypoxia (IHH) and nicotine. *Brain Res*. 2007;1152:17–26
87. Kinney HC, Richerson GB, Dymecki SM, Darnall RA, Nattie EE. The brainstem and serotonin in the sudden infant

- death syndrome. *Annu Rev Pathol.* 2009;4:517–550
88. Cummings KJ, Commons KG, Fan KC, Li A, Nattie EE. Severe spontaneous bradycardia associated with respiratory disruptions in rat pups with fewer brain stem 5-HT neurons. *Am J Physiol Regul Integr Comp Physiol.* 2009; 296(6):R1783–R1796
89. Cummings KJ, Hewitt JC, Li A, Daubenspeck JA, Nattie EE. Postnatal loss of brainstem serotonin neurones compromises the ability of neonatal rats to survive episodic severe hypoxia. *J Physiol.* 2011;589(Pt 21):5247–5256
90. Darnall RA, Schneider RW, Tobia CM, Commons KG. Eliminating medullary 5-HT neurons delays arousal and decreases the respiratory response to repeated episodes of hypoxia in neonatal rat pups. *J Appl Physiol (1985).* 2016;120(5):514–525
91. Lavezzi AM, Weese-Mayer DE, Yu MY, et al. Developmental alterations of the respiratory human retrotrapezoid nucleus in sudden unexplained fetal and infant death. *Auton Neurosci.* 2012;170(1–2):12–19
92. Kon FC, Vázquez RZ, Lang A, Cohen MC. Hippocampal abnormalities and seizures: a 16-year single center review of sudden unexpected death in childhood, sudden unexpected death in epilepsy and SIDS. *Forensic Sci Med Pathol.* 2020;16(3):423–434
93. Lavezzi AM, Mehboob R, Alfonsi G, Ferrero S. Substantia nigra abnormalities provide new insight on the neural mechanisms underlying the sleep-arousal phase dysfunctions in sudden infant death syndrome. *ASN Neuro.* 2020;12:1759091420962695
94. Porzionato A, Macchi V, De Caro R. Central and peripheral chemoreceptors in sudden infant death syndrome. *J Physiol.* 2018;596(15):3007–3019
95. Hunt NJ, Waters KA, Machaalani R. Promotion of the unfolding protein response in orexin/dynorphin neurons in sudden infant death syndrome (SIDS): elevated pPERK and ATF4 expression. *Mol Neurobiol.* 2017;54(9):7171–7185
96. Hunt NJ, Waters KA, Rodríguez ML, Machaalani R. Decreased orexin (hypocretin) immunoreactivity in the hypothalamus and pontine nuclei in sudden infant death syndrome. *Acta Neuropathol.* 2015;130(2):185–198
97. Waters KA, Hunt NJ, Machaalani R. Neuropathology of sudden infant death syndrome: hypothalamus. In: *SIDS Sudden Infant and Early Childhood Death: The Past, the Present and the Future*, Duncan JR, Byard RW, eds. Australia: Adelaide; 2018
98. Haynes RL, Frelinger AL III, Giles EK, et al. High serum serotonin in sudden infant death syndrome. *Proc Natl Acad Sci USA.* 2017;114(29):7695–7700
99. Opdal SH, Rognum TO. The sudden infant death syndrome gene: does it exist? *Pediatrics.* 2004;114(4):e506–e512
100. Opdal SH, Rognum TO. Gene variants predisposing to SIDS: current knowledge. *Forensic Sci Med Pathol.* 2011; 7(1):26–36
101. Tester DJ, Wong LCH, Chanana P, et al. Exome-wide rare variant analyses in sudden infant death syndrome. *J Pediatr.* 2018;203:423–428.e11
102. Rosenthal NA, Currier RJ, Baer RJ, Feuchtbau L, Jelliffe-Pawlowski LL. Undiagnosed metabolic dysfunction and sudden infant death syndrome—a case-control study. *Paediatr Perinat Epidemiol.* 2015;29(2):151–155
103. Hedley PL, Jørgensen P, Schlamowitz S, et al. The genetic basis of long QT and short QT syndromes: a mutation update. *Hum Mutat.* 2009;30(11): 1486–1511
104. Weese-Mayer DE, Ackerman MJ, Marazita ML, Berry-Kravis EM. Sudden infant death syndrome: review of implicated genetic factors. *Am J Med Genet A.* 2007;143A(8):771–788
105. Wang DW, Desai RR, Crotti L, et al. Cardiac sodium channel dysfunction in sudden infant death syndrome. *Circulation.* 2007;115(3):368–376
106. Tan BH, Pundi KN, Van Norstrand DW, et al. Sudden infant death syndrome-associated mutations in the sodium channel beta subunits. *Heart Rhythm.* 2010;7(6):771–778
107. Van Norstrand DW, Asimaki A, Rubinos C, et al. Connexin43 mutation causes heterogeneous gap junction loss and sudden infant death. *Circulation.* 2012;125(3):474–481
108. Andreassen C, Refsgaard L, Nielsen JB, et al. Mutations in genes encoding cardiac ion channels previously associated with sudden infant death syndrome (SIDS) are present with high frequency in new exome data. *Can J Cardiol.* 2013;29(9): 1104–1109
109. Winkel BG, Yuan L, Olesen MS, et al. The role of the sodium current complex in a nonreferred nationwide cohort of sudden infant death syndrome. *Heart Rhythm.* 2015;12(6):1241–1249
110. Zimmer T, Surber R. SCN5A channelopathies—an update on mutations and mechanisms. *Prog Biophys Mol Biol.* 2008;98(2–3):120–136
111. Schwartz PJ, Priori SG, Dumaine R, et al. A molecular link between the sudden infant death syndrome and the long-QT syndrome. *N Engl J Med.* 2000;343(4):262–267
112. Paterson DS, Rivera KD, Broadbelt KG, et al. Lack of association of the serotonin transporter polymorphism with the sudden infant death syndrome in the San Diego Dataset. *Pediatr Res.* 2010;68(5):409–413
113. Hafke A, Schürmann P, Rothämel T, Dörk T, Klitschar M. Evidence for an association of interferon gene variants with sudden infant death syndrome. *Int J Legal Med.* 2019;133(3):863–869
114. Fard D, Lärer K, Rothämel T, et al. Candidate gene variants of the immune system and sudden infant death syndrome. *Int J Legal Med.* 2016; 130(4):1025–1033
115. Cummings KJ, Klotz C, Liu WQ, et al. Sudden infant death syndrome (SIDS) in African Americans: polymorphisms in the gene encoding the stress peptide pituitary adenylate cyclase-activating polypeptide (PACAP). *Acta Paediatr.* 2009;98(3):482–489
116. Barrett KT, Rodikova E, Weese-Mayer DE, et al. Analysis of PAC1 receptor gene variants in Caucasian and African American infants dying of sudden infant death syndrome. *Acta Paediatr.* 2013;102(12):e546–e552
117. Trent M, Dooley DG, Dougé J, Section On Adolescent H; Section on Adolescent Health; Council on Community Pediatrics; Committee on Adolescence. The impact of racism on child and

- adolescent health. *Pediatrics*. 2019; 144(2):e20191765
118. Burris HH, Hwang SS, Collins JW Jr, Kirpalani H, Wright CJ. Re-conceptualizing associations between race and morbidities of extreme prematurity. *J Pediatr*. 2019;207:10–14.e1
119. Lang J, McKie J, Smith H, et al. Adverse childhood experiences, epigenetics and telomere length variation in childhood and beyond: a systematic review of the literature. *Eur Child Adolesc Psychiatry*. 2020;29(10):1329–1338
120. Ridout KK, Khan M, Ridout SJ. Adverse childhood experiences run deep: toxic early life stress, telomeres, and mitochondrial DNA copy number, the biological markers of cumulative stress. *BioEssays*. 2018;40(9):e1800077
121. Ferrante L, Opdal SH, Vege A, Rognum T. Cytokine gene polymorphisms and sudden infant death syndrome. *Acta Paediatr*. 2010;99(3):384–388
122. Ferrante L, Opdal SH, Vege A, Rognum TO. IL-1 gene cluster polymorphisms and sudden infant death syndrome. *Hum Immunol*. 2010;71(4):402–406
123. Opdal SH, Ferrante L, Rognum TO, Stray-Pedersen A. Aquaporin-1 and aquaporin-9 gene variations in sudden infant death syndrome. *Int J Legal Med*. 2021;135(3):719–725
124. Opdal SH, Rognum TO, Vege A, Stave AK, Dupuy BM, Egeland T. Increased number of substitutions in the D-loop of mitochondrial DNA in the sudden infant death syndrome. *Acta Paediatr*. 1998;87(10):1039–1044
125. Opdal SH, Rognum TO, Torgersen H, Vege A. Mitochondrial DNA point mutations detected in four cases of sudden infant death syndrome. *Acta Paediatr*. 1999;88(9):957–960
126. Santorelli FM, Schlessel JS, Slonim AE, DiMauro S. Novel mutation in the mitochondrial DNA tRNA glycine gene associated with sudden unexpected death. *Pediatr Neurol*. 1996;15(2):145–149
127. 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):404–409
128. Forsyth L, Hume R, Howatson A, Busuttil A, Burchell A. Identification of novel polymorphisms in the glucokinase and glucose-6-phosphatase genes in infants who died suddenly and unexpectedly. *J Mol Med (Berl)*. 2005;83(8):610–618
129. Bartick M, Stehel EK, Calhoun SL, et al. Academy of breastfeeding medicine position statement and guideline: infant feeding and lactation-related language and gender. *Breastfeed Med*. 2021;16(8):587–590
130. Moon RY, Carlin RF, Hand I, American Academy of Pediatrics, Task Force on Sudden Infant Death Syndrome, Committee on Fetus and Newborn. Policy statement: sleep-related infant deaths: updated 2022 recommendations for reducing infant deaths in the sleep environment. *Pediatrics*. 2022;150(1):e2022057990
131. Kanetake J, Aoki Y, Funayama M. Evaluation of rebreathing potential on bedding for infant use. *Pediatr Int*. 2003;45(3):284–289
132. Kemp JS, Thach BT. Quantifying the potential of infant bedding to limit CO₂ dispersal and factors affecting rebreathing in bedding. *J Appl Physiol*. 1995;78(2):740–745
133. Kemp JS, Livne M, White DK, Arfken CL. Softness and potential to cause rebreathing: differences in bedding used by infants at high and low risk for sudden infant death syndrome. *J Pediatr*. 1998;132(2):234–239
134. Patel AL, Harris K, Thach BT. Inspired CO₂ and O₂ in sleeping infants rebreathing from bedding: relevance for sudden infant death syndrome. *J Appl Physiol*. 2001;91(6):2537–2545
135. Tuffnell CS, Petersen SA, Wailoo MP. Prone sleeping infants have a reduced ability to lose heat. *Early Hum Dev*. 1995;43(2):109–116
136. Ammari A, Schulze KF, Ohira-Kist K, et al. Effects of body position on thermal, cardiorespiratory and metabolic activity in low birth weight infants. *Early Hum Dev*. 2009;85(8):497–501
137. Yiallourou SR, Walker AM, Horne RS. Prone sleeping impairs circulatory control during sleep in healthy term infants: implications for SIDS. *Sleep*. 2008;31(8):1139–1146
138. Wong FY, Witcombe NB, Yiallourou SR, et al. Cerebral oxygenation is depressed during sleep in healthy term infants when they sleep prone. *Pediatrics*. 2011;127(3):e558–e565
139. Hauck FR, Herman SM, Donovan M, et al. Sleep environment and the risk of sudden infant death syndrome in an urban population: the Chicago Infant Mortality Study. *Pediatrics*. 2003;111(5 Pt 2):1207–1214
140. Li DK, Petitti DB, Willinger M, et al. Infant sleeping position and the risk of sudden infant death syndrome in California, 1997–2000. *Am J Epidemiol*. 2003;157(5):446–455
141. Blair PS, Fleming PJ, Smith LJ, 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):1457–1461
142. Fleming PJ, Blair PS, Bacon C, et al; Confidential Enquiry into Stillbirths and Deaths Regional Coordinators and Researchers. Environment of infants during sleep and risk of the sudden infant death syndrome: results of 1993–5 case-control study for confidential inquiry into stillbirths and deaths in infancy. *BMJ*. 1996;313(7051):191–195
143. Carpenter RG, Irgens LM, Blair PS, et al. Sudden unexplained infant death in 20 regions in Europe: case control study. *Lancet*. 2004;363(9404):185–191
144. Mitchell EA, Tuohy PG, Brunt JM, et al. Risk factors for sudden infant death syndrome following the prevention campaign in New Zealand: a prospective study. *Pediatrics*. 1997;100(5):835–840
145. Waters KA, Gonzalez A, Jean C, Morielli A, Brouillette RT. Face-straight-down and face-near-straight-down positions in healthy, prone-sleeping infants. *J Pediatr*. 1996;128(5 Pt 1):616–625
146. Oyen N, Markestad T, Skaerven R, et al. Combined effects of sleeping position and prenatal risk factors in sudden infant death syndrome: the Nordic Epidemiological SIDS Study. *Pediatrics*. 1997;100(4):613–621
147. Mitchell EA, Thach BT, Thompson JMD, Williams S. Changing infants' sleep position increases risk of sudden infant

- death syndrome. New Zealand Cot Death Study. *Arch Pediatr Adolesc Med.* 1999;153(11):1136–1141
148. Bombard JM, Kortsmat K, Warner L, et al. Vital signs: trends and disparities in infant safe sleep practices - United States, 2009-2015. *MMWR Morb Mortal Wkly Rep.* 2018;67(1):39–46
149. Oden RP, Joyner BL, Ajao TI, Moon RY. Factors influencing African American mothers' decisions about sleep position: a qualitative study. *J Natl Med Assoc.* 2010;102(10):870–872, 875–880
150. Colson ER, McCabe LK, Fox K, et al. Barriers to following the back-to-sleep recommendations: insights from focus groups with inner-city caregivers. *Ambul Pediatr.* 2005;5(6):349–354
151. Mosley JM, Daily Stokes S, Ulmer A. Infant sleep position: discerning knowledge from practice. *Am J Health Behav.* 2007;31(6):573–582
152. Moon RY, Omron R. Determinants of infant sleep position in an urban population. *Clin Pediatr (Phila).* 2002;41(8):569–573
153. Ottolini MC, Davis BE, Patel K, Sachs HC, Gershon NB, Moon RY. Prone infant sleeping despite the "Back to Sleep" campaign. *Arch Pediatr Adolesc Med.* 1999;153(5):512–517
154. Willinger M, Ko C-W, Hoffman HJ, Kessler RC, Corwin MJ. Factors associated with caregivers' choice of infant sleep position, 1994-1998: the National Infant Sleep Position Study. *JAMA.* 2000;283(16):2135–2142
155. Moon RY, Biliter WM. Infant sleep position policies in licensed child care centers after back to sleep campaign. *Pediatrics.* 2000;106(3):576–580
156. Moon RY, Weese-Mayer DE, Silvestri JM. Nighttime child care: inadequate sudden infant death syndrome risk factor knowledge, practice, and policies. *Pediatrics.* 2003;111(4 Pt 1):795–799
157. Von Kohorn I, Corwin MJ, Rybin DV, Heeren TC, Lister G, Colson ER. Influence of prior advice and beliefs of mothers on infant sleep position. *Arch Pediatr Adolesc Med.* 2010;164(4):363–369
158. Kahn A, Groswasser J, Sottiaux M, Rebuffat E, Franco P, Dramaix M. Prone or supine body position and sleep characteristics in infants. *Pediatrics.* 1993;91(6):1112–1115
159. Bhat RY, Hannam S, Pressler R, Rafferty GF, Peacock JL, Greenough A. Effect of prone and supine position on sleep, apneas, and arousal in preterm infants. *Pediatrics.* 2006;118(1):101–107
160. Ariagno RL, van Liempt S, Mirmiran M. Fewer spontaneous arousals during prone sleep in preterm infants at 1 and 3 months corrected age. *J Perinatol.* 2006;26(5):306–312
161. Franco P, Groswasser J, Sottiaux M, Broadfield E, Kahn A. Decreased cardiac responses to auditory stimulation during prone sleep. *Pediatrics.* 1996;97(2):174–178
162. Galland BC, Reeves G, Taylor BJ, Bolton DP. Sleep position, autonomic function, and arousal. *Arch Dis Child Fetal Neonatal Ed.* 1998;78(3):F189–F194
163. Galland BC, Hayman RM, Taylor BJ, Bolton DP, Sayers RM, Williams SM. Factors affecting heart rate variability and heart rate responses to tilting in infants aged 1 and 3 months. *Pediatr Res.* 2000;48(3):360–368
164. Horne RS, Ferens D, Watts AM, et al. The prone sleeping position impairs arousability in term infants. *J Pediatr.* 2001;138(6):811–816
165. Horne RS, Bandopadhyay P, Vitkovic J, Cranage SM, Adamson TM. Effects of age and sleeping position on arousal from sleep in preterm infants. *Sleep.* 2002;25(7):746–750
166. Kato I, Scaillet S, Groswasser J, et al. Spontaneous arousability in prone and supine position in healthy infants. *Sleep.* 2006;29(6):785–790
167. Phillipson EA, Sullivan CE. Arousal: the forgotten response to respiratory stimuli. *Am Rev Respir Dis.* 1978;118(5):807–809
168. Kahn A, Groswasser J, Rebuffat E, et al. Sleep and cardiorespiratory characteristics of infant victims of sudden death: a prospective case-control study. *Sleep.* 1992;15(4):287–292
169. Schechtman VL, Harper RM, Wilson AJ, Southall DP. Sleep state organization in normal infants and victims of the sudden infant death syndrome. *Pediatrics.* 1992;89(5 Pt 1):865–870
170. Harper RM. State-related physiological changes and risk for the sudden infant death syndrome. *Aust Paediatr J.* 1986;22(Suppl 1):55–58
171. Kato I, Franco P, Groswasser J, et al. Incomplete arousal processes in infants who were victims of sudden death. *Am J Respir Crit Care Med.* 2003;168(11):1298–1303
172. Byard RW, Beal SM. Gastric aspiration and sleeping position in infancy and early childhood. *J Paediatr Child Health.* 2000;36(4):403–405
173. Malloy MH. Trends in postneonatal aspiration deaths and reclassification of sudden infant death syndrome: impact of the "Back to Sleep" program. *Pediatrics.* 2002;109(4):661–665
174. Tablizo MA, Jacinto P, Parsley D, Chen ML, Ramanathan R, Keens TG. Supine sleeping position does not cause clinical aspiration in neonates in hospital newborn nurseries. *Arch Pediatr Adolesc Med.* 2007;161(5):507–510
175. Rosen R, Vandenplas Y, Singendonk M, et al. Pediatric gastroesophageal reflux clinical practice guidelines: joint recommendations of the North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition and the European Society for Pediatric Gastroenterology, Hepatology, and Nutrition. *J Pediatr Gastroenterol Nutr.* 2018;66(3):516–554
176. Meyers WF, Herbst JJ. Effectiveness of positioning therapy for gastroesophageal reflux. *Pediatrics.* 1982;69(6):768–772
177. Tobin JM, McCloud P, Cameron DJ. Posture and gastro-oesophageal reflux: a case for left lateral positioning. *Arch Dis Child.* 1997;76(3):254–258
178. Mannen EM, Carroll J, Bumpass DB, et al. *Biomechanical Analysis of Inclined Sleep Products.* Little Rock, AR: University of Arkansas; 2019
179. Malloy MH, Hoffman HJ. Prematurity, sudden infant death syndrome, and age of death. *Pediatrics.* 1995;96(3 Pt 1):464–471
180. Ostfeld BM, Schwartz-Soicher O, Reichman NE, Teitler JO, Hegyi T.

- Prematurity and sudden unexpected infant deaths in the United States. *Pediatrics*. 2017;140(1):e20163334
181. McMullen SL. Transitioning premature infants supine: state of the science. *MCN Am J Matern Child Nurs*. 2013;38(1):8–12
 182. Nightlinger K. Developmentally supportive care in the neonatal intensive care unit: an occupational therapist's role. *Neonatal Netw*. 2011;30(4):243–248
 183. American Academy of Pediatrics Committee on Fetus and Newborn. Hospital discharge of the high-risk neonate. *Pediatrics*. 2008;122(5):1119–1126
 184. Eichenwald EC; Committee on Fetus and Newborn. Diagnosis and management of gastroesophageal reflux in preterm infants. *Pediatrics*. 2018;142(1):e20181061
 185. Gelfer P, Cameron R, Masters K, Kennedy KA. Integrating “Back to Sleep” recommendations into neonatal ICU practice. *Pediatrics*. 2013;131(4):e1264–e1270
 186. Hwang SS, O’Sullivan A, Fitzgerald E, Melvin P, Gorman T, Fiascone JM. Implementation of safe sleep practices in the neonatal intensive care unit. *J Perinatol*. 2015;35(10):862–866
 187. Goodstein MH, Stewart DL, Keels EL, Moon RY; Committee on Fetus and Newborn, Task Force on Sudden Infant Death Syndrome. Transition to a safe home sleep environment for the NICU patient. *Pediatrics*. 2021;148(1):e2021052045
 188. Feldman-Winter L, Goldsmith JP; Committee on Fetus and Newborn, Task Force on Sudden Infant Death Syndrome. Safe sleep and skin-to-skin care in the neonatal period for healthy term newborns. *Pediatrics*. 2016;138(3):e20161889
 189. Moon RY, Oden RP, Joyner BL, Ajao TI. Qualitative analysis of beliefs and perceptions about sudden infant death syndrome in African-American mothers: implications for safe sleep recommendations. *J Pediatr*. 2010;157(1):92–97.e2
 190. Brenner RA, Simons-Morton BG, Bhaskar B, et al. Prevalence and predictors of the prone sleep position among inner-city infants. *JAMA*. 1998;280(4):341–346
 191. Willinger M, Hoffman HJ, Wu K-T, et al. Factors associated with the transition to nonprone sleep positions of infants in the United States: the National Infant Sleep Position Study. *JAMA*. 1998;280(4):329–335
 192. Colvin JD, Collie-Akers V, Schunn C, Moon RY. Sleep environment risks for younger and older infants. *Pediatrics*. 2014;134(2):e406–e412
 193. Kemp JS, Nelson VE, Thach BT. Physical properties of bedding that may increase risk of sudden infant death syndrome in prone-sleeping infants. *Pediatr Res*. 1994;36(1 Pt 1):7–11
 194. Getahun D, Amre D, Rhoads GG, Demissie K. Maternal and obstetric risk factors for sudden infant death syndrome in the United States. *Obstet Gynecol*. 2004;103(4):646–652
 195. U.S. Consumer Product Safety Commission. Safety standard for bassinets and caddles. Vol 89, No. 205. Washington, DC. *Fed Regist*. 2013;63019–63036
 196. U.S. Consumer Product Safety Commission. *Safety Standard for Play Yards*, Vol. 77: No. 168. Washington, DC: Federal Register; 2012
 197. Nakamura S, Wind M, Danello MA. Review of hazards associated with children placed in adult beds. *Arch Pediatr Adolesc Med*. 1999;153(10):1019–1023
 198. U.S. Consumer Product Safety Commission. *Safety Standard for Portable Bed Rails*, Vol. 77: No. 44. Washington, DC: Federal Register; 2012
 199. U.S. Consumer Product Safety Commission. *Safety Standard for Bedside Sleepers*, Vol. 79: No. 10. Washington, DC: Federal Register; 2014
 200. U.S. Consumer Product Safety Commission. *Final Rule: Safety Standard for Infant Sleep Products*. Washington, DC: Federal Register; 2021
 201. Baddock SA, Tipene-Leach D, Williams SM, et al. Wahakura versus bassinet for safe infant sleep: a randomized trial. *Pediatrics*. 2017;139(2):e20160162
 202. Tipene-Leach D, Baddock SA, Williams SM, et al. The Pēpi-Pod study: overnight video, oximetry and thermal environment while using an in-bed sleep device for sudden unexpected death in infancy prevention. *J Paediatr Child Health*. 2018;54(6):638–646
 203. Baddock SA, Tipene-Leach D, Williams SM, et al. Physiological stability in an indigenous sleep device: a randomised controlled trial. *Arch Dis Child*. 2018;103(4):377–382
 204. Thompson EL, Moon RY. Hazard patterns associated with Co-sleepers. *Clin Pediatr (Phila)*. 2016;55(7):645–649
 205. Vege A, Rognum TO. Use of new Nordic criteria for classification of SIDS to re-evaluate diagnoses of sudden unexpected infant death in the Nordic countries. *Acta Paediatr*. 1997;86(4):391–396
 206. Heere M, Moughan B, Alfonsi J, Rodriguez J, Aronoff S. Effect of education and cardboard bassinet distribution on newborn bed-sharing. *Glob Pediatr Health*. 2019;6:2333794X19829173
 207. Ahlers-Schmidt CR, Schunn C, Redmond ML, et al. Qualitative assessment of pregnant women's perceptions of infant sleep boxes. *Glob Pediatr Health*. 2017;4:2333794X17744948
 208. Dalvie N, Nguyen V, Colson E, Loyal J. Mothers' perceptions of the cardboard box as a potential sleep space. *Acad Pediatr*. 2019;19(7):787–792
 209. Blair PS, Pease A, Bates F, et al. Concerns about the promotion of a cardboard baby box as a place for infants to sleep. *BMJ*. 2018;363:k4243
 210. Safe to Sleep campaign. Honor the past, learn for the future. what does a safe sleep environment look like? *NIH Pub*. 20-HD-7462. Washington, DC: U.S. Department of Health and Human Services; 2020
 211. Jackson A, Moon RY. An analysis of deaths in portable cribs and playpens: what can be learned? *Clin Pediatr (Phila)*. 2008;47(3):261–266
 212. Pike J, Moon RY. Bassinet use and sudden unexpected death in infancy. *J Pediatr*. 2008;153(4):509–512
 213. Callahan CW, Sisler C. Use of seating devices in infants too young to sit. *Arch Pediatr Adolesc Med*. 1997;151(3):233–235
 214. Orenstein SR, Whittington PF, Orenstein DM. The infant seat as treatment for

- gastroesophageal reflux. *N Engl J Med*. 1983;309(13):760–763
215. Hutchison BL, Thompson JM, Mitchell EA. Determinants of nonsynostotic plagiocephaly: a case-control study. *Pediatrics*. 2003;112(4):e316
 216. Bass JL, Bull M. Oxygen desaturation in term infants in car safety seats. *Pediatrics*. 2002;110(2 Pt 1):401–402
 217. Kornhauser Cerar L, Scirica CV, Stucin Gantar I, Osredkar D, Neubauer D, Kinane TB. A comparison of respiratory patterns in healthy term infants placed in car safety seats and beds. *Pediatrics*. 2009;124(3):e396–e402
 218. Côté A, Bairam A, Deschenes M, Hatza-kis G. Sudden infant deaths in sitting devices. *Arch Dis Child*. 2008;93(5):384–389
 219. Merchant JR, Worwa C, Porter S, Coleman JM, deRegnier RA. Respiratory instability of term and near-term healthy newborn infants in car safety seats. *Pediatrics*. 2001;108(3):647–652
 220. Willett LD, Leuschen MP, Nelson LS, Nelson RM Jr. Risk of hypoventilation in premature infants in car seats. *J Pediatr*. 1986;109(2):245–248
 221. Peachman RR. Fisher-Price Rock 'n Play sleeper should be recalled, Consumer Reports says. *Consum Rep*. 2019
 222. Batra EK, Midgett JD, Moon RY. Hazards associated with sitting and carrying devices for children two years and younger. *J Pediatr*. 2015;167(1):183–187
 223. Desapriya EB, Joshi P, Subzwari S, Nolan M. Infant injuries from child restraint safety seat misuse at British Columbia Children's Hospital. *Pediatr Int*. 2008;50(5):674–678
 224. Graham CJ, Kittredge D, Stuemy JH. Injuries associated with child safety seat misuse. *Pediatr Emerg Care*. 1992;8(6):351–353
 225. Parikh SN, Wilson L. Hazardous use of car seats outside the car in the United States, 2003-2007. *Pediatrics*. 2010;126(2):352–357
 226. Pollack-Nelson C. Fall and suffocation injuries associated with in-home use of car seats and baby carriers. *Pediatr Emerg Care*. 2000;16(2):77–79
 227. Wickham T, Abrahamson E. Head injuries in infants: the risks of bouncy chairs and car seats. *Arch Dis Child*. 2002;86(3):168–169
 228. Liaw P, Moon RY, Han A, Colvin JD. Infant deaths in sitting devices. *Pediatrics*. 2019;144(1):e20182576
 229. US Consumer Product Safety Commission. *Infant Deaths Prompt CPSC Warning About Sling Carriers for Babies*. Washington, DC: US Consumer Product Safety Commission; 2010
 230. Bergounioux J, Madre C, Crucis-Armengaud A, et al. Sudden deaths in adult-worn baby carriers: 19 cases. *Eur J Pediatr*. 2015;174(12):1665–1670
 231. Madre C, Rambaud C, Avran D, Michot C, Sachs P, Dauger S. Infant deaths in slings. *Eur J Pediatr*. 2014;173(12):1659–1661
 232. Ip S, Chung M, Raman G, Trikalinos TA, Lau J. A summary of the Agency for Healthcare Research and Quality's evidence report on breastfeeding in developed countries. *Breastfeed Med*. 2009;4(Suppl 1):S17–S30
 233. Vennemann MM, Bajanowski T, Brinkmann B, et al; GeSID Study Group. Does breastfeeding reduce the risk of sudden infant death syndrome? *Pediatrics*. 2009;123(3):e406–e410
 234. Hauck FR, Thompson JM, Tanabe KO, Moon RY, Vennemann MM. Breastfeeding and reduced risk of sudden infant death syndrome: a meta-analysis. *Pediatrics*. 2011;128(1):103–110
 235. Thompson JMD, Tanabe K, Moon RY, et al. Duration of breastfeeding and risk of SIDS: an individual participant data meta-analysis. *Pediatrics*. 2017;140(5):e20171324
 236. Maastrup R, Hansen BM, Kronborg H, et al. Breastfeeding progression in preterm infants is influenced by factors in infants, mothers and clinical practice: the results of a national cohort study with high breastfeeding initiation rates. *PLoS One*. 2014;9(9):e108208
 237. Blair PS, Platt MW, Smith IJ, Fleming PJ; CESDI SUDI Research Group. 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):101–106
 238. Franco P, Scaillet S, Wermenbol V, Valente F, Groswasser J, Kahn A. The influence of a pacifier on infants' arousals from sleep. *J Pediatr*. 2000;136(6):775–779
 239. Horne RS, Parslow PM, Ferens D, Watts AM, Adamson TM. Comparison of evoked arousability in breast and formula fed infants. *Arch Dis Child*. 2004;89(1):22–25
 240. Duijts L, Jaddoe VW, Hofman A, Moll HA. Prolonged and exclusive breastfeeding reduces the risk of infectious diseases in infancy. *Pediatrics*. 2010;126(1):e18–e25
 241. Heinig MJ. Host defense benefits of breastfeeding for the infant. effect of breastfeeding duration and exclusivity. *Pediatr Clin North Am*. 2001;48(1):105–123, ix
 242. Kramer MS, Guo T, Platt RW, et al. Infant growth and health outcomes associated with 3 compared with 6 mo of exclusive breastfeeding. *Am J Clin Nutr*. 2003;78(2):291–295
 243. Highet AR, Berry AM, Bettelheim KA, Goldwater PN. Gut microbiome in sudden infant death syndrome (SIDS) differs from that in healthy comparison babies and offers an explanation for the risk factor of prone position. *Int J Med Microbiol*. 2014;304(5–6):735–741
 244. McKenna JJ, Thoman EB, Anders TF, Sadeh A, Schechtman VL, Glotzbach SF. Infant-parent co-sleeping in an evolutionary perspective: implications for understanding infant sleep development and the sudden infant death syndrome. *Sleep*. 1993;16(3):263–282
 245. McKenna JJ, Ball HL, Gettler LT. Mother-infant cosleeping, breastfeeding and sudden infant death syndrome: what biological anthropology has discovered about normal infant sleep and pediatric sleep medicine. *Am J Phys Anthropol*. 2007;Suppl 45:133–161
 246. McKenna J. *Sleeping With Your Baby: A Parent's Guide to Cosleeping*. Washington, DC: Platypus Media, LLC; 2007
 247. Mitchell EA, Thompson JMD. Co-sleeping increases the risk of SIDS, but sleeping in the parents' bedroom lowers it. In: Rognum TO, ed. *Sudden Infant Death Syndrome: New Trends in the Nineties*. Oslo, Norway: Scandinavian University Press; 1995:266–269

248. Tappin D, Ecob R, Brooke H. Bedsharing, roomsharing, and sudden infant death syndrome in Scotland: a case-control study. *J Pediatr*. 2005; 147(1):32–37
249. Mitchell EA, Thompson JM, Zuccollo J, et al. The combination of bed sharing and maternal smoking leads to a greatly increased risk of sudden unexpected death in infancy: the New Zealand SUDI Nationwide Case Control Study. *N Z Med J*. 2017;130(1456): 52–64
250. Ward TC. Reasons for mother-infant bed-sharing: a systematic narrative synthesis of the literature and implications for future research. *Matern Child Health J*. 2015;19(3):675–690
251. Hauck FR, Signore C, Fein SB, Raju TN. Infant sleeping arrangements and practices during the first year of life. *Pediatrics*. 2008;122(Suppl 2): S113–S120
252. Joyner BL, Oden RP, Ajao TI, Moon RY. Where should my baby sleep: a qualitative study of African American infant sleep location decisions. *J Natl Med Assoc*. 2010;102(10):881–889
253. Flick L, White DK, Vemulapalli C, Stulac BB, Kemp JS. Sleep position and the use of soft bedding during bed sharing among African American infants at increased risk for sudden infant death syndrome. *J Pediatr*. 2001;138(3): 338–343
254. Mao A, Burnham MM, Goodlin-Jones BL, Gaylor EE, Anders TF. A comparison of the sleep-wake patterns of cosleeping and solitary-sleeping infants. *Child Psychiatry Hum Dev*. 2004;35(2):95–105
255. Volkovich E, Ben-Zion H, Karny D, Meiri G, Tikotzky L. Sleep patterns of co-sleeping and solitary sleeping infants and mothers: a longitudinal study. *Sleep Med*. 2015;16(11):1305–1312
256. Paul IM, Hohman EE, Loken E, et al. Mother-infant room-sharing and sleep outcomes in the INSIGHT study. *Pediatrics*. 2017;140(1):e20170122
257. Messayke S, Franco P, Forhan A, Dufour MN, Charles MA, Plancoulaine S. Sleep habits and sleep characteristics at age one year in the ELFE birth cohort study. *Sleep Med*. 2020;67: 200–206
258. McKenna JJ, Mosko SS, Richard CA. Bedsharing promotes breastfeeding. *Pediatrics*. 1997;100(2 Pt 1):214–219
259. Gettler LT, McKenna JJ. Evolutionary perspectives on mother-infant sleep proximity and breastfeeding in a laboratory setting. *Am J Phys Anthropol*. 2011;144(3):454–462
260. Raghunath BL, Azhari A, Bornstein MH, Setoh P, Esposito G. Experimental manipulation of maternal proximity during short sequences of sleep and infant calming response. *Infant Behav Dev*. 2020;59:101426
261. Hayes MJ, Roberts SM, Stowe R. Early childhood co-sleeping: parent-child and parent-infant nighttime interactions. *Infant Ment Health J*. 1996; 17(4):348–357
262. Okami P, Weisner T, Olmstead R. Outcome correlates of parent-child bed-sharing: an eighteen-year longitudinal study. *J Dev Behav Pediatr*. 2002; 23(4):244–253
263. Lerner RE, Camerota M, Tully KP, Propert C. Associations between mother-infant bed-sharing practices and infant affect and behavior during the still-face paradigm. *Infant Behav Dev*. 2020;60:101464
264. Mileva-Seitz VR, Luijk MP, van Ijzendoorn MH, et al. Association between infant nighttime-sleep location and attachment security: no easy verdict. *Infant Ment Health J*. 2016;37(1):5–16
265. Bilgin A, Wolke D. Bed-sharing in the first 6 months: associations with infant-mother attachment, infant attention, maternal bonding, and sensitivity at 18 months. *J Dev Behav Pediatr*. 2021;43(1):e9–e19
266. Santos IS, Barros AJ, Barros FC, Munhoz TN, Da Silva BDP, Matijasevich A. Mother-child bed-sharing trajectories and psychiatric disorders at the age of 6 years. *J Affect Disord*. 2017; 208:163–169
267. Shimizu M, Teti DM. Infant sleeping arrangements, social criticism, and maternal distress in the first year. *Infant Child Dev*. 2018;27(3):e2080
268. Beijers R, Cassidy J, Lustermaans H, de Weerth C. Parent-infant room sharing during the first months of life: longitudinal links with behavior during middle childhood. *Child Dev*. 2019;90(4):1350–1367
269. St James-Roberts I, Roberts M, Hovish K, Owen C. Descriptive figures for differences in parenting and infant nighttime distress in the first three months of age. *Prim Health Care Res Dev*. 2016;17(6):611–621
270. Scheers NJ, Dayton CM, Kemp JS. Sudden infant death with external airways covered: case-comparison study of 206 deaths in the United States. *Arch Pediatr Adolesc Med*. 1998;152(6): 540–547
271. Unger B, Kemp JS, Wilkins D, et al. Racial disparity and modifiable risk factors among infants dying suddenly and unexpectedly. *Pediatrics*. 2003;111(2):E127–E131
272. Kemp JS, Unger B, Wilkins D, et al. Unsafe sleep practices and an analysis of bedsharing among infants dying suddenly and unexpectedly: results of a four-year, population-based, death-scene investigation study of sudden infant death syndrome and related deaths. *Pediatrics*. 2000;106(3):E41
273. Drago DA, Dannenberg AL. Infant mechanical suffocation deaths in the United States, 1980-1997. *Pediatrics*. 1999;103(5):e59
274. Blair PS, Mitchell EA, Heckstall-Smith EM, Fleming PJ. Head covering - a major modifiable risk factor for sudden infant death syndrome: a systematic review. *Arch Dis Child*. 2008;93(9): 778–783
275. Baddock SA, Galland BC, Bolton DP, Williams SM, Taylor BJ. Differences in infant and parent behaviors during routine bed sharing compared with cot sleeping in the home setting. *Pediatrics*. 2006;117(5):1599–1607
276. Baddock SA, Galland BC, Taylor BJ, Bolton DP. Sleep arrangements and behavior of bed-sharing families in the home setting. *Pediatrics*. 2007;119(1): e200–e207
277. Ball H. Airway covering during bed-sharing. *Child Care Health Dev*. 2009;35(5):728–737
278. Kattwinkel J, Brooks J, Keenan ME, Malloy MH; Task Force on Infant Sleep Position and Sudden Infant Death Syndrome; American Academy of Pediatrics. Changing concepts of sudden

- infant death syndrome: implications for infant sleeping environment and sleep position. *Pediatrics*. 2000;105(3 Pt 1):650–656
279. 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
 280. Ostfeld BM, Perl H, Esposito L, et al. Sleep environment, positional, lifestyle, and demographic characteristics associated with bed sharing in sudden infant death syndrome cases: a population-based study. *Pediatrics*. 2006;118(5):2051–2059
 281. Scheers NJ, Rutherford GW, Kemp JS. Where should infants sleep? a comparison of risk for suffocation of infants sleeping in cribs, adult beds, and other sleeping locations. *Pediatrics*. 2003;112(4):883–889
 282. Ruys JH, de Jonge GA, Brand R, Engelberts AC, Semmekrot BA. Bed-sharing in the first four months of life: a risk factor for sudden infant death. *Acta Paediatr*. 2007;96(10):1399–1403
 283. Blair PS, Sidebotham P, Evason-Coombe C, Edmonds M, Heckstall-Smith EM, Fleming P. Hazardous co-sleeping environments and risk factors amenable to change: case-control study of SIDS in south west England. *BMJ*. 2009;339:b3666
 284. Rechtman LR, Colvin JD, Blair PS, Moon RY. Sofas and infant mortality. *Pediatrics*. 2014;134(5):e1293–e1300
 285. Salm Ward TC, Ngui EM. Factors associated with bed-sharing for African American and white mothers in Wisconsin. *Matern Child Health J*. 2015;19(4):720–732
 286. Bartick M, Smith LJ. Speaking out on safe sleep: evidence-based infant sleep recommendations. *Breastfeed Med*. 2014;9(9):417–422
 287. Bailey C, Tawia S, McGuire E. Breastfeeding duration and infant sleep location in a cohort of volunteer breastfeeding counselors. *J Hum Lact*. 2020;36(2):354–364
 288. Bovbjerg ML, Hill JA, Uphoff AE, Rosenberg KD. Women who bedshare more frequently at 14 weeks postpartum subsequently report longer durations of breastfeeding. *J Midwifery Womens Health*. 2018;63(4):418–424
 289. Horsley T, Clifford T, Barrowman N, et al. Benefits and harms associated with the practice of bed sharing: a systematic review. *Arch Pediatr Adolesc Med*. 2007;161(3):237–245
 290. Huang Y, Hauck FR, Signore C, et al. Influence of bedsharing activity on breastfeeding duration among US mothers. *JAMA Pediatr*. 2013;167(11):1038–1044
 291. Smith LA, Geller NL, Kellams AL, et al. Infant sleep location and breastfeeding practices in the United States, 2011–2014. *Acad Pediatr*. 2016;16(6):540–549
 292. Ball HL, Howel D, Bryant A, Best E, Russell C, Ward-Platt M. Bed-sharing by breastfeeding mothers: who bedshares and what is the relationship with breastfeeding duration? *Acta Paediatr*. 2016;105(6):628–634
 293. Scragg R, Mitchell EA, Taylor BJ, et al; New Zealand Cot Death Study Group. Bed sharing, smoking, and alcohol in the sudden infant death syndrome. *BMJ*. 1993;307(6915):1312–1318
 294. McGarvey C, McDonnell M, Chong A, O'Regan M, Matthews T. Factors relating to the infant's last sleep environment in sudden infant death syndrome in the Republic of Ireland. *Arch Dis Child*. 2003;88(12):1058–1064
 295. Blair PS, Sidebotham P, Pease A, Fleming PJ. Bed-sharing in the absence of hazardous circumstances: is there a risk of sudden infant death syndrome? an analysis from two case-control studies conducted in the UK. *PLoS One*. 2014;9(9):e107799
 296. Kendall-Tackett K, Cong Z, Hale TW. Mother-infant sleep locations and nighttime feeding behavior: U.S. data from the Survey of Mothers' Sleep and Fatigue. *Clinical Lactation*. 2010;1(Fall):27–31
 297. Carpenter R, McGarvey C, Mitchell EA, et al. Bed sharing when parents do not smoke: is there a risk of SIDS? 7n individual level analysis of five major case-control studies. *BMJ Open*. 2013;3(5):e002299
 298. Arnestad M, Andersen M, Vege A, Rognum TO. Changes in the epidemiological pattern of sudden infant death syndrome in southeast Norway, 1984–1998: implications for future prevention and research. *Arch Dis Child*. 2001;85(2):108–115
 299. McGarvey C, McDonnell M, Hamilton K, O'Regan M, Matthews T. An 8 year study of risk factors for SIDS: bed-sharing versus non-bed-sharing. *Arch Dis Child*. 2006;91(4):318–323
 300. Fu LY, Moon RY, Hauck FR. Bed sharing among black infants and sudden infant death syndrome: interactions with other known risk factors. *Acad Pediatr*. 2010;10(6):376–382
 301. Carroll-Pankhurst C, Mortimer EAJ Jr. Sudden infant death syndrome, bedsharing, parental weight, and age at death. *Pediatrics*. 2001;107(3):530–536
 302. Mitchell E, Thompson J. Who cosleeps? does high maternal body weight and duvet use increase the risk of sudden infant death syndrome when bed sharing? *Paediatr Child Health* 2006; 11(Suppl A):14A–15A
 303. Fleming PJ, Gilbert R, Azaz Y, et al. Interaction between bedding and sleeping position in the sudden infant death syndrome: a population based case-control study. *BMJ*. 1990; 301(6743):85–89
 304. Ponsonby A-L, Dwyer T, Gibbons LE, Cochrane JA, Jones ME, McCall MJ. Thermal environment and sudden infant death syndrome: case-control study. *BMJ*. 1992;304(6822):277–282
 305. Ponsonby A-L, Dwyer T, Gibbons LE, Cochrane JA, Wang Y-G. Factors potentiating the risk of sudden infant death syndrome associated with the prone position. *N Engl J Med*. 1993;329(6):377–382
 306. Iyasu S, Randall LL, Welty TK, et al. Risk factors for sudden infant death syndrome among northern plains Indians. *JAMA*. 2002;288(21):2717–2723
 307. Hutchison BL, Stewart AW, Mitchell EA. The prevalence of cobedding and SIDS-related child care practices in twins. *Eur J Pediatr*. 2010;169(12):1477–1485
 308. Hayward K. Cobedding of twins: a natural extension of the socialization process? *MCN Am J Matern Child Nurs*. 2003;28(4):260–263

309. Tomashek KM, Wallman C; Committee on Fetus and Newborn, American Academy of Pediatrics. Cofbedding twins and higher-order multiples in a hospital setting. *Pediatrics*. 2007;120(6):1359–1366
310. National Association of Neonatal Nurses Board of Directors. Cofbedding of twins or higher-order multiples, NANN position statement #3045. *Adv Neonatal Care*. 2008;9(6):307–313
311. Chiodini BA, Thach BT. Impaired ventilation in infants sleeping facedown: potential significance for sudden infant death syndrome. *J Pediatr*. 1993;123(5):686–692
312. Sakai J, Kanetake J, Takahashi S, Kanawaku Y, Funayama M. Gas dispersal potential of bedding as a cause for sudden infant death. *Forensic Sci Int*. 2008;180(2–3):93–97
313. 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–e219
314. Shapiro-Mendoza CK, Colson ER, Willinger M, Rybin DV, Camperlengo L, Corwin MJ. Trends in infant bedding use: National Infant Sleep Position study, 1993–2010. *Pediatrics*. 2015;135(1):10–17
315. Ponsonby A-L, Dwyer T, Couper D, Cochrane J. Association between use of a quilt and sudden infant death syndrome: case-control study. *BMJ*. 1998;316(7126):195–196
316. Mitchell EA, Scragg L, Clements M. Soft cot mattresses and the sudden infant death syndrome. *N Z Med J*. 1996;109(1023):206–207
317. Mitchell EA, Thompson JMD, Ford RPK, Taylor BJ; New Zealand Cot Death Study Group. Sheepskin bedding and the sudden infant death syndrome. *J Pediatr*. 1998;133(5):701–704
318. Kemp JS, Kowalski RM, Burch PM, Graham MA, Thach BT. Unintentional suffocation by rebreathing: a death scene and physiologic investigation of a possible cause of sudden infant death. *J Pediatr*. 1993;122(6):874–880
319. Brooke H, Gibson A, Tappin D, Brown H. Case-control study of sudden infant death syndrome in Scotland, 1992–5. *BMJ*. 1997;314(7093):1516–1520
320. Gaw CE, Chounthirath T, Midgett J, Quinlan K, Smith GA. Types of objects in the sleep environment associated with infant suffocation and strangulation. *Acad Pediatr*. 2017;17(8):893–901
321. Wilson CA, Taylor BJ, Laing RM, Williams SM, Mitchell EA. Clothing and bedding and its relevance to sudden infant death syndrome: further results from the New Zealand Cot Death Study. *J Paediatr Child Health*. 1994;30(6):506–512
322. Markestad T, Skadberg B, Hordvik E, Morild I, Irgens LM. Sleeping position and sudden infant death syndrome (SIDS): effect of an intervention programme to avoid prone sleeping. *Acta Paediatr*. 1995;84(4):375–378
323. L'Hoir MP, Engelberts AC, van Well GTJ, et al. Risk and preventive factors for cot death in The Netherlands, a low-incidence country. *Eur J Pediatr*. 1998;157(8):681–688
324. Beal SM, Byard RW. Accidental death or sudden infant death syndrome? *J Paediatr Child Health*. 1995;31(4):269–271
325. Schlaud M, Dreier M, Debertin AS, et al. The German case-control scene investigation study on SIDS: epidemiological approach and main results. *Int J Legal Med*. 2010;124(1):19–26
326. Chowdhury RT. *Nursery Product-related Injuries and Deaths among Children under Age Five*. Washington, DC: U.S. Consumer Product Safety Commission; 2017
327. Summe V, Baker RB, Eichel MM. Safety, feasibility, and effectiveness of weighted blankets in the care of infants with neonatal abstinence syndrome: a crossover randomized controlled trial. *Adv Neonatal Care*. 2020;20(5):384–391
328. Ajao TI, Oden RP, Joyner BL, Moon RY. Decisions of black parents about infant bedding and sleep surfaces: a qualitative study. *Pediatrics*. 2011;128(3):494–502
329. 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
330. Joyner BL, Gill-Bailey C, Moon RY. Infant sleep environments depicted in magazines targeted to women of childbearing age. *Pediatrics*. 2009;124(3):e416–e422
331. Goodstein MH, Lagon E, Bell T, Joyner BL, Moon RY. Stock photographs do not comply with infant safe sleep guidelines. *Clin Pediatr (Phila)*. 2018;57(4):403–409
332. Moon RY. “And things that go bump in the night”: nothing to fear? *J Pediatr*. 2007;151(3):237–238
333. Thach BT, Rutherford GW Jr, Harris K. Deaths and injuries attributed to infant crib bumper pads. *J Pediatr*. 2007;151(3):271–274, 274.e1–274.e3
334. Wanna-Nakamura S. *White Paper-Unsafe Sleep Settings: Hazards Associated With the Infant Sleep Environment and Unsafe Practices Used by Caregivers: A CPSC Staff Perspective*. Bethesda, MD: US Consumer Product Safety Commission; 2010.
335. U.S. Consumer Product Safety Commission. *Staff Briefing Package, Crib Bumpers Petition*. Washington, DC: U.S. Consumer Product Safety Commission; 2013
336. 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
337. Yeh ES, Rochette LM, McKenzie LB, Smith GA. Injuries associated with cribs, playpens, and bassinets among young children in the US, 1990–2008. *Pediatrics*. 2011;127(3):479–486
338. Tappin D, Brooke H, Ecob R, Gibson A. Used infant mattresses and sudden infant death syndrome in Scotland: case-control study. *BMJ*. 2002;325(7371):1007–1012
339. Arnestad M, Andersen M, Rognum TO. Is the use of dummy or carry-cot of importance for sudden infant death? *Eur J Pediatr*. 1997;156(12):968–970
340. Mitchell EA, Taylor BJ, Ford RPK, et al. Dummies and the sudden infant death syndrome. *Arch Dis Child*. 1993;68(4):501–504
341. Fleming PJ, Blair PS, Pollard K, et al; CESDI SUDI Research Team. Pacifier use and sudden infant death syndrome: results from the CESDI/SUDI

- case control study. *Arch Dis Child*. 1999;81(2):112–116
342. L'Hoir MP, Engelberts AC, van Well GTJ, et al. Dummy use, thumb sucking, mouth breathing and cot death. *Eur J Pediatr*. 1999;158(11):896–901
 343. Li DK, Willinger M, Petitti DB, Odouli R, Liu L, Hoffman HJ. Use of a dummy (pacifier) during sleep and risk of sudden infant death syndrome (SIDS): population based case-control study. *BMJ*. 2006;332(7532):18–22
 344. Vennemann MM, Bajanowski T, Brinkmann B, Jorch G, Sauerland C, Mitchell EA; GeSID Study Group. Sleep environment risk factors for sudden infant death syndrome: the German Sudden Infant Death Syndrome Study. *Pediatrics*. 2009;123(4):1162–1170
 345. Hauck FR, Omojokun OO, Siadaty MS. Do pacifiers reduce the risk of sudden infant death syndrome? a meta-analysis. *Pediatrics*. 2005;116(5):e716–e723
 346. Mitchell EA, Blair PS, L'Hoir MP. Should pacifiers be recommended to prevent SIDS? *Pediatrics*. 2006;117(5):1755–1758
 347. Moon RY, Tanabe KO, Yang DC, Young HA, Hauck FR. Pacifier use and SIDS: evidence for a consistently reduced risk. *Matern Child Health J*. 2012;16(3):609–614
 348. Franco P, Chabanski S, Scaillet S, Grosswasser J, Kahn A. Pacifier use modifies infant's cardiac autonomic controls during sleep. *Early Hum Dev*. 2004;77(1–2):99–108
 349. Horne RS, Fyfe KL, Odoi A, Athukorala A, Yiallourou SR, Wong FY. Dummy/pacifier use in preterm infants increases blood pressure and improves heart rate control. *Pediatr Res*. 2016;79(2):325–332
 350. Foster JP, Psaila K, Patterson T. Non-nutritive sucking for increasing physiologic stability and nutrition in preterm infants. *Cochrane Database Syst Rev*. 2016;10(10):CD001071
 351. Tonkin SL, Lui D, McIntosh CG, Rowley S, Knight DB, Gunn AJ. Effect of pacifier use on mandibular position in preterm infants. *Acta Paediatr*. 2007;96(10):1433–1436
 352. Hanzer M, Zotter H, Sauseng W, Pfurtscheller K, Müller W, Kerbl R. Pacifier use does not alter the frequency or duration of spontaneous arousals in sleeping infants. *Sleep Med*. 2009;10(4):464–470
 353. Odoi A, Andrew S, Wong FY, Yiallourou SR, Horne RS. Pacifier use does not alter sleep and spontaneous arousal patterns in healthy term-born infants. *Acta Paediatr*. 2014;103(12):1244–1250
 354. Weiss PP, Kerbl R. The relatively short duration that a child retains a pacifier in the mouth during sleep: implications for sudden infant death syndrome. *Eur J Pediatr*. 2001;160(1):60–70
 355. Gartner LM, Morton J, Lawrence RA, et al; American Academy of Pediatrics Section on Breastfeeding. Breastfeeding and the use of human milk. *Pediatrics*. 2005;115(2):496–506
 356. Feldman-Winter L, Kellams A, Peter-Wohl S, et al. Evidence-based updates on the first week of exclusive breastfeeding among infants ≥ 35 weeks. *Pediatrics*. 2020;145(4):e20183696
 357. Over Veiligheid NL. Safe sleeping for your baby. Available at: www.wiegedood.nl/files/download_vs_engels.pdf. Accessed June 1, 2022
 358. Factfile 2. Research background to the reduce the risk of cot death advice by the Foundation for the Study of Infant Deaths. Available at: www.cotmattress.net/SIDS-Guidelines.pdf. Accessed January 10, 2016
 359. Canadian Paediatric Society, Community Paediatrics Committee. Recommendations for the use of pacifiers. *Paediatr Child Health*. 2003;8(8):515–528
 360. Aarts C, Hörnell A, Kylberg E, Hofvander Y, Gebre-Medhin M. Breastfeeding patterns in relation to thumb sucking and pacifier use. *Pediatrics*. 1999;104(4):e50
 361. Benis MM. Are pacifiers associated with early weaning from breastfeeding? *Adv Neonatal Care*. 2002;2(5):259–266
 362. Scott JA, Binns CW, Oddy WH, Graham KI. Predictors of breastfeeding duration: evidence from a cohort study. *Pediatrics*. 2006;117(4):e646–e655
 363. Jaafar SH, Ho JJ, Jahanfar S, Angolkar M. Effect of restricted pacifier use in breastfeeding term infants for increasing duration of breastfeeding. *Cochrane Database Syst Rev*. 2016;(8):CD007202
 364. Kaya V, Aytekin A. Effects of pacifier use on transition to full breastfeeding and sucking skills in preterm infants: a randomised controlled trial. *J Clin Nurs*. 2017;26(13–14):2055–2063
 365. O'Connor NR, Tanabe KO, Siadaty MS, Hauck FR. Pacifiers and breastfeeding: a systematic review. *Arch Pediatr Adolesc Med*. 2009;163(4):378–382
 366. Buccini GDS, Pérez-Escamilla R, Paulino LM, Araújo CL, Venancio SI. Pacifier use and interruption of exclusive breastfeeding: systematic review and meta-analysis. *Matern Child Nutr*. 2017;13(3):e12384
 367. Alm B, Wennergren G, Möllborg P, Lagercrantz H. Breastfeeding and dummy use have a protective effect on sudden infant death syndrome. *Acta Paediatr*. 2016;105(1):31–38
 368. Howard CR, Howard FM, Lanphear B, et al. Randomized clinical trial of pacifier use and bottle-feeding or cupfeeding and their effect on breastfeeding. *Pediatrics*. 2003;111(3):511–518
 369. Eidelman AI, Schanler RJ; Section on Breastfeeding. Breastfeeding and the use of human milk. *Pediatrics*. 2012;129(3):e827–e841
 370. Larsson E. The effect of dummy-sucking on the occlusion: a review. *Eur J Orthod*. 1986;8(2):127–130
 371. American Academy of Pediatric Dentistry, Council on Clinical Affairs. Policy statement on oral habits. Available at: www.aapd.org/assets/news/upload/2003/270.pdf. Accessed June 1, 2022
 372. Niemelä M, Uhari M, Möttönen M. A pacifier increases the risk of recurrent acute otitis media in children in day care centers. *Pediatrics*. 1995;96(5 Pt 1):884–888
 373. Niemelä M, Pihakari O, Pokka T, Uhari M. Pacifier as a risk factor for acute otitis media: a randomized, controlled trial of parental counseling. *Pediatrics*. 2000;106(3):483–488

374. Jackson JM, Mourino AP. Pacifier use and otitis media in infants twelve months of age or younger. *Pediatr Dent*. 1999;21(4):255–260
375. Daly KA, Giebink GS. Clinical epidemiology of otitis media. *Pediatr Infect Dis J*. 2000;19(5 Suppl):S31–S36
376. Darwazeh AM, al-Bashir A. Oral candidal flora in healthy infants. *J Oral Pathol Med*. 1995;24(8):361–364
377. North K, Fleming P, Golding J. Pacifier use and morbidity in the first six months of life. *Pediatrics*. 1999;103(3):E34
378. Niemelä M, Uhari M, Hannuksela A. Pacifiers and dental structure as risk factors for otitis media. *Int J Pediatr Otorhinolaryngol*. 1994;29(2):121–127
379. Uhari M, Mäntysaari K, Niemelä M. A meta-analytic review of the risk factors for acute otitis media. *Clin Infect Dis*. 1996;22(6):1079–1083
380. CPSC Safety Alert: *Strings, Cords and Necklaces Can Strangle Infants*. Washington, DC: U.S. Consumer Product Safety Commission
381. Kraus JF, Greenland S, Bulterys M. Risk factors for sudden infant death syndrome in the US Collaborative Perinatal Project. *Int J Epidemiol*. 1989;18(1):113–120
382. Paris CA, Remler R, Daling JR. Risk factors for sudden infant death syndrome: changes associated with sleep position recommendations. *J Pediatr*. 2001;139(6):771–777
383. Stewart AJ, Williams SM, Mitchell EA, Taylor BJ, Ford RP, Allen EM. Antenatal and intrapartum factors associated with sudden infant death syndrome in the New Zealand Cot Death Study. *J Paediatr Child Health*. 1995;31(5):473–478
384. American Academy of Pediatrics Committee on Fetus and Newborn and ACOG Committee on Obstetric Practice. *Guidelines for Perinatal Care*, 7th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2012
385. Sontag JM, Singh B, Ostfeld BM, Hegyi T, Steinberg MB, Delnevo CD. Obstetricians' and gynecologists' communication practices around smoking cessation in pregnancy, secondhand smoke and sudden infant death syndrome (SIDS): a survey. *Int J Environ Res Public Health*. 2020;17(8):E2908
386. 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):249–257
387. Schoendorf KC, Kiely JL. Relationship of sudden infant death syndrome to maternal smoking during and after pregnancy. *Pediatrics*. 1992;90(6):905–908
388. Malloy MH, Kleinman JC, Land GH, Schramm WF. The association of maternal smoking with age and cause of infant death. *Am J Epidemiol*. 1988;128(1):46–55
389. Haglund B, Cnattingius S. Cigarette smoking as a risk factor for sudden infant death syndrome: a population-based study. *Am J Public Health*. 1990;80(1):29–32
390. Mitchell EA, Ford RP, Stewart AW, et al. Smoking and the sudden infant death syndrome. *Pediatrics*. 1993;91(5):893–896
391. Winickoff JP, Friebely J, Tanski SE, et al. Beliefs about the health effects of “thirdhand” smoke and home smoking bans. *Pediatrics*. 2009;123(1):e74–e79
392. Tirosh E, Libon D, Bader D. The effect of maternal smoking during pregnancy on sleep respiratory and arousal patterns in neonates. *J Perinatol*. 1996;16(6):435–438
393. Franco P, Groswasser J, Hassid S, Lanquart JP, Scailliet S, Kahn A. Prenatal exposure to cigarette smoking is associated with a decrease in arousal in infants. *J Pediatr*. 1999;135(1):34–38
394. Horne RS, Ferens D, Watts AM, et al. Effects of maternal tobacco smoking, sleeping position, and sleep state on arousal in healthy term infants. *Arch Dis Child Fetal Neonatal Ed*. 2002;87(2):F100–F105
395. Sawnani H, Jackson T, Murphy T, Beckerman R, Simakajornboon N. The effect of maternal smoking on respiratory and arousal patterns in preterm infants during sleep. *Am J Respir Crit Care Med*. 2004;169(6):733–738
396. Lewis KW, Bosque EM. Deficient hypoxia awakening response in infants of smoking mothers: possible relationship to sudden infant death syndrome. *J Pediatr*. 1995;127(5):691–699
397. Chang AB, Wilson SJ, Masters IB, et al. Altered arousal response in infants exposed to cigarette smoke. *Arch Dis Child*. 2003;88(1):30–33
398. Parslow PM, Cranage SM, Adamson TM, Harding R, Horne RS. Arousal and ventilatory responses to hypoxia in sleeping infants: effects of maternal smoking. *Respir Physiol Neurobiol*. 2004;140(1):77–87
399. Anderson TM, Lavista Ferres JM, Ren SY, et al. Maternal smoking before and during pregnancy and the risk of sudden unexpected infant death. *Pediatrics*. 2019;143(4):e20183325
400. Zhang K, Wang X. Maternal smoking and increased risk of sudden infant death syndrome: a meta-analysis. *Leg Med (Tokyo)*. 2013;15(3):115–121
401. Mitchell EA, Milerad J. Smoking and the sudden infant death syndrome. *Rev Environ Health*. 2006;21(2):81–103
402. Dietz PM, England LJ, Shapiro-Mendoza CK, Tong VT, Farr SL, Callaghan WM. Infant morbidity and mortality attributable to prenatal smoking in the U.S. *Am J Prev Med*. 2010;39(1):45–52
403. Farber HJ, Walley SC, Groner JA, Nelson KE; Section on Tobacco Control. Clinical practice policy to protect children from tobacco, nicotine, and tobacco smoke. *Pediatrics*. 2015;136(5):1008–1017
404. O'Leary CM, Jacoby PJ, Bartu A, D'Antoine H, Bower C. Maternal alcohol use and sudden infant death syndrome and infant mortality excluding SIDS. *Pediatrics*. 2013;131(3):e770–e778
405. Strandberg-Larsen K, Grønboek M, Andersen AM, Andersen PK, Olsen J. Alcohol drinking pattern during pregnancy and risk of infant mortality. *Epidemiology*. 2009;20(6):884–891
406. Elliott AJ, Kinney HC, Haynes RL, et al. Concurrent prenatal drinking and smoking increases risk for SIDS: Safe Passage Study report. *EClinicalMedicine*. 2020;19:100247

407. Sirieix CM, Tobia CM, Schneider RW, Darnall RA. Impaired arousal in rat pups with prenatal alcohol exposure is modulated by GABAergic mechanisms. *Physiol Rep*. 2015;3(6):e12424
408. Alm B, Wennergren G, Norvenius G, et al. Caffeine and alcohol as risk factors for sudden infant death syndrome. Nordic Epidemiological SIDS Study. *Arch Dis Child*. 1999;81(2):107–111
409. James C, Klenka H, Manning D. Sudden infant death syndrome: bed sharing with mothers who smoke. *Arch Dis Child*. 2003;88(2):112–113
410. Williams SM, Mitchell EA, Taylor BJ. Are risk factors for sudden infant death syndrome different at night? *Arch Dis Child*. 2002;87(4):274–278
411. Rajegowda BK, Kandall SR, Falciglia H. Sudden unexpected death in infants of narcotic-dependent mothers. *Early Hum Dev*. 1978;2(3):219–225
412. Chavez CJ, Ostrea EM Jr, Stryker JC, Smialek Z. Sudden infant death syndrome among infants of drug-dependent mothers. *J Pediatr*. 1979;95(3):407–409
413. Bauchner H, Zuckerman B, McClain M, Frank D, Fried LE, Kayne H. Risk of sudden infant death syndrome among infants with in utero exposure to cocaine. *J Pediatr*. 1988;113(5):831–834
414. Durand DJ, Espinoza AM, Nickerson BG. Association between prenatal cocaine exposure and sudden infant death syndrome. *J Pediatr*. 1990;117(6):909–911
415. Ward SL, Bautista D, Chan L, et al. Sudden infant death syndrome in infants of substance-abusing mothers. *J Pediatr*. 1990;117(6):876–881
416. Rosen TS, Johnson HL. Drug-addicted mothers, their infants, and SIDS. *Ann N Y Acad Sci*. 1988;533:89–95
417. Kandall SR, Gaines J, Habel L, Davidson G, Jessop D. Relationship of maternal substance abuse to subsequent sudden infant death syndrome in offspring. *J Pediatr*. 1993;123(1):120–126
418. Fares I, McCulloch KM, Raju TN. Intrauterine cocaine exposure and the risk for sudden infant death syndrome: a meta-analysis. *J Perinatol*. 1997;17(3):179–182
419. Fulmer M, Zachritz W, Posencheg MA. Intensive care neonates and evidence to support the elimination of hats for safe sleep. *Adv Neonatal Care*. 2020;20(3):229–232
420. Waldhoer T, Heinzl H. Exploring the possible relationship between ambient heat and sudden infant death with data from Vienna, Austria. *PLoS One*. 2017;12(9):e0184312
421. Basu R, Pearson D, Sie L, Broadwin R. A case-crossover study of temperature and infant mortality in California. *Paediatr Perinat Epidemiol*. 2015;29(5):407–415
422. Scheers-Masters JR, Schootman M, Thach BT. Heat stress and sudden infant death syndrome incidence: a United States population epidemiologic study. *Pediatrics*. 2004;113(6):e586–e592
423. Leiss JK, Suchindran CM. Sudden infant death syndrome and local meteorologic temperature in North Carolina. *Am J Epidemiol*. 1996;144(2):111–115
424. Chang HP, Li CY, Chang YH, Hwang SL, Su YH, Chen CW. Sociodemographic and meteorological correlates of sudden infant death in Taiwan. *Pediatr Int*. 2013;55(1):11–16
425. Auger N, Fraser WD, Smargiassi A, Kotsatsky T. Ambient heat and sudden infant death: a case-crossover study spanning 30 years in Montreal, Canada. *Environ Health Perspect*. 2015;123(7):712–716
426. Jhun I, Mata DA, Nordio F, Lee M, Schwartz J, Zanobetti A. Ambient temperature and sudden infant death syndrome in the United States. *Epidemiology*. 2017;28(5):728–734
427. Son JY, Lee JT, Bell ML. Is ambient temperature associated with risk of infant mortality? a multi-city study in Korea. *Environ Res*. 2017;158:748–752
428. Mitchell EA, Stewart AW, Cowan SF. Sudden infant death syndrome and weather temperature. *Paediatr Perinat Epidemiol*. 1992;6(1):19–28
429. Itzhak N, Greenblatt D. Aerodynamic factors affecting rebreathing in infants. *J Appl Physiol (1985)*. 2019;126(4):952–964
430. Ponsonby AL, Dwyer T, Kasl SV, Cochrane JA. The Tasmanian SIDS Case-Control Study: univariable and multivariable risk factor analysis. *Paediatr Perinat Epidemiol*. 1995;9(3):256–272
431. McGlashan ND. Sudden infant deaths in Tasmania, 1980-1986: a seven year prospective study. *Soc Sci Med*. 1989;29(8):1015–1026
432. Coleman-Phox K, Odouli R, Li DK. Use of a fan during sleep and the risk of sudden infant death syndrome. *Arch Pediatr Adolesc Med*. 2008;162(10):963–968
433. Hutcheson R. DTP vaccination and sudden infant deaths - Tennessee. *MMWR Morb Mortal Wkly Rep*. 1979;28:131–132
434. Hutcheson R. Follow-up on DTP vaccination and sudden infant deaths - Tennessee. *MMWR Morb Mortal Wkly Rep*. 1979;28:134–135
435. Bernier RH, Frank JA Jr, Dondero TJ Jr, Turner P. Diphtheria-tetanus toxoids-pertussis vaccination and sudden infant deaths in Tennessee. *J Pediatr*. 1982;101(3):419–421
436. Baraff LJ, Ablon WJ, Weiss RC. Possible temporal association between diphtheria-tetanus toxoid-pertussis vaccination and sudden infant death syndrome. *Pediatr Infect Dis*. 1983;2(1):7–11
437. Griffin MR, Ray WA, Livengood JR, Schaffner W. Risk of sudden infant death syndrome after immunization with the diphtheria-tetanus-pertussis vaccine. *N Engl J Med*. 1988;319(10):618–623
438. Hoffman HJ, Hunter JC, Damus K, et al. Diphtheria-tetanus-pertussis immunization and sudden infant death: results of the National Institute of Child Health and Human Development Cooperative Epidemiological study of sudden infant death syndrome risk factors. *Pediatrics*. 1987;79(4):598–611
439. Taylor EM, Emergy JL. Immunization and cot deaths. *Lancet*. 1982;2(8300):721
440. Flahault A, Messiah A, Jouglu E, Bouvet E, Perin J, Hattori F. Sudden infant death syndrome and diphtheria/tetanus toxoid/pertussis/poliomyelitis immunisation. *Lancet*. 1988;1(8585):582–583

441. Walker AM, Jick H, Perera DR, Thompson RS, Knauss TA. Diphtheria-tetanus-pertussis immunization and sudden infant death syndrome. *Am J Public Health*. 1987;77(8):945–951
442. Jonville-Bera AP, Autret E, Laugier J. Sudden infant death syndrome and diphtheria-tetanus-pertussis-poliomyelitis vaccination status. *Fundam Clin Pharmacol*. 1995;9(3):263–270
443. Stratton K, Almario DA, Wizemann TM, McCormick MC, eds. *Immunization Safety Review Committee, Immunization Safety Review: Vaccinations and Sudden Unexpected Death in Infancy*. Washington, DC: National Academies Press; 2003
444. Miller ER, Moro PL, Cano M, Shimabukuro TT. Deaths following vaccination: what does the evidence show? *Vaccine*. 2015;33(29):3288–3292
445. Moro PL, Arana J, Cano M, Lewis P, Shimabukuro TT. Deaths reported to the vaccine adverse event reporting system, United States, 1997-2013. *Clin Infect Dis*. 2015;61(6):980–987
446. Moro PL, Jankosky C, Menschik D, et al. Adverse events following haemophilus influenzae type b vaccines in the Vaccine Adverse Event Reporting System, 1990-2013. *J Pediatr*. 2015;166(4):992–997
447. Iqbal S, Shi J, Seib K, et al. Preparation for global introduction of inactivated poliovirus vaccine: safety evidence from the US Vaccine Adverse Event Reporting System, 2000-12. *Lancet Infect Dis*. 2015;15(10):1175–1182
448. Mitchell EA, Stewart AW, Clements M, Ford RPK; New Zealand Cot Death Study Group. Immunisation and the sudden infant death syndrome. *Arch Dis Child*. 1995;73(6):498–501
449. Jonville-Béra AP, Autret-Leca E, Barbeil-F, Paris-Llado J; French Reference Centers for SIDS. Sudden unexpected death in infants under 3 months of age and vaccination status- a case-control study. *Br J Clin Pharmacol*. 2001;51(3):271–276
450. Fleming PJ, Blair PS, Platt MW, Tripp J, Smith IJ, Golding J. The UK accelerated immunisation programme and sudden unexpected death in infancy: case-control study. *BMJ*. 2001;322(7290):822
451. Müller-Nordhorn J, Hettler-Chen CM, Keil T, Muckelbauer R. Association between sudden infant death syndrome and diphtheria-tetanus-pertussis immunisation: an ecological study. *BMC Pediatr*. 2015;15(1):1
452. Fine PEM, Chen RT. Confounding in studies of adverse reactions to vaccines. *Am J Epidemiol*. 1992;136(2):121–135
453. Virtanen M, Peltola H, Paunio M, Heinonen OP. Day-to-day reactogenicity and the healthy vaccinee effect of measles-mumps-rubella vaccination. *Pediatrics*. 2000;106(5):E62
454. Vennemann MM, Höffgen M, Bajanowski T, Hense HW, Mitchell EA. Do immunisations reduce the risk for SIDS? a meta-analysis. *Vaccine*. 2007;25(26):4875–4879
455. Centers for Disease Control and Prevention (CDC). Suffocation deaths associated with use of infant sleep positioners—United States, 1997-2011. *MMWR Morb Mortal Wkly Rep*. 2012;61(46):933–937
456. US Consumer Product Safety Commission. Deaths prompt CPSC, FDA warning on infant sleep positioners Bethesda, MD: US Consumer Product Safety Commission; September 2010
457. Bar-Yishay E, Gaides M, Goren A, Szeinberg A. Aeration properties of a new sleeping surface for infants. *Pediatr Pulmonol*. 2011;46(2):193–198
458. Golditz PB, Joy GJ, Dunster KR. Rebreathing potential of infant mattresses and bedcovers. *J Paediatr Child Health*. 2002;38(2):192–195
459. Carolan PL, Wheeler WB, Ross JD, Kemp RJ. Potential to prevent carbon dioxide rebreathing of commercial products marketed to reduce sudden infant death syndrome risk. *Pediatrics*. 2000;105(4 Pt 1):774–779
460. Steinschneider A. Prolonged apnea and the sudden infant death syndrome: clinical and laboratory observations. *Pediatrics*. 1972;50(4):646–654
461. Hodgman JE, Hoppenbrouwers T. Home monitoring for the sudden infant death syndrome. the case against. *Ann N Y Acad Sci*. 1988;533:164–175
462. Ward SL, Keens TG, Chan LS, et al. Sudden infant death syndrome in infants evaluated by apnea programs in California. *Pediatrics*. 1986;77(4):451–458
463. Monod N, Plouin P, Sternberg B, et al. Are polygraphic and cardiopneumographic respiratory patterns useful tools for predicting the risk for sudden infant death syndrome? a 10-year study. *Biol Neonate*. 1986;50(3):147–153
464. Ramanathan R, Corwin MJ, Hunt CE, et al; Collaborative Home Infant Monitoring Evaluation (CHIME) Study Group. Cardiorespiratory events recorded on home monitors: comparison of healthy infants with those at increased risk for SIDS. *JAMA*. 2001;285(17):2199–2207
465. Eichenwald EC; Committee on Fetus and Newborn, American Academy of Pediatrics. Apnea of prematurity. *Pediatrics*. 2016;137(1):e20153757
466. Committee on Fetus and Newborn. American Academy of Pediatrics. Apnea, sudden infant death syndrome, and home monitoring. *Pediatrics*. 2003;111(4 Pt 1):914–917
467. U.S. Department of Health and Human Services, Food and Drug Administration, Center for Devices and Radiological Health. *General Wellness: Policy for Low Risk Devices. Guidance for Industry and Food and Drug Administration Staff*. Washington, DC: U.S. Food and Drug Administration; 2019
468. Anjewierden S, Humpherys J, LaPage MJ, Asaki SY, Aziz PF. Detection of tachyarrhythmias in a large cohort of infants using direct-to-consumer heart rate monitoring. *J Pediatr*. 2021;232:147–153.e1
469. Dangerfield MI, Ward K, Davidson L, Adamian M. Initial experience and usage patterns with the Owlet smart sock monitor in 47,495 newborns. *Glob Pediatr Health*. 2017;4:2333794X17742751
470. Bonafide CP, Jamison DT, Foglia EE. The emerging market of smartphone-integrated infant physiologic monitors. *JAMA*. 2017;317(4):353–354
471. Hutchison BL, Hutchison LA, Thompson JM, Mitchell EA. Plagiocephaly and brachycephaly in the first two years of life: a prospective cohort study. *Pediatrics*. 2004;114(4):970–980

472. van Vlimmeren LA, van der Graaf Y, Boere-Boonekamp MM, L'Hoir MP, Helders PJ, Engelbert RH. Risk factors for deformational plagiocephaly at birth and at 7 weeks of age: a prospective cohort study. *Pediatrics*. 2007;119(2):e408–e418
473. Miller RI, Clarren SK. Long-term developmental outcomes in patients with deformational plagiocephaly. *Pediatrics*. 2000;105(2):E26
474. Panchal J, Amirshaybani H, Gurwitsch R, et al. Neurodevelopment in children with single-suture craniosynostosis and plagiocephaly without synostosis. *Plast Reconstr Surg*. 2001;108(6):1492–1498, discussion 1499–1500
475. Balan P, Kushnerenko E, Sahlin P, Huotilainen M, Näätänen R, Hukki J. Auditory ERPs reveal brain dysfunction in infants with plagiocephaly. *J Craniofac Surg*. 2002;13(4):520–525, discussion 526
476. Chaddock WM, Kast J, Donahue DJ. The enigma of lambdoid positional molding. *Pediatr Neurosurg*. 1997;26(6):304–311
477. Collett BR, Wallace ER, Kartin D, Cunningham ML, Speltz ML. Cognitive outcomes and positional plagiocephaly. *Pediatrics*. 2019;143(2):e20182373
478. Salls JS, Silverman LN, Gatty CM. The relationship of infant sleep and play positioning to motor milestone achievement. *Am J Occup Ther*. 2002;56(5):577–580
479. Kuo YL, Liao HF, Chen PC, Hsieh WS, Hwang AW. The influence of wakeful prone positioning on motor development during the early life. *J Dev Behav Pediatr*. 2008;29(5):367–376
480. Tremblay MS, Chaput JP, Adamo KB, et al. Canadian 24-hour movement guidelines for the early years (0-4 years): an integration of physical activity, sedentary behaviour, and sleep. *BMC Public Health*. 2017;17(Suppl 5):874
481. Gerard CM, Harris KA, Thach BT. Physiologic studies on swaddling: an ancient child care practice, which may promote the supine position for infant sleep. *J Pediatr*. 2002;141(3):398–403
482. van Sleuwen BE, Engelberts AC, Boere-Boonekamp MM, Kuis W, Schulpens TW, L'Hoir MP. Swaddling: a systematic review. *Pediatrics*. 2007;120(4):e1097–e1106
483. Kelly BA, Irigoyen MM, Pomerantz SC, Mondesir M, Isaza-Brando N. Swaddling and infant sleeping practices. *J Community Health*. 2017;42(1):10–14
484. McDonnell E, Moon RY. Infant deaths and injuries associated with wearable blankets, swaddle wraps, and swaddling. *J Pediatr*. 2014;164(5):1152–1156
485. Richardson HL, Walker AM, Horne RS. Influence of swaddling experience on spontaneous arousal patterns and autonomic control in sleeping infants. *J Pediatr*. 2010;157(1):85–91
486. Richardson HL, Walker AM, Horne RS. Minimizing the risks of sudden infant death syndrome: to swaddle or not to swaddle? *J Pediatr*. 2009;155(4):475–481
487. Narangerel G, Pollock J, Manaseki-Holland S, Henderson J. The effects of swaddling on oxygen saturation and respiratory rate of healthy infants in Mongolia. *Acta Paediatr*. 2007;96(2):261–265
488. Kutlu A, Memik R, Mutlu M, Kutlu R, Arslan A. Congenital dislocation of the hip and its relation to swaddling used in Turkey. *J Pediatr Orthop*. 1992;12(5):598–602
489. Chaarani MW, Al Mahmeid MS, Salman AM. Developmental dysplasia of the hip before and after increasing community awareness of the harmful effects of swaddling. *Qatar Med J*. 2002;11(1):40–43
490. Yamamuro T, Ishida K. Recent advances in the prevention, early diagnosis, and treatment of congenital dislocation of the hip in Japan. *Clin Orthop Relat Res*. 1984; (184):34–40
491. Coleman SS. Congenital dysplasia of the hip in the Navajo infant. *Clin Orthop Relat Res*. 1968;56:179–193
492. Tronick EZ, Thomas RB, Daltabuit M. The Quechua manta pouch: a caretaking practice for buffering the Peruvian infant against the multiple stressors of high altitude. *Child Dev*. 1994;65(4):1005–1013
493. Manaseki S. Mongolia: a health system in transition. *BMJ*. 1993;307(6919):1609–1611
494. Franco P, Seret N, Van Hees JN, Scaillet S, Groswasser J, Kahn A. Influence of swaddling on sleep and arousal characteristics of healthy infants. *Pediatrics*. 2005;115(5):1307–1311
495. Abdeyazdan Z, Mohammadian-Ghahfarokhi M, Ghazavi Z, Mohammadzadeh M. Effects of nesting and swaddling on the sleep duration of premature infants hospitalized in neonatal intensive care units. *Iran J Nurs Midwifery Res*. 2016;21(5):552–556
496. Franco P, Scaillet S, Groswasser J, Kahn A. Increased cardiac autonomic responses to auditory challenges in swaddled infants. *Sleep*. 2004;27(8):1527–1532
497. Rubens DD, Vohr BR, Tucker R, O'Neil CA, Chung W. Newborn oto-acoustic emission hearing screening tests: preliminary evidence for a marker of susceptibility to SIDS. *Early Hum Dev*. 2008;84(4):225–229
498. Blair PS, Rubens D, Pease A, et al. Sudden infant death syndrome (SIDS) and the routine otoacoustic emission infant hearing screening test: an epidemiological retrospective case-control study. *BMJ Open*. 2019;9(7):e030026
499. Farquhar LJ, Jennings P. Newborn hearing screen results for infants that died of SIDS in Michigan 2004-2006. *Early Hum Dev*. 2008;84(10):699
500. Chan RS, McPherson B, Zhang VW. Neonatal otoacoustic emission screening and sudden infant death syndrome. *Int J Pediatr Otorhinolaryngol*. 2012;76(10):1485–1489
501. Lavezzi AM, Ottaviani G, Matturri L. Developmental alterations of the auditory brainstem centers—pathogenetic implications in sudden infant death syndrome. *J Neurol Sci*. 2015;357(1–2):257–263
502. Ajzen I. The theory of planned behavior. *Organ Behav Hum Decis Process*. 1991;50:179–211
503. Fishbein M, Ajzen I. *Belief, Attitude, Intention, and Behavior: An Introduction to Theory and Research*. Reading, MA: Addison-Wesley; 1975
504. Prochaska JO, DiClemente CC. Stages of change and the modification of problem behaviors. In: Hersen M, Eisler RM, Miller PM, eds. *Progress in Behaviour Modification*. Sycamore: Sycamore Press; 1992:184–214
505. Janz NK, Becker MH. The health belief model: a decade later. *Health Educ Q*. 1984;11(1):1–47

506. Schultz PW, Nolan JM, Cialdini RB, Goldstein NJ, Griskevicius V. The constructive, destructive, and reconstructive power of social norms. *Psychol Sci*. 2007;18(5):429–434
507. Davey-Rothwell MA, Kuramoto SJ, Latkin CA. Social networks, norms, and 12-step group participation. *Am J Drug Alcohol Abuse*. 2008;34(2):185–193
508. Moon RY, Corwin MJ, Kerr S, et al. Mediators of improved adherence to infant safe sleep using a mobile health intervention. *Pediatrics*. 2019;143(5):e20182790
509. Ahlers-Schmidt CR, Schunn C, Lopez V, et al. A comparison of community and clinic baby showers to promote safe sleep for populations at high risk for infant mortality. *Glob Pediatr Health*. 2016;3:2333794X15622305
510. Hesselink AE, van Poppel MN, van Eijsden M, Twisk JW, van der Wal MF. The effectiveness of a perinatal education programme on smoking, infant care, and psychosocial health for ethnic Turkish women. *Midwifery*. 2012;28(3):306–313
511. Colson ER, Levenson S, Rybin D, et al. Barriers to following the supine sleep recommendation among mothers at four centers for the Women, Infants, and Children Program. *Pediatrics*. 2006;118(2):e243–e250
512. Colson ER, Rybin D, Smith LA, Colton T, Lister G, Corwin MJ. Trends and factors associated with infant sleeping position: the national infant sleep position study, 1993-2007. *Arch Pediatr Adolesc Med*. 2009;163(12):1122–1128
513. Robida D, Moon RY. Factors influencing infant sleep position: decisions do not differ by SES in African-American families. *Arch Dis Child*. 2012;97(10):900–905
514. Moon RY, Mathews A, Oden R, Carlin R. A qualitative analysis of how mothers' social networks are established and used to make infant care decisions. *Clin Pediatr (Phila)*. 2019;58(9):985–992
515. Colson ER, Bergman DM, Shapiro E, Leventhal JH. Position for newborn sleep: associations with parents' perceptions of their nursery experience. *Birth*. 2001;28(4):249–253
516. Mason B, Ahlers-Schmidt CR, Schunn C. Improving safe sleep environments for well newborns in the hospital setting. *Clin Pediatr (Phila)*. 2013;52(10):969–975
517. McKinney CM, Holt VL, Cunningham ML, Leroux BG, Starr JR. Maternal and infant characteristics associated with prone and lateral infant sleep positioning in Washington state, 1996-2002. *J Pediatr*. 2008;153(2):194–198, e191–193
518. Hwang SS, Melvin P, Diop H, Settle M, Mourad J, Gupta M. Implementation of safe sleep practices in Massachusetts NICUs: a state-wide QI collaborative. *J Perinatol*. 2018;38(5):593–599
519. Kellams A, Parker MG, Geller NL, et al. Today's baby quality improvement: safe sleep teaching and role modeling in 8 US maternity units. *Pediatrics*. 2017;140(5):e20171816
520. Kuhlmann S, Ahlers-Schmidt CR, Lukasiewicz G, Truong TM. Interventions to improve safe sleep among hospitalized infants at eight children's hospitals. *Hosp Pediatr*. 2016;6(2):88–94
521. Macklin JR, Gittelman MA, Denny SA, Southworth H, Arnold MW. The EASE quality improvement project: improving safe sleep practices in Ohio children's hospitals. *Pediatrics*. 2016;138(4):e20154267
522. Shadman KA, Wald ER, Smith W, Collier RJ. Improving safe sleep practices for hospitalized infants. *Pediatrics*. 2016;138(3):e20154441
523. Scott EK, Downs SM, Pottenger AK, Bien JP, Saysana MS. Enhancing safe sleep counseling by pediatricians through a quality improvement learning collaborative. *Pediatr Qual Saf*. 2020;5(4):e327
524. Gittelman MA, Fluit K, Anzeljc S, et al. A pilot QI primary care practice program to help reduce infant mortality risks. *Inj Epidemiol*. 2020;7(Suppl 1):25
525. Pretorius KA, Mackert M, Wilcox GB. Sudden infant death syndrome and safe sleep on Twitter: analysis of influences and themes to guide health promotion efforts. *JMIR Pediatr Parent*. 2018;1(2):e10435
526. Moon RY, Carlin RF, Cornwell B, et al. Implications of mothers' social networks for risky infant sleep practices. *J Pediatr*. 2019;212:151–158.e2
527. Yanovitzky I, Blitz CL. Effect of media coverage and physician advice on utilization of breast cancer screening by women 40 years and older. *J Health Commun*. 2000;5(2):117–134
528. Marketing Management Analytics. *Marketing Evolution, Measuring Media Effectiveness: Comparing Media Contribution Throughout the Purchase Funnel*. New York, NY: Magazine Publishers of America; 2006
529. Raines DA. Factors that influence parents' adherence to safe sleep guidelines. *J Obstet Gynecol Neonatal Nurs*. 2018;47(3):316–323
530. Mathews A, Oden R, Joyner B, He J, McCarter R, Moon RY. Differences in African-American maternal self-efficacy regarding practices impacting risk for sudden infant death. *J Community Health*. 2016;41(2):244–249
531. Zundo K, Richards EA, Ahmed AH, Coddington JA. Factors associated with parental compliance with supine infant sleep: an integrative review. *Pediatr Nurs*. 2017;43(2):83–91
532. Carlin RF, Moon RY. Risk factors, protective factors, and current recommendations to reduce sudden infant death syndrome: a review. *JAMA Pediatr*. 2017;171(2):175–180
533. Hwang SS, Corwin MJ. Safe infant sleep practices: parental engagement, education, and behavior change. *Pediatr Ann*. 2017;46(8):e291–e296
534. Hirsch HM, Mullins SH, Miller BK, Aitken ME. Paternal perception of infant sleep risks and safety. *Inj Epidemiol*. 2018;5(Suppl 1):9
535. Austin JE, Nashban CJ, Doering JJ, Davies WH. Prevention messages in parent-infant bed-sharing: message source, credibility, and effectiveness. *Glob Pediatr Health*. 2017;4:2333794X17743403
536. Stevens J, Kelleher KJ. The potential of behavioral economics to promote safe infant sleep practices. *Matern Child Health J*. 2017;21(2):229–233
537. Francis DB. Young Black men's information seeking following celebrity depression disclosure: implications for mental health communication. *J Health Commun*. 2018;23(7):687–694
538. Collins KA. Death by overlaying and wedging: a 15-year retrospective study. *Am J Forensic Med Pathol*. 2001;22(2):155–159