TECHNICAL REPORT

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™

SIDS and Other Sleep-Related Infant Deaths: Evidence Base for 2016 Updated Recommendations for a Safe Infant Sleeping Environment

Rachel Y. Moon, MD, FAAP, TASK FORCE ON SUDDEN INFANT DEATH SYNDROME

Approximately 3500 infants die annually in the United States from sleep-related infant deaths, including sudden infant death syndrome (SIDS), ill-defined deaths, and accidental suffocation and strangulation in bed. After an initial decrease in the 1990s, the overall sleep-related infant death rate has not declined in more recent years. Many of the modifiable and nonmodifiable risk factors for SIDS and other sleep-related infant deaths are strikingly similar. The American Academy of Pediatrics recommends a safe sleep environment that can reduce the risk of all sleep-related infant deaths. Recommendations for a safe sleep environment include supine positioning, use of a firm sleep surface, room-sharing without bed-sharing, and avoidance of soft bedding and overheating. Additional recommendations for SIDS risk reduction include avoidance of exposure to smoke, alcohol, and illicit drugs; breastfeeding; routine immunization; and use of a pacifier. New evidence and rationale for recommendations are presented for skin-to-skin care for newborn infants, bedside and in-bed sleepers, sleeping on couches/armchairs and in sitting devices, and use of soft bedding after 4 months of age. In addition, expanded recommendations for infant sleep location are included. The recommendations and strength of evidence for each recommendation are published in the accompanying policy statement, "SIDS and Other Sleep-Related Infant Deaths: Updated 2016 Recommendations for a Safe Infant Sleeping Environment," which is included in this issue.

SEARCH STRATEGY AND METHODOLOGY

Literature searches with the use of PubMed were conducted for each of the topics in the technical report, concentrating on articles published since 2011 (when the last technical report and policy statement were published[1,2]). All iterations of the search terms were used for each topic area. For example, the pacifier topic search combined either "SIDS,"

abstract

FREE

DOI: 10.1542/peds.2016-2940

PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

FINANCIAL DISCLOSURE: The author has indicated she does not have a financial relationship relevant to this article to disclose.

FUNDING: No external funding.

POTENTIAL CONFLICT OF INTEREST: The author has indicated she has no potential conflicts of interest to disclose.

To cite: Moon RY and AAP 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

"SUID," "sudden death," or "cot death" with "pacifier," "dummy," "soother," and "sucking." A total of 63 new studies were judged to be of sufficiently high quality to be included in this technical report. In addition, because the data regarding bed-sharing have been conflicting, the independent opinion of a biostatistician with special expertise in perinatal epidemiology was solicited. The strength of evidence for recommendations, using the Strength-of-Recommendation Taxonomy,[3] was determined by the task force members. A draft version of the policy statement and technical report was 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.

TABLE 1 Definitions of Terms

Caregivers: Throughout the document, "parents" are used, but this term is meant to indicate any infant caregivers.
Bed-sharing: Parent(s) and infant sleeping together on any surface (bed, couch, chair).
Cosleeping: This term is commonly used, but the task force finds it confusing and it is not used in this document. When used, authors need to make clear whether they are referring to sleeping in close proximity (which does not necessarily entail bed-sharing) or bed-sharing.
Room-sharing: Parent(s) and infant sleeping in the same room on separate surfaces.
Sleep-related infant death: SUID that occurs during an observed or unobserved sleep period.
Sudden infant death syndrome (SIDS): Cause assigned to infant deaths that cannot be explained after a thorough case investigation including a scene investigation, autopsy, and review of the clinical history.[4]
Sudden unexpected infant death (SUID), or sudden unexpected death in infancy (SUDI): A sudden and unexpected death, whether explained or unexplained (including SIDS), occurring during infancy.

SUDDEN UNEXPECTED INFANT DEATH AND SUDDEN INFANT DEATH SYNDROME: DEFINITIONS AND DIAGNOSTIC ISSUES

Sudden unexpected infant death (SUID), also known as sudden unexpected death in infancy (SUDI), is a term used to describe any sudden and unexpected death, whether explained or unexplained (including sudden infant death syndrome [SIDS] and ill-defined deaths), occurring during infancy. After case investigation, SUID can be attributed to causes of death such as suffocation, asphyxia, entrapment, infection, ingestions, metabolic diseases, and trauma (unintentional or nonaccidental). SIDS is a subcategory of SUID and is a cause assigned to infant deaths that cannot be explained after a thorough case investigation including autopsy, a scene investigation, and review of clinical history.[4] (See Table 1 for definitions of terms.) The distinction between SIDS and other SUIDs, particularly those that occur during an unobserved sleep period (ie, sleep-related infant deaths), such as unintentional suffocation, is challenging, cannot be determined by autopsy alone, and may remain unresolved after a full case investigation. A few deaths that are diagnosed as SIDS are found, with further specialized investigations, to be attributable to metabolic disorders or arrhythmia-associated cardiac channelopathies.

Although standardized guidelines for conducting thorough case investigations have been developed (http://www.cdc.gov/sids/pdf/suidi-form2-1-2010.pdf),[5] these guidelines have not been uniformly adopted across the >2000 US medical examiner and coroner jurisdictions.[6] Information from emergency responders, scene investigators, and caregiver interviews may provide additional evidence to assist death certifiers (ie, medical examiners and coroners) in accurately determining the cause of death. However, death certifiers represent a diverse group with varying levels of skill and education. In addition, there are diagnostic preferences. Recently, much attention has focused on reporting differences among death certifiers. On one extreme, some certifiers have abandoned the use of SIDS as a cause-of-death explanation.[6] At the other extreme, some certifiers will not classify a death as suffocation in the absence of a pathologic marker of asphyxia at autopsy (ie, pathologic findings diagnostic of oronasal occlusion or chest compression[7]), even with strong evidence from the scene investigation suggesting a probable unintentional suffocation.

US Trends in SIDS, Other SUIDs, and Postneonatal Mortality

To monitor trends in SIDS and other SUIDs nationally, the United States classifies diseases and injuries according to the International Statistical Classification of Diseases (ICD) diagnostic codes. In the United States, the National Center for Health Statistics assigns a SIDS diagnostic code (International Classification of Diseases, 10th Revision [ICD-10] R95) if the death is classified with terminology such as SIDS (including presumed, probable, or consistent with SIDS), sudden infant death, sudden unexplained death in infancy, sudden unexpected infant death, SUID, or SUDI on the certified death certificate.[8,9] A death will be coded "other ill-defined and unspecified causes of mortality" (ICD-10 R99) if the cause of the death is reported as unknown or unspecified.[8] 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 reported, along with the terms bed or crib. This code also includes deaths while sleeping on couches and armchairs.

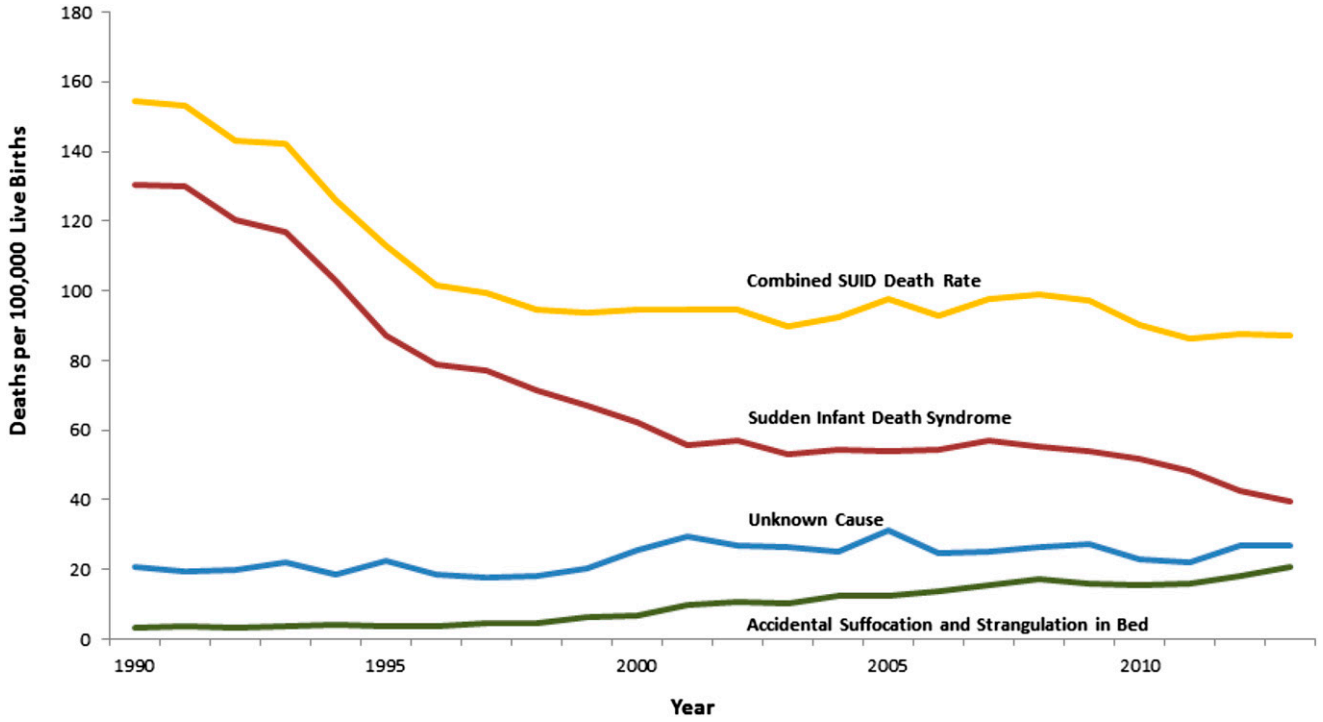

FIGURE 1

Trends in SUID by cause, 1990–2013. Source: Centers for Disease Control and Prevention/National Center for Health Statistics, National Vital Statistics System, compressed mortality file. (Figure duplicated from http://www.cdc.gov/sids/data.htm.)

Although SIDS was defined somewhat loosely until the mid-1980s, 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.[10] The "Back to Sleep" campaign (which is now known as the "Safe to Sleep" campaign[11]) was initiated in 1994 under the leadership of the National Institute of Child Health and Human Development (now the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development) as a joint effort of the Maternal and Child Health Bureau of the Health Resources and Services Administration, the AAP, the SIDS Alliance (now First Candle), and the Association of SIDS and Infant Mortality Programs. Between 1992 and 2001, the SIDS rate declined, with the most dramatic declines in the years immediately after the first nonprone sleep position recommendations, and this decline was consistent with the steady increase in the prevalence of supine sleeping (Fig 1).[12] The US 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 40 in 2013 (the latest year that data are available). In 2013, 1561 infants died of SIDS.[13] Although SIDS rates have declined by >50% since the early 1990s, SIDS remains the leading cause of postneonatal (28 days to 1 year of age) mortality.

The all-cause postneonatal death rate follows a trend similar to the SIDS and SUID rates, with a 26% decline from 1992 to 2001 (from 314 to 231 per 100 000 live births). From 2001 until 2009, postneonatal mortality rates also remained fairly unchanged (from 231 to 222 per 100 000 live births), and then have declined yearly since 2009 to a rate of 193 per 100 000 live births in 2013.[14] 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]),[15,16] and that at least some of the decline in SIDS rates may be explained by increasing rates of these other assigned causes of SUID.[15,17] To account for variations in death certifier classification and to more consistently track SIDS and other sleep-related infant deaths, the National Center for Health Statistics has created the special cause-of-death category SUID. The SUID category captures deaths with an underlying cause coded as ICD-10 R95, R99, and W75.[13] In 2013, SIDS accounted for 46% of the 3422

SUIDs in the United States. Similar to the SIDS rate, the SUID rate also declined in the late 2000s, from 99 per 100 000 live births in 2009 to 87 in 2013.

Racial and Ethnic Disparities

SIDS and SUID mortality rates, like other causes of infant mortality, have notable and persistent racial and ethnic disparities.[14] Despite the decline in SIDS and SUIDs in all races and ethnicities, the rate of SUIDs among non-Hispanic black (172 per 100 000 live births) and American Indian/Alaska Native (191 per 100 000 live births) infants was more than double that of non-Hispanic white infants (84 per 100 000 live births) in 2010–2013 (Fig 2). SIDS rates for Asian/Pacific Islander and Hispanic infants were much lower than the rate for non-Hispanic white infants. Furthermore, similar racial and ethnic disparities are seen with deaths attributed to both ASSB and ill-defined or unspecified deaths (Fig 2). Differences in the prevalence of supine positioning and other sleep environment conditions between racial and ethnic populations may contribute to these disparities.[18] The prevalence of supine positioning in 2010 data from the National Infant Sleep Position Study in white infants was 75%, compared with 53%, 73%, and 80% among black, Hispanic, and Asian infants, respectively (Fig 3).[19] The Pregnancy Risk Assessment Monitoring System also monitors the prevalence of infant sleep position in several states (http://www.cdc.gov/prams/pramstat/index.html). In 2011, 78% of mothers reported that they most often lay their infants on their backs for sleep (26 states reporting and most recent year available), with 80.3% of white mothers and 54% of black mothers reporting supine placement. Parent-infant bed-sharing[20–22] and the use of soft bedding are also more common among black families

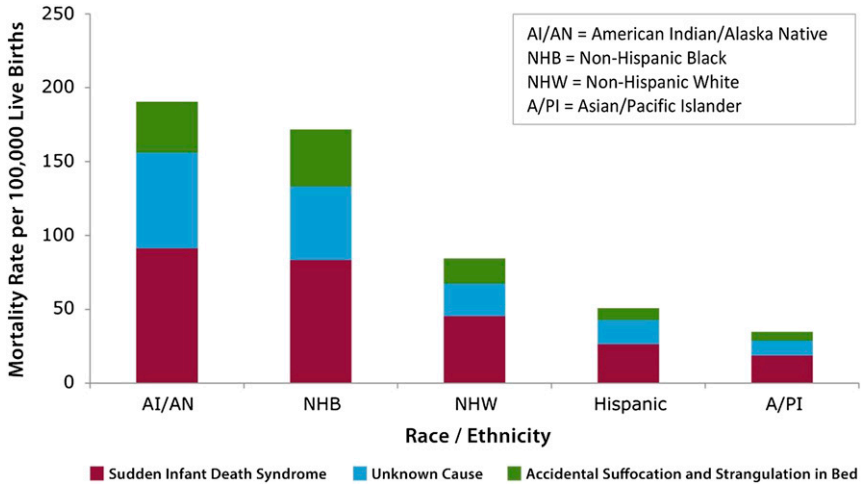

FIGURE 2
SUID by race/ethnicity, 2010–2013. Source: Centers for Disease Control and Prevention/National Center for Health Statistics, National Vital Statistics System, period-linked birth/infant death data. (Figure duplicated from http://www.cdc.gov/sids/data.htm.)

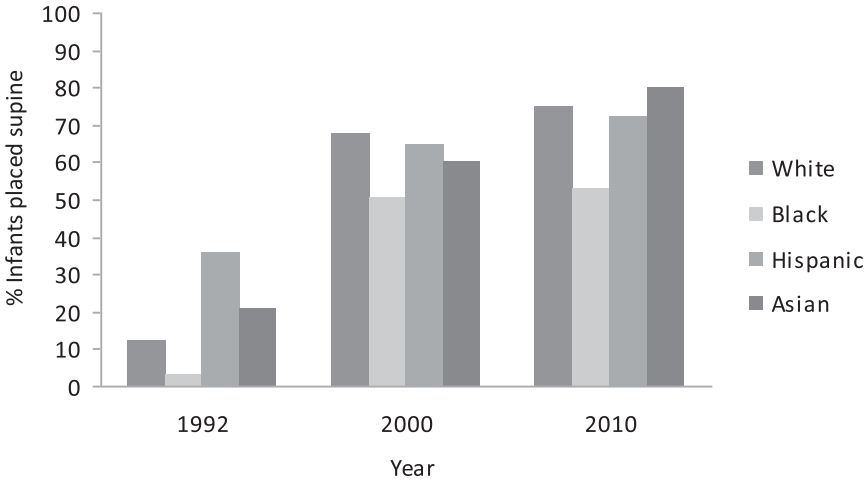

FIGURE 3
Prevalence of supine sleep positioning by maternal race and ethnic origin, 1992–2010. Source: National Infant Sleep Position Study. Note that data collection for the National Infant Sleep Position Study ended in 2010.

than among other racial/ethnic groups.[23–25]

Age at Death

Ninety percent of SIDS cases occur before an infant reaches the age of 6 months.[16] SIDS peaks between 1 and 4 months of age. Although SIDS was once considered a rare event during the first month after birth, in 2004–2006 nearly 10% of cases that were coded as SIDS occurred during this period. SIDS is uncommon after 8 months of age.[16] A similar age distribution is seen for ASSB.[16]

PATHOPHYSIOLOGY AND GENETICS OF SIDS

A working model of SIDS pathogenesis includes a convergence of exogenous triggers or "stressors" (eg, prone sleep position, overbundling, airway obstruction), a critical period of development, and dysfunctional

and/or immature cardiorespiratory and/or arousal systems (intrinsic vulnerability) that lead to a failure of protective responses (Fig 4).[26] The convergence of these factors may ultimately result in a combination of progressive asphyxia, bradycardia, hypotension, metabolic acidosis, and ineffectual gasping, leading to death.[27] Thus, death may occur as a result of the interaction between a vulnerable infant and a potentially asphyxiating and/or overheating sleep environment.[28]

The mechanisms responsible for intrinsic vulnerability (ie, dysfunctional cardiorespiratory and/or arousal protective responses) remain unclear but may be the result of in utero environmental conditions and/or genetically determined maldevelopment or delay in maturation. Infants who die of SIDS are more likely to have been born preterm and/or were growth restricted, which suggests a suboptimal intrauterine environment. Other adverse in utero environmental conditions include exposure to nicotine or other components of cigarette smoke and alcohol.[29]

Recent studies have explored how prenatal exposure to cigarette smoke may result in an increased risk of SIDS. In animal models, exposure to cigarette smoke or nicotine during fetal 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,[30,31] reduces the number of medullary serotonergic (5-hydroxytryptamine [5-HT]) neurons in the raphe obscurus in mice,[32] increases 5-HT and 5-HT turnover in Rhesus monkeys,[33] alters neuronal excitability of neurons in the nucleus tractus solitarius (a brainstem region important for sensory integration) in guinea pigs,[34] and alters fetal autonomic activity

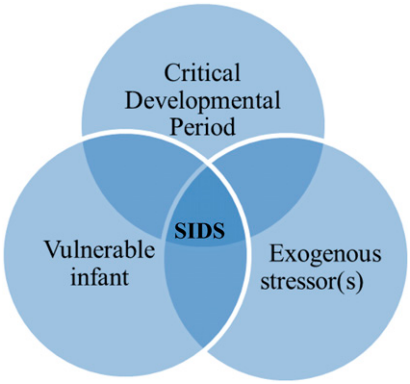

FIGURE 4
Triple risk model for SIDS. Adapted from Filiano and Kinney.[26]

and medullary neurotransmitter receptors, including nicotinic receptors, in baboons.[35–37] From a functional perspective, prenatal exposure to nicotine causes hypoventilation and increased apnea[38,39]; reduces hypercarbia and hypoxia-induced ventilator chemoreflexes in mice, rats,[38–40] and lambs[41]; and blunts arousal in response to hypoxia in rats[40] and lambs.[41]

In human infants, there are strong associations between nicotinic acetylcholine receptors and serotonergic (5-HT) receptors in the brainstem during development,[42] and there is important recent evidence of epigenetic changes in the placentas of infants with prenatal tobacco smoke exposure.[43] Prenatal exposure to tobacco smoke attenuates recovery from hypoxia in preterm infants,[44] decreases heart rate variability in preterm[45] and term[46] infants, and abolishes the normal relationship between heart rate and gestational age at birth.[45] Moreover, infants of smoking mothers exhibit impaired arousal patterns to trigeminal stimulation in proportion to urinary cotinine concentrations.[47] It is important to also note 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 or a 60° head-up tilt is greater than expected.[48] These changes in autonomic function, arousal, and cardiovascular reflexes may all increase an infant's vulnerability to SIDS.

A recent large systematic review of the neuropathologic features of unexplained SUDI, 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."[49] Brainstem abnormalities that involve the 5-HT system in up to 70% of infants who die of SIDS have now been confirmed in several independent data sets and laboratories.[29,50–52] These include decreased 5-hydroxytryptamine 1A (5-HT1A) receptor binding, a relative decreased binding to the 5-HT transporter, increased numbers of immature 5-HT neurons, and decreased tissue levels of 5-HT and the rate-limiting enzyme for 5-HT synthesis, tryptophan hydroxylase.[53] Moreover, there is no evidence of excessive serotonin degradation as assessed by levels of 5-hydroxyindoleacetic acid (the main metabolite of serotonin) or ratios of 5-hydroxyindoleacetic acid to serotonin.[35] This area of 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. Importantly, these findings are not confined to nuclei containing 5-HT neurons but also include relevant projection sites. Other abnormalities in brainstem projection sites have been described as well. For example, abnormalities of Phox2B immune-reactive neurons have been reported in the homologous human retrotrapezoid nucleus, a region of the brainstem that receives important 5-HT

projections and is critical to carbon dioxide chemoreception and implicated in human congenital central hypoventilation syndrome.[54]

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 important in the 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. Recently, abnormalities in the dentate gyrus (a component of the hippocampus) were observed in 41% of 153 infants who died unexpectedly with no apparent cause and 43% of the subset of deaths classified as SIDS. This finding suggests that dysfunction of other brain regions interconnected with the brainstem may participate in the pathogenesis of SIDS.[55] Dentate gyrus bilamination is also found in some cases of temporal lobe epilepsy. A future potential line of investigation is a possible link in brainstem-limbic–related homeostatic instability between SIDS and sudden unexpected death in epilepsy and febrile seizures noted in some cases of sudden unexpected death in childhood.[55]

There are significant associations between brainstem 5-HT1A receptor binding abnormalities and specific SIDS risk factors, including tobacco smoking.[52] These data confirm results from earlier studies in humans[29,53] and are also consistent with studies in piglets that reveal that postnatal exposure to nicotine decreases medullary 5-HT1A receptor immunoreactivity.[56] 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 show diminished ventilator responses to carbon dioxide, dysfunctional heat production and heat loss mechanisms, and altered sleep architecture.[57] 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 auto-resuscitate in response to asphyxia.[58,59] Moreover, animals with 5-HT neuron deficiency caused by direct injection of a 5-HT–selective neurotoxin had impaired arousal in response to hypoxia.[60]

Some cases of SUID have a clear genetic cause, such as medium-chain acyl-coenzyme A dehydrogenase deficiency. A recent study in California showed that the frequency of mutations for undiagnosed inborn errors of metabolism was similar in SIDS and controls and that newborn screening was effectively detecting medium-chain and very-long-chain acyl-coenzyme A dehydrogenase deficiencies that could potentially lead to SUID.[61] There is no evidence of a strong heritable contribution for SIDS; however, genetic alterations that may increase the vulnerability to SIDS have been observed. 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. (For a comprehensive review, see Opdal and Rognum.[62]) 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.[63] 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)[62]; however, at least 1 study did not confirm this association.[64] 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 SIDS[63] but not in a Norwegian population.[62]

It has been estimated that 5% to 10% of infants who die of SIDS have novel mutations in the cardiac sodium or potassium channel genes, resulting in long QT syndrome, as well as in other genes that regulate channel function.[63] 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.[65] There is molecular and functional evidence that implicates specific *SCN5A* (sodium channel gene) β subunits in SIDS pathogenesis.[66] 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.[67] 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 a high prevalence in the National Heart, Lung, and Blood Institute GO Exome Sequencing Project Database.[68] A large study in a nonreferred nationwide Danish cohort estimated that up to 7.5% of SIDS cases may be explained by genetic variants in the sodium channel complex.[69] 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.

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 *PACAP* (pituitary adenylate cyclase-activation polypeptide) gene and the gene of 1 of its receptors (*PAC1*) have received recent attention because of the apparent racial differences in their expression. For example, there were no associations between *PACAP* and SIDS found in white infants, but in SIDS cases in black infants a specific allele was significantly associated.[70] Although in a recent study, a strong association between variants in the *PAC1* gene and SIDS was not found, a number of potential associations between race-specific variants and SIDS were identified; these warrant further study.[71] There have also been a number of reports of polymorphisms or mutations in genes regulating inflammation,[72,73] energy production,[74–76] and hypoglycemia[77] in infants who died of SIDS, but these associations require more study to determine their importance.

RECOMMENDATIONS TO REDUCE THE RISK OF SIDS AND OTHER SLEEP-RELATED INFANT DEATHS

The recommendations outlined herein were developed to reduce the risk of SIDS and sleep-related suffocation, asphyxia, and entrapment among infants in the general population. 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 recommendations are intended for all who care for infants, some recommendations are also directed 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 women who are pregnant or may become pregnant in the near future.

The recommendations, along with the strength of the recommendation, are summarized in the accompanying policy statement.[78] It should be noted that there are no randomized controlled trials with regard to SIDS and other sleep-related deaths; instead, case-control studies are the standard.

The recommendations are based on epidemiologic 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 health care practitioners in conversations with parents and others who care for infants. Health care practitioners are encouraged to have open and nonjudgmental conversations with families about their sleep practices. Individual medical conditions may warrant that a health care provider make different recommendations after weighing the relative risks and benefits.

INFANT SLEEP POSITION

To reduce the risk of SIDS, infants should be placed for sleep in the supine position (wholly on the back) for every sleep period by every caregiver until 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.[79–82] 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.[83,84] Evidence suggests that prone sleeping alters the autonomic control of the infant cardiovascular system during sleep, particularly at 2 to 3 months of age,[85] and may result in decreased cerebral oxygenation.[86] The prone position places infants at high risk of SIDS (odds ratio [OR]: 2.3–13.1).[87–91] In 1 US study, SIDS risk associated with the side position was similar in magnitude to that associated with the prone position (ORs: 2.0 and 2.6, respectively),[88] and a higher population-attributable risk has been reported for the side sleep position than for the prone position.[90,92] Furthermore, the risk of SIDS is exceptionally high for infants who are placed on the side and found on the stomach (OR: 8.7).[88] 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.[90,93] Infants who are unaccustomed to the prone position and who are placed prone for sleep are also at greater risk than those usually placed prone (adjusted OR: 8.7–45.4).[88,94,95] It is therefore critically important that every caregiver use the supine sleep position for every sleep period.

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.

Despite these recommendations, the prevalence of supine positioning has remained stagnant for the past decade.[19] 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.[96–104] However, an infant who wakes frequently is normal and should not be perceived as a poor sleeper. Physiologic studies show that infants are less likely to arouse when they are sleeping in the prone position.[105–113] The ability to arouse from sleep is an important protective physiologic response to stressors during sleep,[114–118] and the infant's ability to sleep for sustained periods may not be physiologically advantageous.

The supine sleep position does not increase the risk of choking and aspiration in infants, even in those with gastroesophageal reflux.

Parents and caregivers continue to be concerned that the infant will choke or aspirate while supine.[96–104] 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 shown an increased incidence of aspiration since the change to supine sleeping.[119–121] Parents and caregivers are often concerned about aspiration when the infant has been diagnosed with gastroesophageal reflux. The AAP concurs with the North American Society for Pediatric Gastroenterology and Nutrition that "the risk of SIDS outweighs the benefit of prone or lateral sleep position on GER [gastroesophageal reflux]; therefore, in most infants from birth to 12 months of age, supine positioning during sleep is recommended…. Therefore, prone positioning is acceptable if the infant is observed and awake, particularly in the postprandial period, but prone positioning during sleep can only be considered in infants with certain upper airway disorders in which the risk of death from GERD [gastroesophageal reflux disease] may outweigh the risk of SIDS."[122] Examples of such upper airway disorders are those in which airway-protective mechanisms are impaired, including infants with anatomic abnormalities, such as type 3 or 4 laryngeal clefts, who have not undergone antireflux surgery. There is no evidence that infants receiving nasogastric or orogastric feedings are at increased risk of aspiration if placed in the supine position. Elevating the head of the infant's crib while the infant is supine is not effective in reducing gastroesophageal reflux[123,124]; in addition, elevating the head of the crib may result in the infant sliding to the foot of the crib into a position that may compromise respiration and therefore is not recommended.

Preterm infants should be placed supine as soon as possible.

Infants born preterm have an increased risk of SIDS,[125,126] and the association between the 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.[94] Therefore, preterm infants should be placed supine for sleep as soon as clinical status has stabilized. The task force concurs with the AAP Committee on Fetus and Newborn that "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."[127] Furthermore, the task force believes that neonatologists, neonatal nurses, and other health care providers responsible for organizing the hospital discharge of infants from NICUs should be vigilant about endorsing the SIDS risk-reduction recommendations from birth. They should model the 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 beforeo discharge from the hospital.[128,129]

As stated in the AAP clinical report, "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."[130] Thereafter, or when the mother needs to sleep or take care of other needs, infants should be placed supine in a bassinet.

Placing infants on the side after birth in newborn nurseries or in mother-infant rooms continues to be a concern. The practice likely occurs because of a belief among nursery 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 health care providers placing infants in the side or prone position, they are likely to infer that supine positioning is not important[131] and therefore may be more likely to copy this practice and use the side or prone position at home.[101,104,132] 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 mother after delivery, with appropriate observation and/or monitoring, is the best approach. When the mother needs to sleep or take care of other needs, the infant should be placed supine in a bassinet.

Once an infant can roll from supine to prone and from prone to supine, the infant may remain in the sleep position that he or she assumes.

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- to 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.[133] Repositioning the sleeping infant to the supine position can be disruptive and may discourage the use of the supine position altogether. 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. If the infant can roll from supine to prone and from prone to supine, the infant can then be allowed to remain in the sleep position that he or she assumes. 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.[134] Thus, parents and caregivers should continue to keep the infant's sleep environment clear of soft or loose bedding and other objects. Parents may be reassured in being advised that the incidence of SIDS begins to decline after 4 months of age.[16]

SLEEP SURFACES

Infants should be placed on a firm sleep surface (eg, a mattress in a safety-approved crib) covered by a fitted sheet with no other bedding or soft objects to reduce the risk of SIDS and suffocation.

To avoid suffocation, rebreathing, and SIDS risk, infants should sleep on a firm surface (eg, safety-approved crib and mattress). The surface should be covered by a fitted sheet without any soft or loose bedding. A firm surface maintains its shape and will not indent or conform to the shape of the infant's head when the infant is placed on the surface. Soft mattresses, including 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.[81,135]

A crib, bassinet, portable crib, or play yard that conforms to the safety standards of the Consumer Product Safety Commission (CPSC) is recommended.

Cribs should meet safety standards of the CPSC,[136] including those for slat spacing, snugly fitting and firm mattresses, and no drop sides. The AAP recommends the use of new cribs, because older cribs may no longer meet current safety standards, may have missing parts, or may be incorrectly assembled. If an older 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.

For some families, the 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. Alternate sleep surfaces, such as portable cribs, play yards, and bassinets that meet safety standards of the CPSC,[137,138] can be used and may be more acceptable for some families because they are smaller and more portable.

Bedside sleepers are attached to the side of the parental bed. The CPSC has published safety standards for bedside sleepers,[139] and they may be considered by some parents as an option. There are no CPSC safety standards for in-bed sleepers. The task force cannot make a recommendation for or against the use of either bedside sleepers or in-bed sleepers, because there have been no studies examining the association between these products and SIDS or unintentional injury and death, including suffocation. Studies of in-bed sleepers are currently underway, but results are not yet available. Parents and caregivers should adhere to the manufacturer's guidelines regarding maximum weight of infants who use these products.[140,141] In addition, with the use of any of these products, other AAP guidelines for safe sleep outlined in this document, including supine positioning and avoidance of soft objects and loose bedding, should be followed.

Mattresses should be firm and maintain their shape even when the fitted sheet designated for that model is used, such that there are no gaps between the mattress and the wall of the bassinet, playpen, portable crib, play yard, or bedside sleeper. Only mattresses designed for the specific product should be used. Pillows or cushions should not be used as substitutes for mattresses or in addition to a mattress. Soft materials or objects, such as pillows, quilts, comforters, or sheepskins, even if covered by a sheet, should not be placed under a sleeping infant.

Mattress toppers, designed to make the sleep surface softer, should not be used for infants younger than 1 year. Any fabric on the crib walls or a canopy should be taut and firmly attached to the frame so as not to create a suffocation risk for the infant.

Infants should not be placed for sleep on adult-sized beds because of the risk of entrapment and suffocation.[142] Portable bed rails (railings installed on the side of the bed that are intended to prevent a child from falling off of the bed) should not be used with infants because of the risk of entrapment and strangulation.[143] The infant should sleep in an area free of hazards, including dangling cords, electric wires, and window-covering cords, because these may present a strangulation risk.

Recently, special crib mattresses and sleep surfaces that claim to reduce the chance of rebreathing carbon dioxide when the infant is in the prone position have been introduced. Although there are no apparent disadvantages of using these mattresses if they meet the safety standards as described previously, there are no studies that show a decreased risk of SUID/SIDS. (See section entitled "Commercial Devices" for further discussion of special mattresses.)

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 young infants.

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/day in a car seat or similar sitting device.[144] However, there are multiple concerns about the use of sitting devices as a usual infant sleep location. Placing an infant in such devices can potentiate gastroesophageal reflux[145] and positional plagiocephaly. 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 of upper airway obstruction and oxygen desaturation.[146–150] A recent 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.[151] In addition, analyses of CPSC data report injuries from falls when car seats are placed on elevated surfaces,[152–156] from strangulation on unbuckled or partially buckled car seat straps,[151] and from suffocation when car seats overturn after being placed on a bed, mattress, or couch.[155] There are also reports of suffocation in infants, particularly those who are younger than 4 months, who are carried in infant sling carriers.[151,157–159] 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, the infant should be repositioned 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.[151] If an infant falls asleep in a sitting device, he or she should be removed from the product and moved to a crib or other appropriate flat surface as soon as is safe and practical. Car seats and similar products are not stable on a crib mattress or other elevated surfaces.[152–156] Infants should not be left unattended in car seats and similar products, nor should they be placed or left in car seats and similar products with the straps unbuckled or partially buckled.[151]

BREASTFEEDING

Breastfeeding is associated with a reduced risk of SIDS. The protective effect of breastfeeding increases with exclusivity. Furthermore, any breastfeeding is more protective against SIDS than no breastfeeding.

The protective role of breastfeeding on SIDS is enhanced when breastfeeding is exclusive and without formula introduction.[160–162] Studies do not distinguish between direct breastfeeding and providing expressed 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% confidence interval [CI]: 0.51–0.81).[160] The German Study of Sudden Infant Death, the largest and most recent case-control study of SIDS, found that exclusive breastfeeding at 1 month of age halved the risk of SIDS (adjusted OR: 0.48; 95% CI: 0.28–0.82).[161] Another meta-analysis of 18 case-control studies found an unadjusted summary OR for any breastfeeding of 0.40 (95% CI: 0.35–0.44) and a pooled adjusted OR of 0.55 (95% CI: 0.44–0.69) (Fig 5).[162] The protective effect of breastfeeding increased with exclusivity, with a univariable summary OR of 0.27 (95% CI: 0.24–0.31) for exclusive breastfeeding of any duration.[162]

Physiologic sleep studies showed that breastfed infants are more easily aroused from sleep than their formula-fed counterparts.[163,164] In

addition, breastfeeding results in a decreased incidence of diarrhea, upper and lower respiratory infections, and other infectious diseases[165] that are associated with an increased vulnerability to SIDS and provides overall immune system benefits attributable to maternal antibodies and micronutrients in human milk.[166,167] 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.[165] 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.[168]

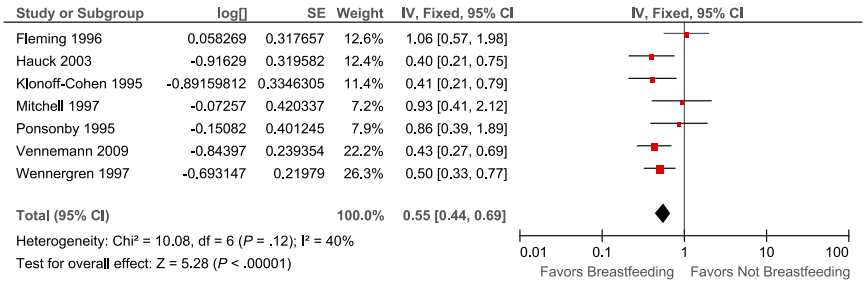

FIGURE 5

Multivariable analysis of any breastfeeding versus no breastfeeding. Adapted from Hauck et al.[162] log[ ], logarithm of the OR; Weight: weighting that the study contributed to the meta-analysis (by sample size); IV, Fixed, 95% CI, fixed-effect OR with 95% CI.

INFANT SLEEP LOCATION

It is recommended that infants sleep in the parents' room, close to the parents' bed, but on a separate surface. The infant's crib, portable crib, play yard, or bassinet should be placed in the parents' bedroom, ideally for the first year of life, but at least for 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.[169,170] Cosleeping arrangements can include bed-sharing or sleeping in the same room in close proximity.[170,171] Bed-sharing refers to a specific type of cosleeping when the infant is sleeping on the same surface with another person.[170] 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 the use of the terms bed-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) (see Table 1).

The AAP recommends room-sharing, because this arrangement decreases the risk of SIDS by as much as 50%[89,91,172,173] and is safer than bed-sharing[89,91,172,173] or solitary sleeping (when the infant is in a separate room).[89,172] In addition, room-sharing 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. Most of the epidemiologic studies on which these recommendations are based include infants up to 1 year of age. Therefore, the AAP recommends that infants room-share, ideally for the first year after birth, but at least for the first 6 months. Although there is no specific evidence for moving an infant to his or her own room before 1 year of age, room-sharing during the first 6 months is especially critical because the rates of SIDS and other sleep-related deaths, particularly those occurring in bed-sharing situations, are highest during that period.

Parent-infant bed-sharing for all or part of sleep duration is common. In 1 national survey for the period 2001–2010, 46% of parents responded that they had shared a bed with their infant (8 months or younger) at some point in the preceding 2 weeks, and 13.5% reported that they usually bed-shared.[174] In another national survey, any bed-sharing was reported by 42% of mothers at 2 weeks of infant age and 27% of mothers at 12 months of infant age.[175] In a third study, almost 60% of mothers of infants from birth to 12 months of age reported bed-sharing at least once.[176] The rate of routine bed-sharing is higher among some racial/ethnic groups, including black, Hispanic, and American Indian/Alaska Native parents/infants.[20,22,174] There are often cultural and personal reasons why parents choose to bed-share, including convenience for feeding (breast or formula), comforting a fussy or sick infant, helping the infant and/or mother sleep better, bonding and attachment, and because it is a family tradition.[175,177] 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.[178] Some parents will use bed-sharing specifically as a safety strategy if the infant sleeps in the prone position[23,178] or there is concern about environmental dangers, such as vermin or stray gunfire.[178]

Parent-infant bed-sharing continues to be highly controversial. Although electrophysiologic and behavioral studies offer a strong case for its effect in facilitating breastfeeding,[179–181] and although many parents believe that they can maintain vigilance of the infant while they are asleep and bed-sharing,[178] epidemiologic studies have shown that bed-sharing is associated with a number of conditions that are risk factors for SIDS, including soft bedding,[182–185] head covering,[186–189] and, for infants of smokers, increased exposure to tobacco smoke.[190] In addition, bed-sharing is associated with an increased risk of SIDS; a recent meta-analysis of 11 studies investigating the association of bed-sharing and SIDS showed a summary OR of 2.88 (95% CI: 1.99–4.18) with bed-sharing.[191] 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.[192,193] Infants younger than 4 months[194] and those born preterm and/or with low birth weight[195] are at the highest risk, possibly because immature motor skills and muscle strength make it difficult to escape potential threats.[191] In recent years, the concern among public health officials about bed-sharing has increased, because there have been increased reports of SUIDs occurring in high-risk sleep environments, particularly bed-sharing and/or sleeping on a couch or armchair.[196–198]

On the other hand, some breastfeeding advocacy groups encourage safer bed-sharing to promote breastfeeding,[199] and debate continues as to the safety of this sleep arrangement for low-risk, breastfed infants. In an analysis from 2 case-control studies in England (1993–1996 and 2003–2006), Blair et al[200] reported an adjusted OR of bed-sharing (excluding bed-sharing on a sofa) for infants in the absence of parental alcohol or tobacco use of 1.1 (95% CI: 0.6–2.9). For infants younger than 98 days, the OR was 1.6 (95% CI: 0.96–2.7).[200] These findings were independent of feeding method. The study lacked power to examine this association in older infants, because there was only 1 SIDS case in which bed-sharing was a factor in the absence of other risk factors. Breastfeeding was more common among bed-sharing infants, and the protective effect of breastfeeding was found only for infants who slept alone. The controls in these analyses were infants who were not bed-sharing/sofa-sharing regardless of room location; thus, they included infants who were room-sharing or sleeping in a separate room. In addition, the control infants included those whose parent(s) smoked or used alcohol. It is possible that this choice of controls overestimated their risk, leading to smaller ORs for risk among the cases (ie, biasing the results toward the null).

Carpenter et al[201] analyzed data from 19 studies across the United Kingdom, Europe, and Australasia to determine the risk of SIDS from bed-sharing when an infant is breastfed, the parents do not smoke, and the mother has not taken alcohol or drugs. When neither parent smoked, in the absence of other risk factors, the adjusted OR for bed-sharing versus room-sharing for all breastfed infants was 2.7 (95% CI: 1.4–5.3).[201] For breastfed infants younger than 3 months, in the absence of other risk factors, the adjusted OR for bed-sharing versus room-sharing was 5.1 (95% CI: 2.3–11.4). The study lacked power to examine this association in breastfed infants 3 months and older. Moreover, the large proportion of missing data for maternal alcohol and drug use is a limitation, although the authors used appropriate multiple imputation techniques for addressing these missing data.

The task force, recognizing the controversial nature of the recommendations about bed-sharing and the different methods and interpretations of these 2 sets of analyses outlined previously, requested an independent review of both articles by Dr Robert Platt, a biostatistician with expertise in perinatal epidemiology from McGill University in Canada. Dr Platt has no connection to the task force, nor does he have a vested interest in the recommendations. Dr Platt provided the following conclusion:

The fundamental difference in conclusions is that Blair et al conclude that bed-sharing in the absence of other risk factors (smoking, alcohol) does not convey an increased risk of SIDS, while Carpenter et al conclude the opposite. In both studies, the no-other-risk-factors group is limited in size, and the number of exposed cases is very small. In Blair et al, there are only 24 cases who bed-shared in the absence of these hazards. In Carpenter et al, although the total number of SIDS cases (1472) is more than 3 times the number of cases in the Blair study (400), the number of cases who bed-shared in the absence of these hazards was only 12 (personal communication, Professor Robert Carpenter, January 25, 2016). Therefore, the Carpenter results should be interpreted with some caution as well. In conclusion, both studies have strengths and weaknesses, and while on the surface the studies appear to contradict each other, I do not believe that their data support definitive differences between the 2 studies. There is some evidence of an increased risk in the no-other-risk-factor setting, in particular in the youngest age groups. However, based on concerns about sample size limitations, we are not able to say how large that increased risk is. Clearly, these data do not support a definitive conclusion that bed-sharing in the youngest

age group is safe, even under less hazardous circumstances.

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. Several products designed for in-bed use are currently under study, but results are not yet available. Bedside sleepers, which attach to the side of the parental bed and for which the CPSC published standards in 2013, may be considered by some parents as an option. The task force cannot make a recommendation for or against the use of either bedside sleepers or in-bed sleepers, because there have been no studies examining the association between these products and SIDS or unintentional injury and death, including suffocation. (See section entitled "Sleep Surfaces" for further discussion of sleepers.)

Infants who are brought into the bed for feeding or comforting should be returned 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,[202] but most of these were cross-sectional studies, which do not enable the determination of a temporal relationship: that is, whether bed-sharing promotes breastfeeding or whether breastfeeding promotes bed-sharing, and whether women who prefer one practice are also likely to prefer the other.[203] However, a more recent longitudinal study provides strong evidence that bed-sharing promotes breastfeeding duration, with the greatest effect among frequent bed-sharers.[202] 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 (adjusted OR: 2.46; 95% CI: 1.76–3.45) or partial breastfeeding (adjusted OR: 1.75; 95% CI: 1.33–2.31).[204] Although bed-sharing may facilitate breastfeeding,[175] there are other factors, such as intent, that influence successful breastfeeding.[205] 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.[194] The risk of bed-sharing is higher the longer the duration of bed-sharing during the night,[91] especially when associated with other risks.[89,90,206,207] 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.[90,207] 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.

Sleeping on couches and armchairs places infants at an extraordinarily high risk of infant death, including SIDS,[87,89,90,173,200,207] suffocation through entrapment or wedging between seat cushions, or overlay if another person is also sharing this surface.[197] Therefore, parents and other caregivers should 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 mothers, because 25% of mothers in 1 study reported falling asleep during the night when breastfeeding their infant on one of these surfaces.[176] Infants should never be placed on a couch or armchair for sleep.

Guidance for parents who fall asleep while feeding their infant.

The safest place for an infant to sleep is on a separate sleep surface designed for infants close to the parent's bed. However, the AAP acknowledges that parents frequently fall asleep while feeding the infant. Evidence suggests that it is less hazardous to fall asleep with the infant in the adult bed than on a sofa or armchair, should the parent fall asleep.[87,89,90,173,200,207] It is important to note that a large percentage of infants who die of SIDS are found with their head covered by bedding.[186] Therefore, there should be no pillows, sheets, blankets, or any other items in the bed that could obstruct infant breathing[87,182] or cause overheating.[208–211] 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 infant should be placed back on a separate sleep surface as soon as the parent awakens.[89,90,206,207]

There are specific circumstances that, in case-control studies and case series, have been shown to substantially increase the risk of SIDS or unintentional injury or death while bed-sharing, and these should be avoided at all times.

The task force emphasizes that certain circumstances greatly increase the risk of bed-sharing for both breastfed and formula-fed infants. Bed-sharing is especially dangerous in the following circumstances, and these should be avoided at all times:

- when one or both parents are smokers, even if they are not smoking in bed (OR: 2.3–21.6)[89,90,191,200,201,206,212];
- when the mother smoked during pregnancy[89,90,191,206,212];
- when the infant is younger than 4 months of age, regardless of

parental smoking status (OR: 4.7–10.4)[89,91,173,191,201,207,213,214];

- when the infant is born preterm and/or with low birth weight[195];
- when the infant is bed-sharing on excessively soft or small surfaces, such as waterbeds, sofas, and armchairs (OR: 5.1–66.9)[87,89,90,173,200,207];
- when soft bedding accessories such as pillows or blankets are used (OR: 2.8–4.1)[87,215];
- when there are multiple bed-sharers (OR: 5.4)[87];
- when the parent has consumed alcohol (OR: 1.66–89.7)[91,196,200,201] and/or illicit or sedating drugs[201]; and
- when the infant is bed-sharing with someone who is not a parent (OR: 5.4).[87]

A retrospective series of SIDS cases reported that mean maternal body weight was higher for bed-sharing mothers than for non–bed-sharing mothers.[216] 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.[217]

The safety and benefits of cobedding twins and higher-order multiples have not been established. 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.[218] However, the benefits of cobedding twins and higher-order multiples have not been established.[219–221] Twins and higher-order multiples are often born preterm and with low birth weights, so they are at increased risk of SIDS.[125,126] Furthermore, cobedding increases the potential for overheating and rebreathing, and size discordance between multiples may increase the risk of unintentional suffocation.[220] Most cobedded twins are placed on the side rather than supine.[218] Finally, cobedding of twins and higher-order multiples in the hospital setting may encourage parents to continue this practice at home.[220] 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, sheepskins, and loose bedding, such as blankets and nonfitted sheets, away from the infant's sleep area to reduce the risk of SIDS, suffocation, entrapment, and strangulation.

Soft objects and loose bedding can obstruct an infant's airway and increase the risk of SIDS,[87,182] suffocation, and rebreathing.[79,81,82,135,222–224] In the United States, nearly 55% of infants are placed to sleep underneath or on top of bedding such as thick blankets, quilts, and pillows.[25] The prevalence of bedding use is highest among infants whose mothers are teenagers, from minority racial groups, and among those without a college education.

Pillows, quilts, comforters, sheepskins, and other soft bedding can be hazardous when placed under the infant[87,182,210,225–229] or left loose in the infant's sleep area.[90,182,215,224,228–234] Bedding in the sleeping environment increases SIDS risk fivefold, independent of sleep position,[87,182] and this risk increases to 21-fold when the infant is placed prone.[87,182] Many infants who die of SIDS are found in the supine position but with their heads covered by loose bedding.[90,225,226,230] In addition, infants who bed-share (share a sleep surface) have a higher SIDS risk when sleeping on a soft as opposed to a firm surface.[215]

In addition to SIDS risk, soft objects and loose bedding in the sleeping environment may also lead to unintentional suffocation.[134,224,235] 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.[224] In addition, a CPSC report of sleep-related infant deaths in 2009–2011 found that most deaths attributed to suffocation (regardless of whether infant was sleeping in a crib, on a mattress, or in a play yard) involved extra bedding, such as pillows or blankets.[235] 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.[134]

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.[236] 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.[236] Images of infants sleeping with blankets, pillows, and other soft objects are widespread in popular magazines targeted to families with newborn infants.[237] 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.

To avoid suffocation, rebreathing, and SIDS risk, infants should sleep on a firm

surface (see section entitled "Sleep Surfaces" for a definition of a firm surface).[135] Because pillows, quilts, and comforters can obstruct the infant's airway (nose or mouth), they should never be used in the infant's sleeping environment. Infant sleep clothing, such as sleeping sacks, are designed to keep the infant warm and can be used in place of blankets to prevent the possibility of head covering or entrapment. However, care must be taken to select appropriately sized clothing and to avoid overheating. Nursing and hospital staff should model safe sleep arrangements to new parents after delivery.

Bumper pads are not recommended; they have been implicated in deaths attributable to suffocation, entrapment, and strangulation and, with new safety standards for crib slats, are not necessary for safety against head entrapment.

Bumper pads and similar products attaching to crib slats or sides are frequently used with the thought of protecting infants from injury. Initially, bumper pads were developed to prevent head entrapment between crib slats.[238] However, newer crib standards requiring crib slat spacing to be <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 et al,[239] which used 1985–2005 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. 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.[240] 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 the deaths. Two more recent analyses of CPSC data also came 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 the manufacturer's instructions and in the absence of other unsafe sleep risk factors.[241] Scheers et al,[242] in their re-analysis, 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, others[239,243] have concluded that the 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. In addition, most bumper pads obscure infant and parent visibility, which may increase parental anxiety.[236,238] 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

Consider offering a pacifier at naptime and bedtime.

Multiple case-control studies[87,91,207,244–250] and 2 meta-analyses[251,252] have reported a protective effect of pacifiers on the incidence of SIDS, particularly when used at the time of the last sleep period, with decreased risk of SIDS ranging from 50% to 90%. Furthermore, 1 study found that pacifier use favorably modified the risk profile of infants who sleep in the prone/side position, bed-share, or use soft bedding.[253] The mechanism for this apparent strong protective effect is still unclear, but favorable modification of autonomic control during sleep[254] and maintaining airway patency during sleep[255] have been proposed. Physiologic studies of the effect of pacifier use on arousal are conflicting; 1 study found that pacifier use decreased arousal thresholds,[163] but others have found no effects on arousability with pacifier use.[256,257] 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.[163,258] Two studies have shown that pacifier use is most protective when used for all sleep periods.[207,250] However, these studies also showed an increased risk of SIDS when the pacifier was usually used but not used the last time the infant was placed for sleep; the significance of these findings is yet unclear.

Although some SIDS experts and policy makers endorse pacifier use recommendations that are similar to those of the AAP,[259,260] concerns about possible deleterious effects of pacifier use have prevented others from making a recommendation for pacifier use as a risk-reduction strategy.[261] Although several observational studies[262–264] 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.[265] Furthermore, a systematic review found that the highest level of evidence (ie, from clinical trials) does not support an adverse relationship between pacifier use and breastfeeding duration or exclusivity.[266] 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.[266,267] However, some have also raised the concern that studies that show no effect of pacifier introduction on breastfeeding duration or exclusivity may not account for early weaning or failure to establish breastfeeding.[268] The AAP policy statement "Breastfeeding and the Use of Human Milk" includes a recommendation that pacifiers can be used during breastfeeding but that implementation should be delayed until breastfeeding is well established.[269] Infants who are not being directly breastfed can begin pacifier use as soon as desired.

Some dental malocclusions have been found more commonly among pacifier users than nonusers, but the differences generally disappeared after pacifier cessation.[270] The American Academy of Pediatric Dentistry policy statement 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.[271] Pacifier use is associated with an approximate 1.2- to 2-fold increased risk of otitis media, particularly between 2 and 3 years of age.[272,273] The incidence of otitis media is generally lower in the first year after birth, especially the first 6 months, when the risk of SIDS is the highest.[274–279] 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.[275–277]

Because of the risk of strangulation, pacifiers should not be hung around the infant's neck. Pacifiers that attach to the infant's clothing should not be used with sleeping infants. Objects, such as stuffed toys, that may present a suffocation or choking risk, should not 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.[248,249] One study from the United States showed a protective effect of infant finger sucking (reported as "thumb sucking") against SIDS (adjusted OR: 0.43; 95% CI: 0.25–0.77), but it was less protective than pacifier use (adjusted OR: 0.07 [95% CI: 0.01–0.64] if the infant also sucked the thumb; adjusted OR: 0.08 [95% CI: 0.03–0.23] if the infant did not suck the thumb).[249] Another study from The Netherlands did not show an association between usual finger sucking (reported as "thumb sucking") and SIDS risk (OR: 1.38; 95% CI: 0.35–1.51), but the wide CI suggests that there was insufficient power to detect a significant association.[248]

PRENATAL AND POSTNATAL EXPOSURES (INCLUDING SMOKING AND ALCOHOL)

Pregnant women should obtain regular prenatal care.

There is substantial epidemiologic evidence linking a lower risk of SIDS for infants whose mothers obtain regular prenatal care.[280–283] Women should obtain prenatal care from early in the pregnancy, according to established guidelines for frequency of prenatal visits.[284]

Smoking during pregnancy, in the pregnant woman's environment, and in the infant's environment should be avoided.

Maternal smoking during pregnancy has been identified as a major risk factor in almost every epidemiologic study of SIDS.[285–288] Smoke in the infant's environment after birth has been identified as a separate major risk factor in a few studies,[286,289] 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 extinguished[290]; there is no research to date on the significance of third-hand smoke with regard to SIDS risk. Smoke exposure adversely affects infant arousal[291–297]; in addition, smoke exposure increases the risk of preterm birth and low birth weight, both risk factors for SIDS. The effect of tobacco smoke exposure on SIDS risk is dose-dependent. The risk of SIDS is particularly high when the infant bed-shares with an adult smoker (OR: 2.3–21.6), even when the adult does not smoke in bed.[89,90,191,200,201,206,212,298] It is estimated that one-third of SIDS deaths could be prevented if all maternal smoking during pregnancy was eliminated.[299,300] The AAP supports the elimination of all tobacco smoke exposure, both prenatally and environmentally.

Avoid alcohol and illicit drug use during pregnancy and after the infant's birth.

Several studies have specifically investigated the association of SIDS with prenatal and postnatal exposure to alcohol or illicit drug use, although substance abuse often involves more than one substance and it is often difficult to separate out these variables from each other and from smoking. However, 1 study in Northern Plains American Indian infants found that periconceptional

maternal alcohol use (adjusted OR: 6.2; 95% CI: 1.6–23.3) and maternal first-trimester binge drinking (adjusted OR: 8.2; 95% CI: 1.9–35.3)[211] were associated with increased SIDS risk, independent of prenatal cigarette smoking exposure. A retrospective study from Western Australia found that a maternal alcoholism diagnosis recorded during pregnancy (adjusted hazard ratio: 6.92; 95% CI: 4.02–11.90) or within 1 year postpregnancy (adjusted hazard ratio: 8.61; 95% CI: 5.04–14.69) 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.[301] Another study from Denmark, based on prospective data on maternal alcohol use, has also shown a significant relationship between maternal binge drinking and postneonatal infant mortality, including SIDS.[302] Parental alcohol and/or illicit drug use in combination with bed-sharing places the infant at particularly high risk of SIDS and unintentional suffocation.[91,196]

Rat models have shown increased arousal latency to hypoxia in rat pups exposed to prenatal alcohol.[303] Furthermore, postmortem studies in Northern Plains American Indian infants showed 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 on univariate analysis but was not significant when prenatal smoking and case versus control status was in the model.[29] However, this study had limited power for multivariate analysis because of the small sample size. One study found an association of SIDS with heavy alcohol consumption in the 2 days before the death.[304] Several studies have found a particularly strong association when alcohol consumption or illicit drug use occurs in combination with bed-sharing.[89–91,305]

Studies investigating the relationship of illicit drug use and SIDS have focused on specific drugs or illicit drug use in general. One study found maternal cannabis use to be associated with an increased risk of SIDS (adjusted OR: 2.35; 95% CI: 1.36–4.05) at night but not during the day.[306] In utero exposure to opiates (primarily methadone and heroin) has been shown in retrospective studies to be associated with an increased risk of SIDS.[307,308] With the exception of 1 study that did not show an increased risk,[309] population-based studies have generally shown an increased risk with in utero cocaine exposure.[310–312] 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: 2.5–5.1), heroin (OR: 2.3; 95% CI: 1.3–4.0), methadone and heroin (OR: 3.2; 95% CI: 1.2–8.6), and cocaine (OR: 1.6; 95% CI: 1.2–2.2), even after controlling for race/ethnicity, maternal age, parity, birth weight, year of birth, and maternal smoking.[313] 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 drugs in general.[314]

OVERHEATING, FANS, AND ROOM VENTILATION

Avoid overheating and head covering in infants.

The amount of clothing or blankets covering an infant and the room temperature are associated with an increased risk of SIDS.[208–211] Infants who sleep in the prone position have a higher risk of overheating than supine sleeping infants.[210] However, the definition of overheating in the studies that found an increased risk of SIDS varies. It is therefore difficult to provide specific room temperature guidelines to avoid overheating.

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.[186] It is not known whether the risk related to head covering is due to overheating, hypoxia, or rebreathing.

Some have suggested that room ventilation may be important. One study found that bedroom heating, compared with no bedroom heating, increases SIDS risk (OR: 4.5),[315] and another study showed a decreased risk of SIDS in a well-ventilated bedroom (windows and doors open; OR: 0.4).[316] In 1 study, the use of a fan appeared to reduce the risk of SIDS (adjusted OR: 0.28; 95% CI: 0.10–0.77).[317] However, because of the possibility of recall bias, the small sample size of controls who used 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. On the basis of available data, the task force cannot make a recommendation on the use of a fan as a SIDS risk-reduction strategy.

IMMUNIZATIONS

Infants should be immunized in accordance with AAP and Centers for Disease Control and Prevention recommendations.

The incidence of SIDS peaks at a time when infants are receiving numerous immunizations. Case reports of a cluster of deaths shortly

after immunization with diphtheria-tetanus toxoids-pertussis vaccine in the late 1970s created concern of a possible causal relationship between vaccinations and SIDS.[318–321] Case-control studies were performed to evaluate this temporal association. Four of the 6 studies showed no relationship between diphtheria-tetanus toxoids-pertussis vaccination and subsequent SIDS[322–325]; the other 2 suggested a temporal relationship, but only in specific subgroup analysis.[326,327] In 2003, the Institute of Medicine reviewed available data and concluded the following: "The evidence favors rejection of a causal relationship between exposure to multiple vaccinations and SIDS."[328] Several analyses of the US Vaccine Adverse Event Reporting System database have shown no relationship between vaccines and SIDS.[329–331] In addition, several large-population case-control trials consistently have found vaccines to be protective against SIDS[332–335]; however, confounding factors (social, maternal, birth, and infant medical history) may account for this protective effect.[336] It also has been theorized that the decreased SIDS rate immediately after vaccination was attributable to infants being healthier at the time of immunization, or "the healthy vaccinee effect."[337] Recent illness would both place infants at higher risk of SIDS and make them more likely to have immunizations deferred.[338]

Recent studies have attempted to control for confounding by social, maternal, birth, and infant medical history.[332,334,338] A meta-analysis of 4 studies found a multivariate summary OR for immunizations and SIDS to be 0.54 (95% CI: 0.39–0.76), indicating that the risk of SIDS is halved by immunization.[338] 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. These studies have been largely focused on the correlations between the sleep environment and SIDS. Our current understanding is that the cause of SIDS is multifactorial and that death results from the interaction between a vulnerable infant and a potentially asphyxiating sleep environment. 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 ASTM International (known previously as the American Society for Testing and Materials). The AAP concurs with the US Food and Drug Administration and CPSC that manufacturers should not claim that a product or device protects against SIDS 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 of 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 devices[339]; other incidents have occurred when infants have slipped out of the restraints or rolled into a prone position while using the device.[240,340] Because of the lack of evidence that they are effective against SIDS, suffocation, or gastroesophageal reflux and because of the potential for suffocation and entrapment risk, the AAP concurs with the CPSC and the US Food and Drug Administration 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. With the use of a head box model, Bar-Yishay et al[341] found that a permeable sleeping surface exhibited significantly better aeration properties in dispersing carbon dioxide and in preventing its accumulation. 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 finding could be potentially protective against overheating, which has been identified as a risk factor for SIDS. Colditz et al[342] also performed studies both in vitro and in vivo, showing better diffusion and less accumulation of carbon dioxide with a mesh mattress. However, Carolan et al[343] found that even porous surfaces are associated with carbon dioxide accumulation and rebreathing thresholds unless there is an active carbon dioxide dispersal system. 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 the other manufacturing requirements are met, including being designed for a particular crib, having a firm surface, and maintaining its shape even when the fitted sheet designated for that model is used, such that there are no gaps between the mattress and the side of the crib, bassinet, portable crib, or play yard.

HOME MONITORS, SIDS, AND BRIEF RESOLVED UNEXPLAINED EVENTS (FORMERLY APPARENT LIFE-THREATENING EVENTS)

There is no evidence that apparent life-threatening events are precursors to SIDS. Furthermore, infant home cardiorespiratory monitors should not be used as a strategy to reduce the risk of SIDS.

For many years, it was believed that brief resolved unexplained events (BRUEs; formerly known as apparent life-threatening events [ALTEs]) were the predecessors of SIDS, and home apnea monitors were used as a strategy for preventing SIDS.[344] However, the use of home cardiorespiratory monitors has not been documented to decrease the incidence of SIDS.[345–348] Home cardiorespiratory monitors are sometimes prescribed for use at home to detect apnea and bradycardia and, when pulse oximetry is used, decreases in oxyhemoglobin saturation for infants at risk of these conditions.[349] Routine in-hospital cardiorespiratory monitoring before discharge from the hospital has not been shown to detect infants at risk of SIDS. There are no data that other commercial devices that are designed to monitor infant vital signs reduce the risk of SIDS.

TUMMY TIME

Supervised, awake tummy time is recommended to facilitate development and to minimize development of positional plagiocephaly.

Positional plagiocephaly, or plagiocephaly without synostosis (PWS), can be associated with a supine sleeping position (OR: 2.5).[350] 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.[350–352] Children with developmental delay and/or neurologic injury have increased rates of PWS, although a causal relationship has not been shown.[350,353–356] In healthy normal children, the incidence of PWS decreases spontaneously from 20% at 8 months to 3% at 24 months of age.[351] Although data to make specific recommendations as to how often and how long tummy time should be undertaken are lacking, the task force concurs with the AAP Section on Neurologic Surgery that "a certain amount of prone positioning, or 'tummy time,' while the infant is awake and being observed is recommended to help prevent the development of flattening of the occiput and to facilitate development of the upper shoulder girdle strength necessary for timely attainment of certain motor milestones."[357] The AAP clinical report "Prevention and Management of Positional Skull Deformities in Infants"[357] provides additional detail on the prevention, diagnosis, and management of positional plagiocephaly.

SWADDLING

There is no evidence to recommend swaddling as a strategy to reduce the risk of SIDS. Infants who are swaddled have an increased risk of death if they are placed in or roll to the prone position. If swaddling is used, infants should always be placed 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. Swaddling, when done correctly, can be an effective technique to help calm infants and promote sleep.[358,359]

Some have argued that swaddling can alter certain risk factors for SIDS, thus reducing the risk of SIDS. For instance, it has been suggested that the physical restraint associated with swaddling may prevent infants placed supine from rolling to the prone position.[358] One study suggested a decrease in SIDS rate with swaddling if the infant was supine, but notably, there was an increased risk of SIDS if the infant was swaddled and placed in the prone position.[210] Although another study found a 31-fold increase in SIDS risk with swaddling, the analysis was not stratified by sleep position.[196] 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 of SIDS if the infant is either placed or rolls to the prone position when swaddled.[210,359] 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.[360] Thus, if swaddling is used, the infant should be placed wholly supine, and swaddling should be discontinued as soon as the infant begins to attempt to roll. 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. There is no evidence with regard to SIDS risk related to the arms swaddled in or out.

There is some evidence that swaddling may cause detrimental physiologic consequences. For example, it can cause an increase in respiratory rate,[361] and tight

swaddling can reduce the infant's functional residual lung capacity.[358,362,363] Tight swaddling can also exacerbate hip dysplasia if the hips are kept in extension and adduction,[364–367] 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 head covering 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.[368,369] However, 1 study found no increase in abdominal skin temperature when infants were swaddled in a light cotton blanket from the shoulders down.[362]

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 shown that, in general, swaddling decreases startling,[361] increases sleep duration, and decreases spontaneous awakenings.[370] 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.[361] 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.[361] In contrast, another study has shown infants to be more easily arousable[370] and to have increased autonomic (subcortical) responses[371] to an auditory stimulus when swaddled.[371] 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. Finally, there is no evidence with regard to SIDS risk related to the arms swaddled in or out.

In summary, it is recognized that swaddling is one of many child care practices that can be used to calm infants, promote sleep, and encourage the use of the supine position. However, there is no evidence to recommend routine swaddling as a strategy to reduce the risk of SIDS. The risk of death is high if a swaddled infant is placed in or rolls to the prone position. If infants are swaddled, they should always be placed on the back. When an infant exhibits signs of attempting to roll, swaddling should no longer be used. Moreover, as many have advocated, swaddling must be correctly applied to avoid the possible hazards, such as hip dysplasia, head covering, and strangulation. Importantly, swaddling does not reduce the necessity to follow recommended safe sleep practices.

POTENTIAL TOXICANTS

There is no evidence substantiating a causal relationship between various toxicants to SIDS.

Many theories link various toxicants and SIDS.[372–374] Although 1 ecological study found a correlation of the maximal recorded nitrate levels of drinking water with local SIDS rates in Sweden,[375] no case-control study has shown a relationship between nitrates in drinking water and SIDS. Furthermore, an expert group in the United Kingdom analyzed data pertaining to a hypothesis that SIDS is related to toxic gases, such as antimony, phosphorus, or arsenic, being released from mattresses[376,377] and found the toxic gas hypothesis unsubstantiated.[378] Finally, 2 case-control studies found that wrapping mattresses in plastic to reduce toxic gas emission did not protect against SIDS.[230,379]

HEARING SCREENS

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

One retrospective case-control study examined the use of newborn transient evoked otoacoustic emission hearing screening tests as a tool to identify infants at subsequent risk of SIDS.[380] Infants who subsequent died 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. Methodologic concerns have been raised about the validity of the study methods used in this study,[381,382] and these results have not been substantiated by others. A larger, but non–peer-reviewed, report of hearing screening data in Michigan[383] and a peer-reviewed retrospective study in Hong Kong[383,384] showed no relationship between hearing screening test results and SIDS cases. Until additional data are available, hearing screening should not be considered as a valid screening tool to determine which infants may be at subsequent risk of SIDS. Furthermore, an increased risk of SIDS should not be inferred from an abnormal hearing screen result.

EDUCATIONAL INTERVENTIONS

Educational and intervention campaigns are often effective in altering practice.

Intervention campaigns for SIDS have been extremely effective, especially with regard to the avoidance of prone positioning.[385] Furthermore, primary care–based educational interventions, particularly those that address caregiver concerns and misconceptions about safe sleep recommendations,

can be effective in altering practice. For instance, addressing concerns about infant comfort, choking, and aspiration while the infant is sleeping supine is helpful.[19,96,97,386] However, many families report not receiving information consistent with AAP recommendations. When a nationally representative sample of mothers of young infants were asked about information received from their pediatricians, only 54.5% had received a recommendation to place their infant supine for sleep, 19.9% had received information about appropriate sleep location, and 11.0% had received information about pacifier use.[387] Primary care providers should be encouraged to develop quality-improvement initiatives to improve adherence to safe sleep recommendations among their patients.

In addition, modeling of unsafe sleep practices by health care and child care providers may increase the prevalence of these unsafe practices.[388–390] Modeling of unsafe practices may occur because professionals are not convinced of the utility of the safe sleep recommendations or have concerns about the supine sleep position, particularly with regard to infant comfort, choking, and aspiration.[391–395] Interventions that address provider concerns are effective in improving behavior.[391,396–398]

MEDIA MESSAGES

Media and manufacturers should follow safe sleep guidelines in their messaging and advertising.

A recent 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.[237] Media exposures (including movie, television, magazines, newspapers, and Web sites), manufacturer advertisements, and store displays affect individual behavior by influencing beliefs and attitudes. Frequent exposure to health-related media messages can affect individual health decisions,[399,400] and media messages have been very influential in decisions regarding sleep position.[101,104] Media and advertising messages contrary to safe sleep recommendations may create misinformation about safe sleep practices.

Media and manufacturer messaging and advertising should follow safe sleep guidelines in text, photos, and illustrations. In addition, public health departments and organizations that provide safe sleep information should review, revise, and reissue this information at least every 5 years to ensure that each generation of new parents receives appropriate information.

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.[78]

ACKNOWLEDGMENTS

We acknowledge the contributions provided by others to the collection and interpretation of data examined in preparation of this report. We are particularly grateful for the independent biostatistical report submitted by Robert W. Platt, PhD.

LEAD AUTHOR

Rachel Y. Moon, MD, FAAP

TASK FORCE ON SUDDEN INFANT DEATH SYNDROME

Rachel Y. Moon, MD, FAAP, Chairperson
Robert A. Darnall, MD
Lori Feldman-Winter, MD, MPH, FAAP
Michael H. Goodstein, MD, FAAP
Fern R. Hauck, MD, MS

CONSULTANTS

Marian Willinger, PhD – Eunice Kennedy Shriver *National Institute for Child Health and Human Development*
Carrie K. Shapiro-Mendoza, PhD, MPH – *Centers for Disease Control and Prevention*

STAFF

James Couto, MA

ABBREVIATIONS

AAP: American Academy of Pediatrics
ASSB: accidental suffocation or strangulation in bed
CI: confidence interval
CPSC: Consumer Product Safety Commission
ICD: International Statistical Classification of Diseases and Related Health Problems
ICD-10: International Classification of Diseases, 10th Revision
OR: odds ratio
PWS: plagiocephaly without synostosis
SIDS: sudden infant death syndrome
SUDI: sudden unexpected death in infancy
SUID: sudden unexpected infant death
5-HT: 5-hydroxytryptamine (serotonin)
5-HT1A: 5-hydroxytryptamine 1A (serotonin 1A)

REFERENCES

1. Moon RY; Task Force on Sudden Infant Death Syndrome. SIDS and other sleep-related infant deaths: expansion of recommendations for a safe infant sleeping environment. *Pediatrics*. 2011;128(5). Available at: www.pediatrics.org/cgi/content/full/128/5/e1341
2. Moon RY; Task Force on Sudden Infant Death Syndrome. SIDS and other sleep-related infant deaths: expansion of recommendations for a safe infant sleeping environment. *Pediatrics*. 2011;128(5):1030–1039
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. Willinger M, James LS, Catz C. Defining the sudden infant death syndrome (SIDS): deliberations of an expert panel convened by the National Institute of Child Health and Human Development. *Pediatr Pathol*. 1991;11(5):677–684

5. Centers for Disease Control and Prevention. Sudden unexplained infant death investigation reporting form (SUIDIRF). Available at: www.cdc.gov/SIDS/SUIDRF.htm. Accessed January 10, 2016

6. 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

7. 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

8. 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

9. 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

10. Kattwinkel J, Brooks J, Myerberg D; American Academy of Pediatrics 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

11. National Institute of Child Health and Human Development/National Institutes of Health. Safe to Sleep campaign. Available at: www.nichd.nih.gov/sts. Accessed September 21, 2016

12. National Infant Sleep Position Study Web site. Available at: http://slone-web2.bu.edu/ChimeNisp/Main_Nisp.asp. Accessed January 10, 2016

13. 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

14. US Department of Health and Human Services. Linked birth/infant death records [CDC WONDER online database]. Available at: http://wonder.cdc.gov/lbd.html. Accessed June 1, 2016

15. Malloy MH, MacDorman M. Changes in the classification of sudden unexpected infant deaths: United States, 1992–2001. *Pediatrics*. 2005;115(5):1247–1253

16. 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

17. 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

18. Hauck FR, Moore CM, Herman SM, et al. The contribution of prone sleeping position to the racial disparity in sudden infant death syndrome: the Chicago Infant Mortality Study. *Pediatrics*. 2002;110(4):772–780

19. 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

20. 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

21. 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

22. 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

23. 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

24. Rasinski KA, Kuby A, Bzdusek SA, Silvestri JM, Weese-Mayer DE. Effect of a sudden infant death syndrome risk reduction education program on risk factor compliance and information sources in primarily black urban communities. *Pediatrics*. 2003;111(4 pt 1). Available at: www.pediatrics.org/cgi/content/full/111/4/e347

25. 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

26. 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

27. Kinney HC. Brainstem mechanisms underlying the sudden infant death syndrome: evidence from human pathologic studies. *Dev Psychobiol*. 2009;51(3):223–233

28. Goldstein RD, Trachtenberg FL, Sens MA, Harty BJ, Kinney HC. Overall postneonatal mortality and rates of SIDS. *Pediatrics*. 2016;137(1):1–10

29. 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

30. Browne CJ, Sharma N, Waters KA, Machaalani R. The effects of nicotine on the alpha-7 and beta-2 nicotinic acetycholine receptor subunits in the developing piglet brainstem. *Int J Dev Neurosci*. 2010;28(1):1–7

31. 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

32. 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
33. 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
34. 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
35. Duncan JR, Paterson DS, Hoffman JM, et al. Brainstem serotonergic deficiency in sudden infant death syndrome. *JAMA*. 2010;303(5):430–437
36. 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 (1985)*. 2009;107(5):1579–1590
37. 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
38. St-John WM, Leiter JC. Maternal nicotine depresses eupneic ventilation of neonatal rats. *Neurosci Lett*. 1999;267(3):206–208
39. Eugenín 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
40. 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
41. 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
42. Duncan JR, Paterson DS, Kinney HC. The development of nicotinic receptors in the human medulla oblongata: inter-relationship with the serotonergic system. *Auton Neurosci*. 2008;144(1–2):61–75
43. 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
44. 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
45. Thiriez G, Bouhaddi M, Mourot L, et al. Heart rate variability in preterm infants and maternal smoking during pregnancy. *Clin Auton Res*. 2009;19(3):149–156
46. 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
47. Richardson HL, Walker AM, Horne RS. Maternal smoking impairs arousal patterns in sleeping infants. *Sleep*. 2009;32(4):515–521
48. 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
49. 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
50. 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
51. 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
52. Machaalani R, Say M, Waters KA. Serotoninergic receptor 1A in the sudden infant death syndrome brainstem medulla and associations with clinical risk factors. *Acta Neuropathol*. 2009;117(3):257–265
53. Paterson DS, Trachtenberg FL, Thompson EG, et al. Multiple serotonergic brainstem abnormalities in sudden infant death syndrome. *JAMA*. 2006;296(17):2124–2132
54. 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
55. Kinney HC, Cryan JB, Haynes RL, et al. Dentate gyrus abnormalities in sudden unexplained death in infants: morphological marker of underlying brain vulnerability. *Acta Neuropathol*. 2015;129(1):65–80
56. Say M, Machaalani R, Waters KA. Changes in serotoninergic receptors 1A and 2A in the piglet brainstem after intermittent hypercapnic hypoxia (IHH) and nicotine. *Brain Res*. 2007;1152:17–26
57. 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
58. 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
59. 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

60. 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

61. Rosenthal NA, Currier RJ, Baer RJ, Feuchtbaum L, Jelliffe-Pawlowski LL. Undiagnosed metabolic dysfunction and sudden infant death syndrome—a case-control study. *Paediatr Perinat Epidemiol*. 2015;29(2):151–155

62. Opdal SH, Rognum TO. Gene variants predisposing to SIDS: current knowledge. *Forensic Sci Med Pathol*. 2011;7(1):26–36

63. 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

64. 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

65. Wang DW, Desai RR, Crotti L, et al. Cardiac sodium channel dysfunction in sudden infant death syndrome. *Circulation*. 2007;115(3):368–376

66. 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

67. 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

68. Andreasen 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

69. 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

70. 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

71. 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). Available at: www.pediatrics.org/cgi/content/full/102/12/e546

72. Ferrante L, Opdal SH, Vege A, Rognum T. Cytokine gene polymorphisms and sudden infant death syndrome. *Acta Paediatr*. 2010;99(3):384–388

73. 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

74. 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

75. 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

76. 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

77. 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

78. American Academy of Pediatrics 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

79. Kanetake J, Aoki Y, Funayama M. Evaluation of rebreathing potential on bedding for infant use. *Pediatr Int*. 2003;45(3):284–289

80. Kemp JS, Thach BT. Quantifying the potential of infant bedding to limit CO2 dispersal and factors affecting rebreathing in bedding. *J Appl Physiol (1985)*. 1995;78(2):740–745

81. 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

82. Patel AL, Harris K, Thach BT. Inspired CO(2) and O(2) in sleeping infants rebreathing from bedding: relevance for sudden infant death syndrome. *J Appl Physiol (1985)*. 2001;91(6):2537–2545

83. Tuffnell CS, Petersen SA, Wailoo MP. Prone sleeping infants have a reduced ability to lose heat. *Early Hum Dev*. 1995;43(2):109–116

84. 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

85. 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

86. 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). Available at: www.pediatrics.org/cgi/content/full/127/3/e558

87. 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

88. 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

89. Blair PS, Fleming PJ, Smith IJ, et al; CESDI SUDI Research Group. Babies sleeping with parents: case-control study of factors influencing the risk of the sudden infant death syndrome. *BMJ*. 1999;319(7223):1457–1461

90. 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

91. 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

92. 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

93. 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

94. 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

95. 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

96. 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

97. 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

98. Mosley JM, Daily Stokes S, Ulmer A. Infant sleep position: discerning knowledge from practice. *Am J Health Behav*. 2007;31(6):573–582

99. Moon RY, Omron R. Determinants of infant sleep position in an urban population. *Clin Pediatr (Phila)*. 2002;41(8):569–573

100. 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

101. 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

102. Moon RY, Biliter WM. Infant sleep position policies in licensed child care centers after back to sleep campaign. *Pediatrics*. 2000;106(3):576–580

103. 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

104. 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

105. 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

106. 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

107. 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

108. 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

109. 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

110. 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

111. Horne RS, Ferens D, Watts AM, et al. The prone sleeping position impairs arousability in term infants. *J Pediatr*. 2001;138(6):811–816

112. Horne RS, Bandopadhayay 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

113. Kato I, Scaillet S, Groswasser J, et al. Spontaneous arousability in prone and supine position in healthy infants. *Sleep*. 2006;29(6):785–790

114. Phillipson EA, Sullivan CE. Arousal: the forgotten response to respiratory stimuli. *Am Rev Respir Dis*. 1978;118(5):807–809

115. 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

116. 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

117. Harper RM. State-related physiological changes and risk for the sudden infant death syndrome. *Aust Paediatr J*. 1986;22(suppl 1):55–58

118. 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

119. Byard RW, Beal SM. Gastric aspiration and sleeping position in infancy and early childhood. *J Paediatr Child Health*. 2000;36(4):403–405

120. 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

121. 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

122. Vandenplas Y, Rudolph CD, Di Lorenzo C, et al; North American Society for Pediatric Gastroenterology Hepatology and Nutrition; European Society for Pediatric Gastroenterology Hepatology and Nutrition. Pediatric gastroesophageal reflux clinical practice guidelines: joint recommendations of the North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition (NASPGHAN) and the European Society for Pediatric Gastroenterology, Hepatology, and Nutrition (ESPGHAN). *J Pediatr Gastroenterol Nutr*. 2009;49(4):498–547

123. Meyers WF, Herbst JJ. Effectiveness of positioning therapy for gastroesophageal reflux. *Pediatrics*. 1982;69(6):768–772

124. 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

125. Malloy MH, Hoffman HJ. Prematurity, sudden infant death syndrome, and age of death. *Pediatrics*. 1995;96(3 pt 1):464–471

126. 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

127. American Academy of Pediatrics Committee on Fetus and Newborn. Hospital discharge of the high-risk neonate. *Pediatrics*. 2008;122(5):1119–1126

128. Gelfer P, Cameron R, Masters K, Kennedy KA. Integrating "Back to Sleep" recommendations into neonatal ICU practice. *Pediatrics*. 2013;131(4). Available at: www.pediatrics.org/cgi/content/full/131/4e1264

129. 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

130. Feldman-Winter L, Goldsmith JP; AAP Committee on Fetus and Newborn; AAP 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

131. Moon RY, Oden RP, Joyner BL, Ajao TI. Qualitative analysis of beliefs and perceptions about sudden infant death syndrome (SIDS) among African-American mothers: implications for safe sleep recommendations. *J Pediatr*. 2010;157(1):92–97, e92

132. 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

133. 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

134. Colvin JD, Collie-Akers V, Schunn C, Moon RY. Sleep environment risks for younger and older infants. *Pediatrics*. 2014;134(2). Available at: www.pediatrics.org/cgi/content/full/134/2/e406

135. 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

136. US Consumer Product Safety Commission. *Crib Safety Tips: UseYour Crib Safely. CPSC Document 5030*. Washington, DC: US Consumer Product Safety Commission; 2011

137. Consumer Product Safety Commission. Safety standard for bassinets and cradles. *Fed Reg*. 2013;78(205):63019–63036

138. Consumer Product Safety Commission. Safety standard for play yards. *Fed Reg*. 2012;77(168):52220–52228

139. Consumer Product Safety Commission. Safety standards for bedside sleepers. *Fed Reg*. 2014;79(10):2581–2589

140. 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

141. Pike J, Moon RY. Bassinet use and sudden unexpected death in infancy. *J Pediatr*. 2008;153(4):509–512

142. 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

143. Consumer Product Safety Commission. Safety standard for portable bed rails: final rule. *Fed Reg*. 2012;77(40):12182–12197

144. Callahan CW, Sisler C. Use of seating devices in infants too young to sit. *Arch Pediatr Adolesc Med*. 1997;151(3):233–235

145. Orenstein SR, Whitington PF, Orenstein DM. The infant seat as treatment for gastroesophageal reflux. *N Engl J Med*. 1983;309(13):760–763

146. Bass JL, Bull M. Oxygen desaturation in term infants in car safety seats. *Pediatrics*. 2002;110(2 pt 1):401–402

147. 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). Available at: www.pediatrics.org/cgi/content/full/124/3/e396

148. Côté A, Bairam A, Deschenes M, Hatzakis G. Sudden infant deaths in sitting devices. *Arch Dis Child*. 2008;93(5):384–389

149. 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

150. 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

151. 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

152. 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

153. Graham CJ, Kittredge D, Stuemky JH. Injuries associated with child safety seat misuse. *Pediatr Emerg Care*. 1992;8(6):351–353

154. Parikh SN, Wilson L. Hazardous use of car seats outside the car in the United States, 2003–2007. *Pediatrics*. 2010;126(2):352–357

155. 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

156. Wickham T, Abrahamson E. Head injuries in infants: the risks of bouncy chairs and car seats. *Arch Dis Child*. 2002;86(3):168–169

157. 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

158. Madre C, Rambaud C, Avran D, Michot C, Sachs P, Dauger S. Infant deaths in slings. *Eur J Pediatr*. 2014;173(12):1659–1661

159. Consumer Product Safety Commission. Safety standard for sling carriers. *Fed Reg*. 2014;79(141):42724–42734

160. 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

161. 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). Available at: www.pediatrics.org/cgi/content/full/123/3/e406

162. 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

163. 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

164. 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

165. Duijts L, Jaddoe VW, Hofman A, Moll HA. Prolonged and exclusive breastfeeding reduces the risk of infectious diseases in infancy. *Pediatrics*. 2010;126(1). Available at: www.pediatrics.org/cgi/content/full/126/1/e18

166. 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

167. 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

168. 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

169. 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

170. 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. *Yearb Phys Anthropol*. 2007;134(S4S):133–161

171. McKenna J. *Sleeping With Your Baby: A Parent's Guide to Cosleeping*. Washington, DC: Platypus Media, LLC; 2007

172. 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

173. 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

174. Colson ER, Willinger M, Rybin D, et al. Trends and factors associated with infant bed sharing, 1993-2010: the National Infant Sleep Position Study. *JAMA Pediatr*. 2013;167(11):1032–1037

175. 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

176. 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. *Clin Lactation*. 2010;1(1):27–31

177. 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

178. 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

179. Mosko S, Richard C, McKenna J. Infant arousals during mother-infant bed sharing: implications for infant sleep and sudden infant death syndrome research. *Pediatrics*. 1997;100(5):841–849

180. McKenna JJ, Mosko SS, Richard CA. Bedsharing promotes breastfeeding. *Pediatrics*. 1997;100(2 pt 1):214–219

181. 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

182. 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

183. Unger B, Kemp JS, Wilkins D, et al. Racial disparity and modifiable risk factors among infants dying suddenly and unexpectedly. *Pediatrics*. 2003;111(2). Available at: www.pediatrics.org/cgi/content/full/111/2/e127

184. 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). Available at: www.pediatrics.org/cgi/content/full/106/3/e41

185. Drago DA, Dannenberg AL. Infant mechanical suffocation deaths in the United States, 1980-1997. *Pediatrics*. 1999;103(5). Available at: www.pediatrics.org/cgi/content/full/103/5/e59

186. 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

187. 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

188. Baddock SA, Galland BC, Taylor BJ, Bolton DP. Sleep arrangements and behavior of bed-sharing families in the home setting. *Pediatrics*. 2007;119(1). Available at: www.pediatrics.org/cgi/content/full/119/1/e200

189. Ball H. Airway covering during bed-sharing. *Child Care Health Dev*. 2009;35(5):728–737

190. Kattwinkel J, Brooks J, Keenan ME, Malloy MH; American Academy of Pediatrics. Task Force on Infant Sleep Position and Sudden Infant Death Syndrome. Changing concepts of sudden infant death syndrome: implications for infant sleeping environment and sleep position. *Pediatrics*. 2000;105(3 pt 1):650–656

191. 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):44–48, e42

192. 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

193. 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

194. 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

195. 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

196. Blair PS, Sidebotham P, Evason-Coombe C, Edmonds M, Heckstall-Smith EM, Fleming P. Hazardous cosleeping environments and risk factors amenable to change: case-control study of SIDS in south west England. *BMJ*. 2009;339:b3666

197. Rechtman LR, Colvin JD, Blair PS, Moon RY. Sofas and infant mortality. *Pediatrics*. 2014;134(5). Available at: www.pediatrics.org/cgi/content/full/134/5/e1293

198. 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

199. Bartick M, Smith LJ. Speaking out on safe sleep: evidence-based infant sleep recommendations. *Breastfeed Med*. 2014;9(9):417–422

200. 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

201. Carpenter R, McGarvey C, Mitchell EA, et al. Bed sharing when parents do not smoke: is there a risk of SIDS? An individual level analysis of five major case-control studies. *BMJ Open*. 2013;3(5):e002299

202. 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

203. 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

204. 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

205. Ball HL, Howel D, Bryant A, Best E, Russell C, Ward-Platt M. Bed-sharing by breastfeeding mothers: who bed-shares and what is the relationship with breastfeeding duration? *Acta Paediatr*. 2016;105(6):628–634

206. 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

207. 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

208. 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

209. 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

210. 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

211. 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

212. 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

213. 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
214. Academy of Breastfeeding Medicine Protocol Committee. ABM clinical protocol #6: guideline on co-sleeping and breastfeeding. Revision, March 2008. *Breastfeed Med*. 2008;3(1):38–43
215. 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
216. Carroll-Pankhurst C, Mortimer EAJ Jr. Sudden infant death syndrome, bedsharing, parental weight, and age at death. *Pediatrics*. 2001;107(3):530–536
217. 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 1):14A–15A
218. 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
219. Hayward K. Cobedding of twins: a natural extension of the socialization process? *MCN Am J Matern Child Nurs*. 2003;28(4):260–263
220. Tomashek KM, Wallman C; American Academy of Pediatrics Committee on Fetus and Newborn. Cobedding twins and higher-order multiples in a hospital setting. *Pediatrics*. 2007;120(6):1359–1366
221. National Association of Neonatal Nurses Board of Directors. NANN Position Statement 3045: cobedding of twins or higher-order multiples. *Adv Neonatal Care*. 2008;9(6):307–313
222. Chiodini BA, Thach BT. Impaired ventilation in infants sleeping facedown: potential significance for sudden infant death syndrome. *J Pediatr*. 1993;123(5):686–692
223. 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
224. 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
225. 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
226. Mitchell EA, Scragg L, Clements M. Soft cot mattresses and the sudden infant death syndrome. *N Z Med J*. 1996;109(1023):206–207
227. 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
228. 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
229. 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
230. 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
231. 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
232. 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
233. Beal SM, Byard RW. Accidental death or sudden infant death syndrome? *J Paediatr Child Health*. 1995;31(4):269–271
234. 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
235. Chowdhury RT. *Nursery Product-Related Injuries and Deaths Among Children Under Age Five*. Washington, DC: US Consumer Product Safety Commission; 2014
236. 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
237. Joyner BL, Gill-Bailey C, Moon RY. Infant sleep environments depicted in magazines targeted to women of childbearing age. *Pediatrics*. 2009;124(3). Available at: www.pediatrics.org/cgi/content/full/124/3/e416
238. Moon RY. "And things that go bump in the night": nothing to fear? *J Pediatr*. 2007;151(3):237–238
239. 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
240. 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; July 2010
241. US Consumer Product Safety Commission. *Staff Briefing Package, Crib Bumpers Petition*. Washington, DC: US Consumer Product Safety Commission; May 15, 2013
242. Scheers NJ, Woodard DW, Thach BT. Crib bumpers continue to cause infant deaths: a need for a new preventive approach. *J Pediatr*. 2016;169:93–97.e1
243. 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
244. 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

245. 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

246. Mitchell EA, Taylor BJ, Ford RPK, et al. Dummies and the sudden infant death syndrome. *Arch Dis Child*. 1993;68(4):501–504

247. 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

248. 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

249. 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

250. 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

251. Hauck FR, Omojokun OO, Siadaty MS. Do pacifiers reduce the risk of sudden infant death syndrome? A meta-analysis. *Pediatrics*. 2005;116(5). Available at: www.pediatrics.org/cgi/content/full/116/5/e716

252. Mitchell EA, Blair PS, L'Hoir MP. Should pacifiers be recommended to prevent sudden infant death syndrome? *Pediatrics*. 2006;117(5):1755–1758

253. 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

254. Franco P, Chabanski S, Scaillet S, Groswasser J, Kahn A. Pacifier use modifies infant's cardiac autonomic controls during sleep. *Early Hum Dev*. 2004;77(1-2):99–108

255. 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

256. 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

257. 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

258. 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

259. Nederlands Centrum Jeugdgezondheit. Safe sleeping for your baby. Available at: www.wiegedood.nl/files/download_vs_engels.pdf. Accessed January 10, 2016

260. Foundation for the Study of Infant Deaths. 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

261. Canadian Paediatric Society Community Paediatrics Committee. Recommendations for the use of pacifiers. *Paediatr Child Health*. 2003;8(8):515–528

262. 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). Available at: www.pediatrics.org/cgi/content/full/104/4/e50

263. Benis MM. Are pacifiers associated with early weaning from breastfeeding? *Adv Neonatal Care*. 2002;2(5):259–266

264. Scott JA, Binns CW, Oddy WH, Graham KI. Predictors of breastfeeding duration: evidence from a cohort study. *Pediatrics*. 2006;117(4). Available at: www.pediatrics.org/cgi/content/full/117/4/e646

265. Jaafar SH, Jahanfar S, Angolkar M, Ho JJ. Pacifier use versus no pacifier use in breastfeeding term infants for increasing duration of breastfeeding. *Cochrane Database Syst Rev*. 2011;3:CD007202

266. O'Connor NR, Tanabe KO, Siadaty MS, Hauck FR. Pacifiers and breastfeeding: a systematic review. *Arch Pediatr Adolesc Med*. 2009;163(4):378–382

267. 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

268. 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

269. Eidelman AI, Schanler RJ; Section on Breastfeeding. Breastfeeding and the use of human milk. *Pediatrics*. 2012;129(3). Available at: www.pediatrics.org/cgi/content/full/129/3/e827

270. Larsson Erik. The effect of dummy-sucking on the occlusion: a review. *Eur J Orthodont*. 1986;8(2):127–130

271. American Academy of Pediatric Dentistry, Council on Clinical Affairs. Policy statement on oral habits. Chicago, IL: American Academy of Pediatric Dentistry; 2000. Available at: www.aapd.org/media/Policies_Guidelines/P_OralHabits.pdf. Accessed January 10, 2016

272. 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

273. 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

274. Jackson JM, Mourino AP. Pacifier use and otitis media in infants twelve months of age or younger. *Pediatr Dent*. 1999;21(4):255–260

275. Daly KA, Giebink GS. Clinical epidemiology of otitis media. *Pediatr Infect Dis J*. 2000;19(5 suppl):S31–S36

276. Darwazeh AM, al-Bashir A. Oral candidal flora in healthy infants. *J Oral Pathol Med*. 1995;24(8):361–364

277. North K, Fleming P, Golding J. Pacifier use and morbidity in the first six months of life. *Pediatrics*. 1999;103(3). Available at: www.pediatrics.org/cgi/content/full/103/3/E34

278. 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

279. 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

280. 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

281. 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

282. 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

283. 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

284. American Academy of Pediatrics Committee on Fetus and Newborn; ACOG Committee on Obstetric Practice. *Guidelines for Perinatal Care*. 7th ed. Elk Grove Village, IL: American Academy of Pediatrics; 2012

285. 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

286. Schoendorf KC, Kiely JL. Relationship of sudden infant death syndrome to maternal smoking during and after pregnancy. *Pediatrics*. 1992;90(6):905–908

287. 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

288. 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

289. Mitchell EA, Ford RP, Stewart AW, et al. Smoking and the sudden infant death syndrome. *Pediatrics*. 1993;91(5):893–896

290. Winickoff JP, Friebely J, Tanski SE, et al. Beliefs about the health effects of "thirdhand" smoke and home smoking bans. *Pediatrics*. 2009;123(1). Available at: www.pediatrics.org/cgi/content/full/123/1/e74

291. 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

292. Franco P, Groswasser J, Hassid S, Lanquart JP, Scaillet S, Kahn A. Prenatal exposure to cigarette smoking is associated with a decrease in arousal in infants. *J Pediatr*. 1999;135(1):34–38

293. 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

294. 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

295. 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

296. 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

297. Parslow PM, Cranage SM, Adamson TM, Harding R, Horne RS. Arousal and ventilatory responses to hypoxia in sleeping infants: effects of maternal smoking [published correction appears in *Respir Physiol Neurobiol*. 2004;143(1):99]. *Respir Physiol Neurobiol*. 2004;140(1):77–87

298. 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

299. Mitchell EA, Milerad J. Smoking and the sudden infant death syndrome. *Rev Environ Health*. 2006;21(2):81–103

300. 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

301. 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). Available at: www.pediatrics.org/cgi/content/full/131/3/e770

302. 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

303. 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

304. 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

305. James C, Klenka H, Manning D. Sudden infant death syndrome: bed sharing with mothers who smoke. *Arch Dis Child*. 2003;88(2):112–113

306. 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

307. Rajegowda BK, Kandall SR, Falciglia H. Sudden unexpected death in infants of narcotic-dependent mothers. *Early Hum Dev*. 1978;2(3):219–225

308. 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

309. 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

310. Durand DJ, Espinoza AM, Nickerson BG. Association between prenatal cocaine exposure and sudden infant death syndrome. *J Pediatr*. 1990;117(6):909–911

311. 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

312. Rosen TS, Johnson HL. Drug-addicted mothers, their infants, and SIDS. *Ann N Y Acad Sci*. 1988;533:89–95

313. 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

314. 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

315. 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

316. McGlashan ND. Sudden infant deaths in Tasmania, 1980-1986: a seven year prospective study. *Soc Sci Med*. 1989;29(8):1015–1026

317. 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

318. Hutcheson R. DTP vaccination and sudden infant deaths—Tennessee. *MMWR Morb Mortal Wkly Rep*. 1979;28:131–132

319. Hutcheson R. Follow-up on DTP vaccination and sudden infant deaths—Tennessee. *MMWR*. 1979;28:134–135

320. 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

321. 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

322. 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

323. 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

324. Taylor EM, Emergy JL. Immunization and cot deaths. *Lancet*. 1982;2(8300):721

325. Flahault A, Messiah A, Jougla E, Bouvet E, Perin J, Hatton F. Sudden infant death syndrome and diphtheria/tetanus toxoid/pertussis/poliomyelitis immunisation. *Lancet*. 1988;1(8585):582–583

326. 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

327. 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

328. Immunization Safety Review Committee. Stratton K, Almario DA, Wizemann TM, McCormick MC, eds. *Immunization Safety Review: Vaccinations and Sudden Unexpected Death in Infancy*. Washington, DC: National Academies Press; 2003

329. Miller ER, Moro PL, Cano M, Shimabukuro TT. Deaths following vaccination: what does the evidence show? *Vaccine*. 2015;33(29):3288–3292

330. 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

331. 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

332. Mitchell EA, Stewart AW, Clements M; New Zealand Cot Death Study Group. Immunisation and the sudden infant death syndrome. *Arch Dis Child*. 1995;73(6):498–501

333. Jonville-Béra AP, Autret-Leca E, Barbeillon 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

334. 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

335. 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

336. Fine PEM, Chen RT. Confounding in studies of adverse reactions to vaccines. *Am J Epidemiol*. 1992;136(2):121–135

337. 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). Available at: www.pediatrics.org/cgi/content/full/106/5/e62

338. 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

339. Centers for Disease Control and Prevention. Suffocation deaths associated with use of infant sleep positioners—United States, 1997-2011. *MMWR Morb Mortal Wkly Rep*. 2012;61(46):933–937

340. US Consumer Product Safety Commission. Deaths prompt CPSC, FDA warning on infant sleep positioners. Available at: www.cpsc.gov/en/Newsroom/News-Releases/2010/

Deaths-prompt-CPSC-FDA-warning-on-infant-sleep-position. Accessed September 21, 2016

341. 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

342. Colditz PB, Joy GJ, Dunster KR. Rebreathing potential of infant mattresses and bedcovers. *J Paediatr Child Health*. 2002;38(2):192–195

343. 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

344. Steinschneider A. Prolonged apnea and the sudden infant death syndrome: clinical and laboratory observations. *Pediatrics*. 1972;50(4):646–654

345. Hodgman JE, Hoppenbrouwers T. Home monitoring for the sudden infant death syndrome: the case against. *Ann N Y Acad Sci*. 1988;533:164–175

346. 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

347. 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

348. 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

349. American Academy of Pediatrics Committee on Fetus and Newborn. Apnea, sudden infant death syndrome, and home monitoring. *Pediatrics*. 2003;111(4 pt 1):914–917

350. Hutchison BL, Thompson JM, Mitchell EA. Determinants of nonsynostotic plagiocephaly: a case-control study. *Pediatrics*. 2003;112(4). Available at: www.pediatrics.org/cgi/content/full/112/4/e316

351. 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

352. 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). Available at: www.pediatrics.org/cgi/content/full/119/2/e408

353. Miller RI, Clarren SK. Long-term developmental outcomes in patients with deformational plagiocephaly. *Pediatrics*. 2000;105(2). Available at: www.pediatrics.org/cgi/content/full/105/2/E26

354. Panchal J, Amirsheybani H, Gurwitch 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

355. 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

356. Chadduck WM, Kast J, Donahue DJ. The enigma of lambdoid positional molding. *Pediatr Neurosurg*. 1997;26(6):304–311

357. Laughlin J, Luerssen TG, Dias MS; Committee on Practice and Ambulatory Medicine; Section on Neurological Surgery. Prevention and management of positional skull deformities in infants. *Pediatrics*. 2011;128(6):1236–1241

358. 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

359. van Sleuwen BE, Engelberts AC, Boere-Boonekamp MM, Kuis W, Schulpen TW, L'Hoir MP. Swaddling: a systematic review. *Pediatrics*. 2007;120(4). Available at: www.pediatrics.org/cgi/content/full/120/4/e1097

360. McDonnell E, Moon RY. Infant deaths and injuries associated with wearable blankets, swaddle wraps, and swaddling. *J Pediatr*. 2014;164(5):1152–1156

361. 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

362. 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

363. 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

364. 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

365. 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

366. 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

367. Coleman SS. Congenital dysplasia of the hip in the Navajo infant. *Clin Orthop Relat Res*. 1968;56:179–193

368. 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

369. Manaseki S. Mongolia: a health system in transition. *BMJ*. 1993;307(6919):1609–1611

370. 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

371. Franco P, Scaillet S, Groswasser J, Kahn A. Increased cardiac autonomic responses to auditory

challenges in swaddled infants. *Sleep*. 2004;27(8):1527–1532

372. Patriarca M, Lyon TD, Delves HT, Howatson AG, Fell GS. Determination of low concentrations of potentially toxic elements in human liver from newborns and infants. *Analyst (Lond)*. 1999;124(9):1337–1343
373. Kleemann WJ, Weller JP, Wolf M, Tröger HD, Blüthgen A, Heeschen W. Heavy metals, chlorinated pesticides and polychlorinated biphenyls in sudden infant death syndrome (SIDS). *Int J Legal Med*. 1991;104(2):71–75
374. Erickson MM, Poklis A, Gantner GE, Dickinson AW, Hillman LS. Tissue mineral levels in victims of sudden infant death syndrome I. Toxic metals—lead and cadmium. *Pediatr Res*. 1983;17(10):779–784
375. George M, Wiklund L, Aastrup M, et al. Incidence and geographical distribution of sudden infant death syndrome in relation to content of nitrate in drinking water and groundwater levels. *Eur J Clin Invest*. 2001;31(12):1083–1094
376. Richardson BA. Sudden infant death syndrome: a possible primary cause. *J Forensic Sci Soc*. 1994;34(3):199–204
377. Sprott TJ. Cot death—cause and prevention: experiences in New Zealand 1995-2004. *J Nutr Environ Med*. 2004;14(3):221–232
378. Department of Health. *Expert Group To Investigate Cot Death Theories (Chair, Lady S. Limerick)*. London, United Kingdom: HMSO; 1998
379. Blair P, Fleming P, Bensley D, Smith I, Bacon C, Taylor E. Plastic mattresses and sudden infant death syndrome. *Lancet*. 1995;345(8951):720
380. 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
381. Hamill T, Lim G. Otoacoustic emissions does not currently have ability to detect SIDS. *Early Hum Dev*. 2008;84(6):373
382. Krous HF, Byard RW. Newborn hearing screens and SIDS. *Early Hum Dev*. 2008;84(6):371
383. 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
384. Chan RS, McPherson B, Zhang VW. Neonatal otoacoustic emission screening and sudden infant death syndrome. *Int J Pediatr Otorhinolaryngol*. 2012;76(10):1485–1489
385. Hauck FR, Tanabe KO. Sids. *BMJ Clin Evid*. 2009;2009(315):1–13
386. 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). Available at: www.pediatrics.org/cgi/content/full/118/2/e243
387. Eisenberg SR, Bair-Merritt MH, Colson ER, Heeren TC, Geller NL, Corwin MJ. Maternal report of advice received for infant care. *Pediatrics*. 2015;136(2):e315–e322
388. 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
389. 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
390. 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–e193
391. Moon RY, Calabrese T, Aird L. Reducing the risk of sudden infant death syndrome in child care and changing provider practices: lessons learned from a demonstration project. *Pediatrics*. 2008;122(4):788–798
392. Moon RY, Oden RP. Back to sleep: can we influence child care providers? *Pediatrics*. 2003;112(4):878–882
393. Lerner H, McClain M, Vance JC. SIDS education in nursing and medical schools in the United States. *J Nurs Educ*. 2002;41(8):353–356
394. Price SK, Gardner P, Hillman L, Schenk K, Warren C. Changing hospital newborn nursery practice: results from a statewide "Back to Sleep" nurses training program. *Matern Child Health J*. 2008;12(3):363–371
395. Cowan S, Pease A, Bennett S. Usage and impact of an online education tool for preventing sudden unexpected death in infancy. *J Paediatr Child Health*. 2013;49(3):228–232
396. Colson ER, Joslin SC. Changing nursery practice gets inner-city infants in the supine position for sleep. *Arch Pediatr Adolesc Med*. 2002;156(7):717–720
397. Voos KC, Terreros A, Larimore P, Leick-Rude MK, Park N. Implementing safe sleep practices in a neonatal intensive care unit. *J Matern Fetal Neonatal Med*. 2015;28(14):1637–1640
398. Goodstein MH, Bell T, Krugman SD. Improving infant sleep safety through a comprehensive hospital-based program. *Clin Pediatr (Phila)*. 2015;54(3):212–221
399. 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
400. Magazine Publishers of America; Marketing Evolution. *Measuring Media Effectiveness: Comparing Media Contribution Throughout the Purchase Funnel*. New York, NY: Magazine Publishers of America; 2006