

Sudden Infant Death Syndrome (SIDS or Cot Death)

Infant Sleep, Breast Feeding, and Infant Sleeping Arrangements

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INTRODUCTION

The sudden death of an infant under one year of age which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and a review of the clinical history.

Willinger, 1989, p. 73.

There is no such thing as a baby, there is a baby and someone.

D. Winnicott

For species such as primates, the mother is the environment.

Sarah Blaffer Hrdy, 1999.

In Western industrialized societies pediatric health professionals generally encourage child care practices believed to foster social and biological independence in their infants, as early in life as possible. Birth is commonly viewed as the moment in which the newborn becomes an independent being from the mother, since the mother's body is seen no longer to directly regulate the infant's physiology through the placenta. In these cultures the establishment of early infant/child independence is the developmental goal, autonomy the desired outcome (McKenna, 2002; Trevathan & McKenna, 1994; Young & Fleming, 1998).

An important question raised by anthropological studies, however, is whether historically recent recommended child care patterns that emerge from this view presume infants to be more physiologically independent from their caregivers than they actually are. By ignoring the infant's evolutionary history, such as a human infant's innate need for frequent night-time breast feeds and maternal contact, are critical aspects of the infant's biology being mismatched with rapidly changing patterns of infant care that ultimately deprive the infant of needed if not critical external regulatory maternal stimuli and support (LeVine, Dixon, & LeVine, 1994; Lipsitt, 1981; Lozoff, 1982; Lozoff & Brittenham, 1979; McKenna, 1992, 2001, 2002; Montago, 1978).

Indeed, only relatively recently has the significance of underlying biological regulatory effects induced by maternal contact, caregiving style, and proximity been recognized. The idea that patterns of human contact during sleep (Mosko, Richard, McKenna, Drummond, 1996; Mosko, Richard, McKenna, Drummond, & Mukai, 1997a), degrees of parental emotional responsivity (Kelmanson, 1993), infant sleep positioning (Guntheroth & Spiers, 1992), and method and style of feeding (Gartner, 1997) could significantly affect infant survival is a challenge to the traditional Western paradigm that assumes social care to be, for the most part, unrelated to the autonomy and integrity of the infant's fundamental physiology. Until approximately 20 years ago, for infants living in the industrialized West none of these specific factors was predicted nor thought to play any significant role in affecting infant survival per se (Byard & Krous, 2001; Rognum, 1995). But especially among primate species who are born less neurologically mature at birth than most other mammals, visual, auditory, olfactory, tactile, and movement cues and signals emanating from their caregivers are now described as playing a critical "regulatory" role in how infants breathe, sleep, feed, grow, produce stress hormones, and thermo-regulate (Korner & Thomon, 1972).

[Despite this knowledge, it is not always the case that what the human infant's sensory system is designed ideally by evolution to experience from its parent as, for example, its mother's milk delivered through her breast in conjunction with forms of nighttime cosleeping can be obtained.] This is because learned (culturally favored) patterns of child care are extremely variable and subject to rapid modification and change. Moreover, child care patterns emerge not always from studies of human infant biology or what constitutes empirically validated, *ideal* infant care (assuming there is such a thing), but rather from considerations of how compatible they are with current lifestyles, ideologies, and social values. Moreover,

recommended child care practices generally consider, for example, whether or not particular forms are believed to produce desired adult behavioral or personality characteristics (Super & Harkness, 1982).

The problem is that, despite culture, the infant's biology remains relatively constant (Super & Harkness, 1982). And the most optimal choices as regards infant care (from a biological perspective) are not always the ones encouraged by the societies or families within which choices are made. For example, and independent of culture, for all infants everywhere, method of feeding (exclusive breast, bottle, or mixed), sleeping arrangements (social or alone), and infant sleep position (back, tummy, or side) are known to change neonatal and infant physiology in similar ways. And it appears that the only true micro environment to which a human infant is fully adapted is the mother's body (Hrdy, 1999) and, hence, when deprived of maternal contact infantile deficits may find increased opportunities to conspire therein increasing the chances of a SIDS death (McKenna, 1986; McKenna et al., 1993).

SIDS: HISTORY AND EPIDEMIOLOGY

Since 1962 when the sudden infant death syndrome (SIDS, crib or cot death) was first recognized and defined as a distinct medical entity, approximately 280,000 infants under one year of age (living in the Western industrialized world) have died from this puzzling syndrome. Adding to the sadness and difficulties of having to accept the sudden death of an otherwise healthy-appearing infant, no explanation or description of what actually kills the SIDS victim can yet be offered to grieving parents and relatives. A "diagnosis by exclusion," that is, *death by nothing concluded upon postmortem examination*, is all that continues to qualify a deceased infant to be considered another tragic SIDS fatality.

The most intriguing clue to understanding SIDS remains the unique age distribution of its victims. No other human malady except botulism is so heavily concentrated around such a narrow developmental period. Ninety percent of SIDS deaths occur before 6 months of age, mostly between 2 and 4 months; rarely do such deaths occur beyond 12 months of age (Ariagno & Glotzbach, 1991; Byard & Krouss, 2001).

But the primary causes of SIDS are still unknown. The most compelling general hypothesis is that the fatal

event is related to the control of breathing and/or arousal during sleep. Risks form SIDS, which ordinarily involves infants primarily in the first year of life, and especially between 2 to 12 weeks of life, are suspected to be increased by deficits or assaults to the fetus's nervous system incurred in the womb. Maternal smoking during pregnancy is the most significant risk factor to the fetus prenatally, making the infant susceptible to SIDS postnatally, but maternal smoking cannot explain all infant deaths from SIDS. Smoking during a pregnancy is known to produce low birth weight and/or prematurity, which are confounding factors that also predispose some infants to death.

Other SIDS risk factors include the prone infant sleep position (see below), lack of breast feeding, young maternal age, chaotic lifestyles, membership in an indigenous minority or racially impoverished groups, especially involving mothers who are poor and lack both education and prenatal support (Byard & Krouss, 2001).

Some SIDS victims still seem to differ from surviving healthy babies not so much in kind as in degree (Schwartz & Sagatini, 1988). These SIDS infants appear to suffer from subtle deficits that develop during intrauterine life and are not apparent in the neonate (Valdes-Dapena, 1980, 1988). Researchers believe that the actual expression of the fatal deficit is likely to be influenced by, if not dependent on, a number of co-factors that converge at a vulnerable moment in the infant's life (Barnett, 1980; Byard & Krouss, 2001; Rognum, 1995). Nobody can yet delimit all of the appropriate SIDS co-factors or explain why co-factors seem to have differential effects on infants. But it is extremely likely that certain factors are more relevant to some SIDS victims than to others. For example, for some infants, a contributing SIDS risk factor might be the lack of breast feeding (Fredrickson, Sorenson, & Biddle, 1993; Hoffman, Damus, Hillman, & Krongrad, 1988); while for another it might be sleeping face down (prone) in the presence of an upper respiratory infection that diminishes the potency (muscle tone) of airway passages (Blackwell, Saadi, Raza, Weir, & Busuttill, 1993). In certain predisposed infants, efficient respiratory control might also be jeopardized by infantile hypothermia, induced by atmospheric temperature, humidity, or too much bundling up (overheating) in cold weather (Ariagno & Glotzbach, 1991; Fleming et al., 1996).

One group of researchers suggests that between 28% and 52% of SIDS victims found "faced straight down" may have actually suffocated, especially those who were sleeping on beanbag cushions. Unable to dislodge themselves

from the pockets formed by such cushions, the infants may have been forced to rebreathe lethal doses of their own expelled carbon dioxide (Byard & Krous, 2001; Kemp & Thach, 1991).

Though this may be changing with current recommendations to position infants on the back or supine position for sleep, SIDS occurred most frequently in winter, and infants tended to die in the early morning or evening hours, when the infant is out of sight of the caregiver and presumably asleep. However, SIDS is also known to occur while babies are riding around in strollers, sitting in car seats, dozing in baby carriers, and even sleeping on their mother's chests, following a breast feeding episode (Blair et al., 1999; Fleming et al., 1996; Guntheroth & Spiers, 1992; McKenna, 1986).

The SIDS population is exceedingly heterogeneous. No single, consistent criterion or pathological marker can be used to either predict potential SIDS victims or identify them upon postmortem autopsy. Nor is there an animal model of SIDS; it is not known to occur in any

species other than humans (see Byard & Krous, 2001; McKenna, 1986; Rognum, 1995; for reviews).

In the United States, SIDS rates are highest among both Native American and poor African Americans whose mothers are less than 20 years of age, smoke during their pregnancies, are unmarried, and lack access to prenatal care (Hoffman et al., 1988). SIDS rates are lowest in diverse Asian (see below), Swedish, Finnish, Norwegian, English, and Israeli populations, where mothers tend to be older, do not smoke during pregnancy, and place their infants in the supine (back) or side position for sleep. Especially in Asian cultures where SIDS rates are consistently low, infants typically room share and sleep within arm's reach of the mother, or they sleep as they do in China and Japan in direct contact with their mother's body during the first few years of life while the mothers are breast feeding intensively (Gantly, Davies, & Murcott, 1993; Nelson et al., 2001).

SIDS is virtually unknown in China, Thailand, Cambodia, Vietnam, and Nepal, as Figure 1 indicates.

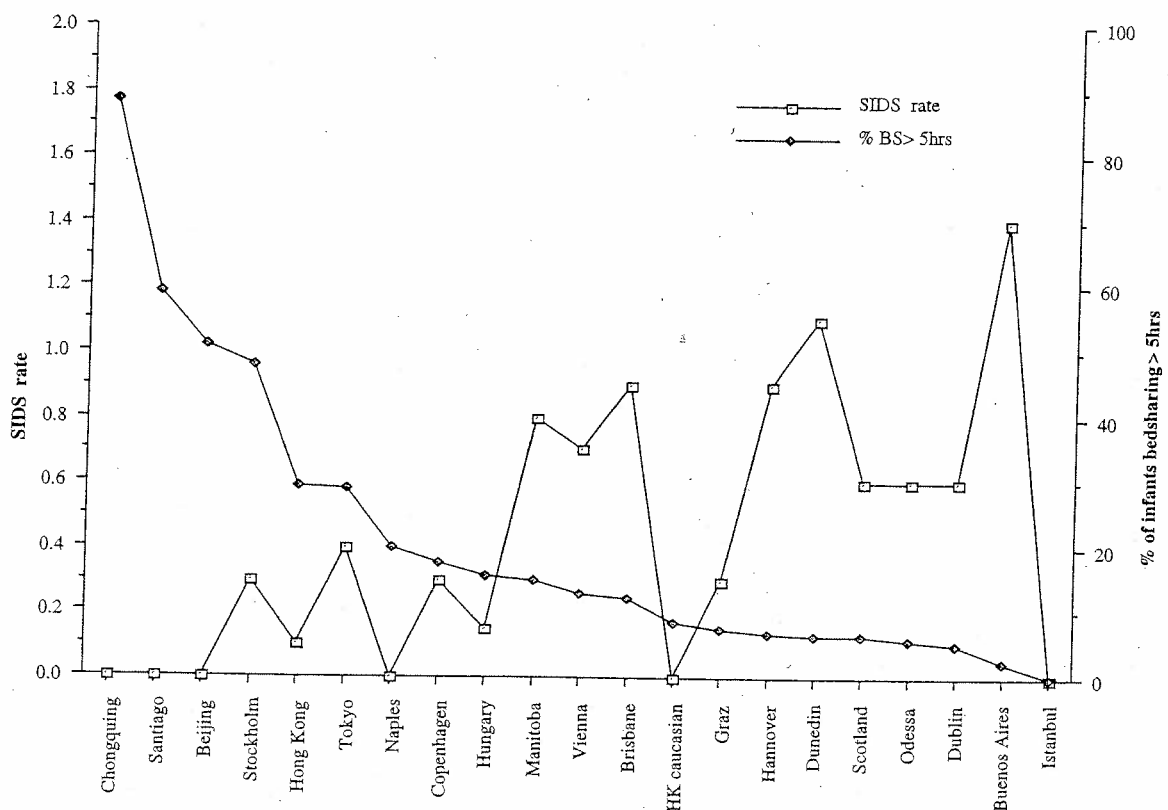


Figure 1. SIDS rates (1995) and the prevalence of bedsharing >5 h per night across cultures. (Source: Nelson et al. (2001). *Early Human Development*, 62, 43–55.)

In contrast, the rates among Inuit Alaskan Indians can range from 4 to 6 per 1,000 live births, and as high as 9–15 per 1,000 live births among impoverished native Canadian Indians, the Cree (Wilson, 1990).

One study revealed that about 18% of all SIDS deaths involve premature infants. Low birthweight is a risk factor, as is the experience of one or more of an "apparent life-threatening event" (ALTE), characterized by a loss of muscle tone accompanied by gasping or choking, listlessness, color changes, or a cessation of breathing. Approximately 6% of infants who experience an ALTE die from SIDS (Ariagno & Glotzbach, 1991; Byard & Krous, 2001).

Various studies report that before their deaths, some SIDS infants slept for longer periods of time, awoke less often, and had more difficulty awakening or arousing than healthy infants with whom they were compared (Einspieler, Widder, Holzer, & Kenner, 1984; Harper et al., 1988). At birth, some SIDS infants received lower Apgar scores and gained weight more slowly. And some exhibited less frequent but more sustained heart-rate variability, and fewer but longer breathing pauses (apneas) (Harper et al., 1981; Hoppenbrouwers, Hodgman, Arakawa, & Serman, 1989).

In a major study in the United States conducted in the 1980's funded by the National Institutes of Child and Maternal Health, 756 SIDS victims were compared with 1,600 control infants. The research team found that many SIDS victims had had colds and bouts of diarrhea or vomiting within two weeks of death. A significant number had also exhibited droopiness, irritability, or some form of breathing distress involving a rapid heartbeat 24 hours before they died. However, researchers believe that all these symptoms were acting in secondary fashion rather than as primary causes of SIDS (Hoffman et al., 1988). As few as 10% of all SIDS victims had had symptoms associated with a potential SIDS event before their deaths. This includes full-term infants with clinical histories of apneas as well as preterm underweight babies who experience "apneas of prematurity" (Ariagno & Glotzbach, 1991; Byard & Krous, 2001; Rognum, 1995).

Only a relatively low number of symptomatic infants actually die of SIDS. As a result, the medical community is engaged in a volatile debate about whether or not infants with a history of repeated apneas should be sent home from hospital with breathing monitors. At any given time, between 40,000 and 45,000 monitors are put to use in the United States; yet, no data indicate that

monitors prevent SIDS deaths, and no data suggest how or under what circumstances infants die from SIDS when monitors are in use. At present, the effectiveness of home monitors in preventing SIDS deaths is highly questionable (Byard & Krous, 2001).

SLEEP PHYSIOLOGY RESEARCH AND SIDS

The architecture of infant sleep, breathing patterns, and arousal have been intensely studied by SIDS researchers, as have the neuro-structural, neuro-chemical, and physiological systems that underlie, influence, or control these activities (Mosko et al., 1997, 1996). The possibility of fast-acting bacteria (infections) in the nose and respiratory tract, in combination with environmental factors (prone sleeping on soft mattresses, overheating, and maternal smoking) in addition to infantile internal deficits are known to increase SIDS risks (Byard & Krous, 2001).

Researchers note that SIDS tends to occur after abnormalities of the cardio-respiratory control system have failed to monitor some combination of oxygen levels, breathing, heart-rate rhythmicity, body temperature, or the arousal responses needed to reinitiate breathing after a normal breathing pause or apnea. Essentially, the cardio-respiratory system is thought to collapse (Byard & Krous, 2001; Harper et al., 1981; Hoppenbrouwers et al., 1989; Kahn, Picard, & Blum, 1986; Kinney et al., 1995; McCulloch, Brouillette, Guzzetta, & Hunt, 1982; Rognum, 1995; Schwartz & Sagatini, 1988; Shannon, Kelly, & O'Connell, 1977 for reviews).

The unfolding pattern of sleep itself, including how and when human infants arouse or awaken from sleep, is believed to be controlled by the primitive brain stem, located at the central base of the brain. This area is composed of clusters of differentiated cells that receive and send messages to and from the heart, hormonal centers, lungs, muscles surrounding the ribs, diaphragm, and airway passages, as well as structures that specifically help to balance the proper amounts of oxygen and carbon dioxide (CO₂) in the blood. Also controlled by the brain stem is the amount of time spent in various stages of sleep during any given sleep period—for example, in light sleep, (stages 1 or 2), in deep stages (stages 3 and 4), or in rapid eye movement (REM, i.e., active sleep) (Mosko et al., 1996; Mosko et al., 1997; Harper et al., 1981; Hoppenbrouwers et al., 1989). Sleep architecture,

including the form and timing of arousals, are all influenced by external stimuli as well, such as feeding method and the presence or absence of a co-sleeping partner, and must, therefore, be considered alongside any analysis of internally-based sleeping mechanisms (Mosko et al., 1997).

Kinney et al. (1995) studies an area of the brain (the arcuate nucleus) located on the ventral surface of the brain stem, an important area that monitors the proper balance of CO₂ and oxygen. Recall that when CO₂ builds up in the blood, the respiratory neurons are activated to expel it, thereby causing fresh oxygen to be inhaled, reducing the acidity of the blood. A significant number of SIDS victims compared with control infants had fewer "acetylcholine binding sites" in this area of the brain. This suggests that in a variety of different circumstances, prone sleeping included, infants may not have the optimal or even minimal ability to arouse to reinitiate breathing following some type of apnea or exposure to their own exhaled CO₂ if it is trapped; for example, in a mattress as the baby lies face down, or if the infant is under thick blankets. Or it might mean that infants simply cannot arouse to breathe after particularly long breathing pauses or apneas.

CHILD CARE PRACTICES 2002: INSIGHTS FROM PARENTAL SOCIAL BEHAVIOR RATHER THAN FROM BIOLOGY?

In the past 12 years child care practices have proven to be the single most important set of factors for reducing the chances of an infant dying of SIDS (Guntheroth & Spiers, 1992; McKenna, 2002; McKenna & Bernshaw, 1995). The discovery that merely placing infants in the supine (back), rather than in the prone (belly) sleep position, could reduce SIDS rates by as much as 50%–90% continues to astonish many SIDS researchers around the world. Indeed, the prone infant sleep position is likely as important to understanding the probability of SIDS as are the primary deficits. If SIDS researchers were asked just a decade ago to prioritize SIDS research areas according to how likely they were to yield clues about reducing SIDS risks quickly and significantly, child care practices over which both parents and professionals assert control would not have been ranked very high. Instead, knowledge of brain mechanisms that control breathing likely

would have been considered where the answer to SIDS death would be found. Yet, epidemiological findings across cultures now show consistently that, in the absence of maternal smoking, where child care patterns include the back (supine) infant sleep position, exclusive breast feeding, increased infant holding, maternal emotional responsiveness, routine daily structures (compared with chaotic households), SIDS rates are low (Azaz et al., 1992; Balarajan, Raleigh, & Botting, 1989; Blair et al., 1999; Davies, 1985; Farooqui, Perry, & Beeves, 1991; Fleming et al., 1996; Gantley et al., 1993; Kibel & Davies, 2000; Lee, Chan, Davies, Lau, & Yip, 1989; Mitchell & Scragg, 1995; Nelson et al., 2001; Takeda, Yamashita, & Miyazaki, 1987; Tasaki, 1988).

From an anthropological perspective, which is integrative and holistic, it is not surprising that child care practices in relationship to SIDS prevention should prove to be so important. Several different lines of evidence indicate that the social care of infants is virtually synonymous with physiological regulation (see McKenna, 1986; McKenna & Mosko, 2001). In short, human infants need contact with the mother's body, and lots of it! Indeed, no skills or capabilities of the newborn make sense except in light of its mother's body. It is the dyad, and not the infant, that constitutes the major unit for study and analysis. Winnecott's famous statement, "There is no such thing as a baby, there is a baby and someone" is, from a scientific and cultural perspective, perhaps one of the most profound and accurate descriptions proposed for infants in the last century. This is because at birth the human infant brain is only 25% of its adult brain weight. Human infants are the least neurologically mature primate of all the primates, and subject to the most extensive external regulation and support, for the longest period of time. This suggests that, in order for human infants to survive, and for human (parental) reproductive success to be maximized, natural selection likely favored the co-evolution of highly motivated caregivers on one hand, alongside highly responsive infants on the other—infants designed to respond to and depend on external parental sensory stimuli. From both an evolutionary and developmental perspective, then, parental contact and proximity with infants (while awake and asleep) can be seen to represent a developmental bridge for the infant, extending into postnatal environments the role that the mother played prenatally in regulating important aspects of her infant's continuing development (McKenna, 1995; McKenna & Mosko, 2001).

Hundreds of laboratory studies from the last 20 years confirm this view. These studies demonstrate, for example, the beneficial physiological effects of mothers holding their preterm and newborn infants using the "kangaroo" method of baby care, or skin-to-skin contact, which has the effects of increasing the infant's skin temperature, stabilizing heart rates and reducing apneas and crying, and improving sleep and digestion. All of these findings are consistent with an evolutionary perspective on the how human infants develop optimally (Anderson 1991; Field, 1995; Kagan, 1984; Konner, 1981; Konner & Wothman, 1980; Korner, Guilleminault, Van den Hoed, & Baldwin, 1978; Ludington-Hoe, 1990; Ludington-Hoe, Hadeed, & Anderson, 1991; Ludington-Hoe et al., 1992; Reite & Field, 1985). Laboratory studies of non-human primates and other mammals also confirm that even short-term separation of primate infants from their mothers induces deleterious physiological consequences such as loss of skin temperature, cardiac arrhythmias, depressed immune responses, and increased stress involving adreno-cortico-throphic hormone release and, in some cases, a reduction in the number of antibodies in the infant's blood (Reite & Capitanio, 1985; Reite, Harbeck & Hoffman, 1981; Reite, Seiler, & Short, 1978; Reite & Snyder, 1982; Stewart & Stewart, 1991; for a review).

Natural selection appears to have favored infant responsiveness to postnatal parental sensory stimuli among primates in much the same way that it favored fetal responsiveness to, and regulation by, the mother's physiological and/or behavioral status prenatally, by way of fetal-maternal physiological exchanges. Thus, it is a reasonable assumption that research into the effects of adult contact on human infant sleep physiology might likely reveal some clues to the SIDS mystery.

All human beings practiced diverse forms of parent-infant co-sleeping up until the last 100 years or so, and contrary to popular thinking the Western societal practice of infants sleeping in social isolation represents an historically and biologically novel sleep environment, the consequences of which (either short or long term) have never been considered, nor experimentally explored (McKenna & Mosko, 2001; Mosko, McKenna, Dickel, & Hunt, 1993). Surveys of contemporary infant sleeping practices reveal that approximately two thirds of the world's cultures habitually practice mother-infant co-sleeping on the same bed or sleeping surface, and the fraction is much higher if the definition of co-sleeping is extended to include sleeping in the same room (Barry &

Paxson, 1971). Thus, solitary sleeping among infants is a relatively recent and mostly Western innovation.

In fact, compared with quickly changing Western cultural ideas about where and how infants "should" sleep, we have suggested that the mechanisms that control human infant sleep are unable to change as quickly, and, where infants sleep alone, their sleep, breathing, thermoregulation, and arousal mechanisms are functioning in environments for which they were not designed by evolution (McKenna & Mosko, 2001; see also McKenna, 1986, 2002; McKenna, 1991, 1993). Since pediatric sleep researchers have never explored nor considered the impact of social sleep on early neonatal and infant development, we do not know if the recent shift by some world cultures to solitary sleep environments is beneficial, benign, or deleterious, or under what particular social or physical circumstances the effects of co-sleeping can be altered (McKenna, 1995). The question that must be asked is: Why has Western science never seriously asked if it is safe for human infants to sleep alone?

SIDS RATES ACROSS CULTURES

As reported elsewhere in more detail (McKenna, 1996; McKenna & Mosko, 1991) if natural selection designed the developing human infant's sleep, breathing, and arousal patterns in association with parental contact, as we contend, this perspective gives us an initial basis for postulating (and possibly for better understanding) how and why related physiological control systems might go awry, or somehow function less efficiently when and if sleep environments diverge from the evolutionarily stable ones. If we assume for the moment that all known SIDS risk factors can be held constant, and that no genetic factors predispose some populations more than others to SIDS, then we should find lower SIDS rate in societies, or in segments within a society, in which parent-infant co-sleeping occurs.

As Figure 1 shows, cross-cultural data from urban, industrial, Asian countries generally support aspects of this prediction, but such comparisons are, admittedly, difficult. In Japan, for example, where infant-mother co-sleeping on futons continues to be the norm (Takeda, 1987), current published rates for SIDS are some of the lowest in the world (0.15/1,000 births in Tokyo, 1978; 0.053/1,000 in Fukuoka, 1986; and 0.22/1,000 births in Saga) (Tasaki et al., 1988). The most recent estimate for

the national SIDS rate in Japan is 0.3 per 1,000 live births (see McKenna, 2002, for data). These data do not, of course, prove that co-sleeping is protective against SIDS. It may well be that SIDS is under-reported in Japan, or that it is misdiagnosed as infantile suffocation. Japanese medical scientists have not participated in international SIDS research studies to the extent that American and European scientists have, so the postmortem procedures they employ to identify SIDS may not be appropriate. Nevertheless, these low SIDS rates deserve explanation and further research.

In 1985, Davies reported on the rarity of SIDS in Hong Kong. He used postmortem diagnostic protocols that, on review for a follow-up study by Lee et al. (1989), were judged comparable with Western diagnostic standards by John Emery, a renowned SIDS researcher from Great Britain. Davies found that even in a context of poverty and overcrowded conditions, where the incidence of SIDS should be high, the rates were 0.036 per 1,000 live births, or approximately 50–70 times less common than in Western societies. This finding is even more surprising because breast feeding is not common (of 175 infants at 2, 4, and 6 months of age, the percentage of infants nursing was 9%, 4%, and 2%, respectively), although co-sleeping and the supine sleep position for infants represent the cultural norm.

Davies proposed that proximity to the parent while the infant is asleep may be one reason why the rates are so low, as well as the typical (supine) sleeping position of Chinese infants. The author asked, "whether the possible influences of life style and caretaking practices in cot death are being underestimated in preference for more exotic and esoteric explanations" (Davies, 1985)—a viewpoint not unlike that of Azaz et al. (1992) and Emery (1983), who also implicate, for some English infants, the importance of caregiving environments and other behavioral-socioeconomic factors. A follow-up on Davies' work by Lee et al. (1989) confirms the relative rarity of cot deaths in Hong Kong, finding a slightly higher rate of deaths per 1,000 live births (0.3, compared with 0.04/1,000 reported by Davies).

A third study confirmed the rarity of SIDS in infants of Asian origin living in England and Wales, particularly infants of mothers born in India and Bangladesh, but also infants of mothers with African origins. As the authors point out, Asian women have few illegitimate births, few births at younger ages, and few of them smoke (Balarajan et al., 1989)—all of which seem to reduce the risks of

infants dying of SIDS. No mention was made of any possible differences in sleeping patterns that could explain the lower SIDS rate among the Asian subgroup, although it is likely that these infants were sleeping in proximity to their parents.

These low SIDS rates continue in Asian ethnic groups even after they immigrate to Western (non-co-sleeping) cultures, where most continue their traditional caregiving practices which include co-sleeping (Gantley, Davies, & Murcott, 1993). One study reports that among five Asian American subgroups living in California, the incidence of SIDS ranged from a low of 0.9 deaths per 1,000 live births to a high of 1.5 per 1,000. The variability was related directly to the duration of residence in the United States: the longer the group lived in the United States, the higher the SIDS rates (Grether, Schulman & Croen, 1990), leading us to ask if the trend toward higher SIDS rates reflects the adoption of more "American" patterns of infant sleep management (i.e., solitary infant sleep), among other things.

Data from other industrial societies, among which at least some general comparisons of SIDS rates can be made, also tend to support the general hypothesis that increased nocturnal contact between the parent and infant may reduce the chances of SIDS among some infants. For example, in cultures in which infants are less likely to have their own room or in which infants are more likely to be in close proximity to a parent during the night, SIDS rates tend to be lower (Blair et al., 1999).

Most recently, the International SIDS Child Care Study reports that some of the cultures in which either no SIDS are reported—or its citizens are unaware of SIDS-like deaths—are those cultures that report the highest bed-sharing rates and other forms of co-sleeping, such as room-sharing with parents, are the norm. Moreover, in that same study, it is clear that many cultures that report the lowest bed-sharing or room-sharing have the highest SIDS (Nelson et al., 2001) (See Figure 1).

Even under the best of circumstances, admittedly SIDS is difficult to diagnose. Because it is relatively rare, and because postmortem procedures for identifying SIDS are not necessarily standardized internationally, it is difficult to interpret differences in SIDS rates across cultures. Since parent-infant co-sleeping is hypothesized to be relevant only to some subclasses of potential SIDS victims, proving the hypothesis becomes even more difficult.

In a series of sleep laboratory studies of mother-infant bed-sharing our research team studied "normal" infant sleep in a context that attempted to match the

context within which both night-time breast feeding and infant sleep evolved: the mother–infant co-sleeping microenvironment. This new paradigm for studying infant sleep emerges from a biocultural and evolutionary perspective on infants, and the mother–infant dyad, which assumes the species-wide (universality) and biological appropriateness of infant–parent co-sleeping. This perspective builds from the premise that under safe sleeping conditions, and for the vast majority of infants, infant–parent co-sleeping should be inherently protective and promote infant survival and, hence, parental reproductive success. For reasons presented in great detail elsewhere, my colleagues and I have hypothesized that the sensory-rich co-sleeping micro environment may change the sleep physiology and architecture of the human infant in ways helpful in resisting some types of SIDS (McKenna, 1986, 1995; McKenna & Mosko, 2001; McKenna et al., 1993; Mosko et al., 1993; Mosko et al., 1997a).

Our studies which have now been confirmed by others show that: (1) co-sleeping mothers and infant exhibit high levels of arousal overlap, both longer (epochal) and smaller physiologically defined transient arousals; (2) infants exhibit more frequent state shifts, that is they move from one stage of sleep to another, or awaken more frequently, while co-sleeping and spent more time, at the same time, in the same state of sleep and wakefulness while in the same bed; and (3) as compared with infants sleeping alone, on average bed-sharing infants spent less time in deep stages of sleep (stage 3 or 4) and co-sleeping mothers intervened during the arousals of their infants possibly prolonging the duration of those arousals (see McKenna, 1986; McKenna & Mosko, 2001; McKenna et al., 1997; McKenna, Mosko, Dungy, & McAnich 1990; Mosko et al., 1993; Mosko et al., 1996b; Richard, Mosko, & McKenna, 1996).

Behavioral analysis of mothers and infants observed from the video tapes taken through infra-red cameras revealed that (1) during the bed-sharing night infants face toward each other (face-to-face) for the vast majority of the night (between 72% and 100% of the time (Richard et al., 1996) especially after nursing; (2) on average, on bed-sharing nights the frequency of breast feeding episodes doubled, average intervals between feeds were reduced by half, while the average total nightly duration of breast feeding virtually tripled on the bed-sharing night compared with the solitary nights (McKenna et al., 1997); (3) on average, mothers induce between 10% and 27% of their infants' total behavioral arousal patterns while

co-sleeping, while in turn, the infant can induce over half of their mother's total arousals although the total number of minutes slept increased for both the mother and infant on the bed-sharing night, contrary to popular conceptions; (4) bed-sharing mothers inspect, re-blanket, reposition, and adjust their infants while sleeping over four times more frequently (on average) than they do when each partner sleeps alone (Barone, 2001); (5) through a combination of active or passive embracing, touching, enclosing, and breast-feeding, bed-sharing infants and mothers are in physical contact ranging from 28% to 99% of the observed period compared with a low of 2%, to a high of 14% on solitary sleep nights (see Barone, 2002; McKenna et al., 1994; Richard et al., 1996).

One additional observation is that when infants sleep in the same bed with mother, the mother almost always places her infant for sleep in the safer supine position; but on the solitary nights, when the same infants are placed in a crib by their mothers in an adjacent room, mothers often placed their infants in the more dangerous prone position, even though on the co-sleeping nights always the supine position was used (Richard et al., 1996). Supine, or back-sleeping makes a great deal of sense when it is observed that the infant cannot breast feed, or control access both to and from the breast if sleeping on its belly. Back-sleeping infants arouse more frequently and have far more control over their universe than do prone-sleeping infants, and supine-sleeping infants experience greater protection from SIDS (Barone, 2001; Guntheroth & Spiers, 1992).

HOW CAN THESE FINDINGS BE RELATED TO SIDS PREVENTION?

At present, five laboratories around the world are currently studying and quantifying both the physiological and behavioral differences between bed-sharing and solitary-sleeping mother–infant pairs. In at least three of these laboratories the findings suggest that bed-sharing, occurring under safe environmental circumstances, could potentially make it more difficult for the range of SIDS defects to find expression. For example, the finding that co-sleeping mothers and infants exhibit synchronous, partner-induced physiological arousals, although not very surprising, is potentially important because of the suspected relationship between infantile arousal deficiencies and some cases of SIDS. As described earlier,

Kinney et al. (1995) found that some SIDS victims had reduced acetylcholine receptor sites in the brain stem, suggesting that arousals may be affected adversely. Bed-sharing, by increasing the type and number of arousals, could potentially compensate for such a deficiency (Mosko et al., 1997a, 1997b). It may be that co-sleeping provides the infant with increased opportunities to practice arousing, thereby becoming more proficient at it. Moreover, these responses increase the overall amount of physiological variation (stage shifting, for example) experienced throughout the infant's night-time sleep period. Overall, co-sleeping partner-induced arousals may facilitate the synchronous maturity and coupling of cardiorespiratory systems and the various central nervous system subsystems involved in arousal and/or the shift from sleep to wakefulness. It is possible that these linkages among the infant's physiological subsystems, which interact during arousals, may not occur as easily, as often, or as quickly if infants regularly sleep alone (McKenna et al., 1994).

Our finding that bed-sharing infants spend less time in deep stages of sleep, that is, stages 3 and 4, and more time in stages 1 and 2 is also potentially important. If these findings are confirmed, it may suggest that solitary sleep environments may accelerate the maturation of deep sleep, possibly before arousal mechanisms are maximally efficient to handle arousals during some physiological crises, such as a prolonged apnea. Less deep sleep, and more light sleep from which arousals to terminate apneas is easier, could be adaptive for infants.

Also, the face-to-face orientation and proximity that occurs often during co-sleeping raises the possibility that the infant's atmospheric CO₂ is elevated enough at times to stimulate respiration (Mosko et al., 1997a). In addition to examining the actual distance between the mother's and infant's faces from the videotapes, we are currently measuring the CO₂ content of air over a range of distances from women's faces (Mosko et al., 1998), and the amount of CO₂ available to the infant when its face is partially covered by a blanket. The concentration of CO₂ measured at distances comparable with those that often separate co-sleeping mothers and infants was within the range shown in steady-state breathing studies to increase ventilation in young infants.

Our finding that in the co-sleeping environment mothers continuously inspect, attend to, and more frequently (visually) "check out" their infants also elucidates another type of arousal occurring in the co-sleeping but not

the solitary sleep environment. At least 6–10 times during the co-sleeping night, mothers lean over and inspect their infants. Often during these periods, mothers reposition their infant's blankets, sometimes repeatedly, as if ventilating. Mothers appear to be ensuring that the infant is not in any apparent danger or distress (McKenna & Mosko, 2000; Young & Fleming, 1998). Though we cannot say for certain what motivates this behavior, it seems reasonable to speculate that these infant-directed behaviors increase the likelihood of a mother discovering and intervening to reverse a potentially dangerous condition or situation. As mentioned above, perhaps these activities induce infant arousals at times when there would have been no arousal had the infant been sleeping alone. Such maternal-induced infant arousals from diverse physiological states may provide the infant with practice in arousal (Mosko et al., 1993).

Finally, that infants exhibit significantly more breast feeding activities while bed-sharing (twice as many breast feeding sessions, for three times the total nightly duration as solitary sleeping infants) is potentially very important with respect to protection from SIDS (McKenna et al., 1997). Two recent epidemiological studies suggest that, indeed, breast-feeding lowers the risk of SIDS (Hoffman et al., 1988; Mitchell et al., 1992), while two others suggest that the extent of protection may be dose specific, that is, the more breast feeding that occurs the greater the protection (Fredricksen et al., 1993). While a protective effect has not been found in every study (Gilbert, Wigfield, & Fleming, 1995) many international SIDS prevention campaigns, including those in the United States, encourage or recommend breast feeding as a way to reduce SIDS.

The positive association found by Scragg, Stewart, Mitchell, Ford, & Thompson (1995) between Maori maternal smoking, bed-sharing, and increased SIDS risk is important, and justifies a recommendation against bed-sharing where mothers smoke. And the data may well apply to the other at-risk populations that Mitchell et al. (1992) cite where maternal smoking, drug and alcohol use, dangerous furniture, and other identifiable bed-sharing hazards, where multiple known risk factors interact with bed-sharing, endanger infants... But the special adverse characteristics of particular populations on which positive statistical associations are found between bed-sharing and infant deaths do not justify sweeping conclusions, namely that under all circumstances and in all families and cultures, bed-sharing, or co-sleeping in

general, in whatever form it takes, causes, or necessarily increases, the risk of SIDS and should therefore always be advised against. Such unqualified conclusions and recommendations increasingly are being rejected by the majority of scientists (see *Mothering Magazine*, Special Issue, *Sleeping With Baby: Top Scientists Speak Out*, September–October Issue, 2002).

If a scientifically valid understanding of the potential benefits or risks of co-sleeping/bed-sharing are ever to be achieved, anthropologists, forensic pathologists, and epidemiologists must work together. New ethnographically sensitive and appropriate epidemiological variables and categories must be adopted which more precisely capture, describe, and classify the diverse social and physical environmental factors that characterize and differentiate co-sleeping environments and the participants from solitary sleeping environments and participants. Moreover, it is imperative that we re-conceptualize from a biological and not strictly a cultural point of view the biological appropriateness of breast feeding and parents and infants sleeping alongside each other, and that the existence of dangerous co-sleeping conditions is no more an argument against the potential benefits to infants and parents of sleeping together than the existence of dangerous solitary infant sleep environments constitutes a valid argument against the safety of all solitary infant sleep. No environment is risk free (McKenna, 1995).

Finally, just as a SIDS can occur independently of known risk factors in a solitary sleep environment, without solitariness being thought of as a "causal" factor, so too can a SIDS death occur while co-sleeping, just as independently and without any contributory "causal" role played by the parents. The tendency to assume some contribution to the SIDS death by virtue of the parents presence, but not to consider parental absence as a contributory factor in a SIDS death, reflects hidden cultural assumptions and ingrained cultural ideologies and expectations.

CONCLUSION

The causes of SIDS are complex. There is no one type of SIDS death and, hence, there will never likely be just one way to prevent it. While all types of research on SIDS must, of course, continue, I argue here that regardless of whatever the primary causes of SIDS prove to be, they will only be understood alongside and in relation to the

infant's biologically expectable micro environment, which involves reference to mother's caregiving behavior and method of feeding. It will be critical to remember that since SIDS occurs when human infants sleep, the species-wide, "normal" and healthy pattern of infant sleep is social. In fact, so entwined is the biology of mother–infant co-sleeping with nocturnal breast feeding, that any study that purports to understand biologically "normal" infant sleep without understanding how these two activities interrelate must be considered incomplete, inaccurate, or both (McKenna & Bernshaw, 1995). That infant–parent co-sleeping represents the evolutionarily stable and most adaptive context for the development of healthy infants is not to say that modern sleeping structures or conditions are always safe. But it is important to differentiate between the act of mothers and infants sleeping in proximity and contact which is adaptive, from the conditions within which they do so, which may not be.

While much research is needed to test the hypothesis that increased parental contact during the night will reduce the chances of an infant dying of SIDS, a recognition of the legitimacy of diverse sleeping arrangements for infants, including diverse forms of co-sleeping, is necessary in order to reach a complete understanding of SIDS, a sleep disorder for which the existing research paradigms have proven inadequate.

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Tobacco Use in Medical Anthropological Perspective

Merrill Singer

INTRODUCTION

Tobacco is known to be the one commercially sold product that if used as directed by the manufacturer will lead to certain disease and death. Additionally, unlike a genetically caused disease, the health consequences of tobacco use are directly tied to behavior and to the cultural and social structuring of people's ideas, actions, and relationships. For all of these reasons, tobacco use should be of primary interest to medical anthropology. In fact, however, as discussed below, there have been relatively few focused anthropological accounts of tobacco use in a cultural and social context. This avoidance is noteworthy, reflecting both the fact that topics of study within medical anthropology (like other fields) accord greater or lesser social rewards for researchers as well as the fact that in the West, where most medical anthropologists are found, tobacco use lacks extensive symbolic or other cultural embellishment. However, from a strictly health standpoint, tobacco use is of far greater direct consequence than most topics regularly studied by medical anthropologists. Further, because of the role it has played over time in inter-group social relationships, especially between colonial powers and colonized peoples and between dominant and subordinate social classes, tobacco also has had an enormous indirect impact on human health.

TOBACCO AND HEALTH

The significant health consequences of smoking are now widely known. Three commonly lethal diseases, particular, have been closely linked to the use of tobacco: coronary heart disease, lung cancer, and chronic obstructive pulmonary disease. Other fatal or disabling diseases known to be caused by or made worse by smoking include peripheral vascular disease, hypertension, and myocardial infarction. Smoking also causes cancer of the mouth, throat, bladder, and other organs. As anthropologists Marvin Nichter and Elizabeth Cartwright (1991, p. 237) argue, smoking damages the health of families in three additional ways: it leads to or complicates chronic illness thereby reducing the ability of adults to care for and socialize children; it diverts scarce household resources from healthier items; and it exposes children to secondary smoke, a known cause of disease. Current estimates are that 3,000 lung cancer deaths and 62,000 deaths from coronary heart disease in adults who do not smoke are caused each year in the United States alone by exposure to second-hand tobacco smoke. Among children, second-hand smoke is associated with sudden infant death syndrome (SIDS), chronic middle ear infections, and respiratory infections such as asthma (National Cancer Institute, 1999).

In 1989 the World Health Organization estimated that worldwide 2.5 million people die each year from