49 The potential benefits of infant-parent co-sleeping in relation to SIDS prevention: Overview and critique of epidemiological bed sharing studies

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In western industrialized societies, pediatric health professionals generally encourage child care practices believed to foster social and biological independence in infants, as early in life as possible. Birth is commonly viewed as the moment at which the newborn becomes an independent being from the mother, since the mother's body is no longer seen as the direct regulator of 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 (1). However, an important question raised by other disciplines, especially anthropology, is whether the recent recommended child care patterns that have emerged from this view presume infants to be more physiologically independent from their caregivers than they actually are. By ignoring the infant's evolutionary history, are we mismatching the more conservative (i.e., unalterable) aspects of the infant's biology with rapidly changing patterns of infant care, patterns that clearly promote the social best interests of parents, but not necessarily or always the psychological or biological best interests of infants?

Our research on infant-parent co-sleeping is attempting to define and quantify the social and physiological differences between exclusively breast-fed co-sleeping and solitary-sleeping infants, and to evaluate these differences in terms of their relationship to known SIDS risk factors. This research emerges from a biocultural and evolutionary perspective that assumes the species-wide (universality) and biological appropriateness of infant-parent co-sleeping. we should expect that, under safe sleeping conditions, and for the vast majority of infants, this sleeping arrangement should be inherently beneficial. For reasons presented in great detail elsewhere, we hypothesize 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 (2–6).

The first part of this paper will briefly discuss the justification for studying infant-parent co-sleeping and for proposing the hypothesis. The second section describes our co-sleeping research methods and presents a limited summary of some of our data, collected mostly from the first phase of an analysis of video tapes of mother-infant pairs sleeping together and apart over successive nights. Our purpose here is to provide an overview of selected early results and to suggest how the data relate to other SIDS research findings. This discussion will illustrate what kinds of questions emerge when the legitimacy of the co-sleeping environment is acknowledged.

In the third part of the paper, I explain why I disagree with the New Zealand Cot Death Study research team's unqualified conclusion that bed sharing causes SIDS (7–9). I show why such a sweeping conclusion is inaccurate, oversimplified, and not universally applicable, and suggest that its lack of specificity may do more harm than good. I suggest that if this conclusion is accepted without distinguishing between the diverse forms that bed sharing/co-sleeping can take, and the variable circumstances within which bed sharing safety is determined, we may well deprive some infants of a type of parental contact and proximity in which optimal health, including possible protection against SIDS, is provided. Certainly, if accepted uncritically, the New Zealand conclusion will lead some to think quite erroneously (with perhaps tragic consequences) that a mother's body, rather than being a source of nurturance, emotional satisfaction, and infant survival, is no more than a hazard over which neither the mother nor the infant has control.

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TO Rognum (ed.) *Sudden Infant Death Syndrome: New Trends in the Nineties*

Why study infant-parent co-sleeping in relation to SIDS?

Given that all human beings practiced parent-infant co-sleeping until about 150 years ago, the western societal practice of having infants sleep in isolation represents a historically and biologically novel sleep environment, the consequences of which (either short- or long-term) have never been considered nor experimentally explored. Compared with quickly changing western cultural ideas about where and how infants "should" sleep, the mechanisms that control human infant sleep are unable to change as quickly. Furthermore, where infants sleep alone, their sleep, breathing, and arousal mechanisms are functioning in environments for which they were not designed by evolution. Since pediatric sleep researchers have never explored the impact of social sleep on early neonatal and infant development, we do not know if the recent shift by some cultures to solitary-sleep environments is beneficial, benign, or deleterious (6).

What is known from anthropological studies is that continuous contact and carrying (including the co-evolution of infant-parent co-sleeping with breast-feeding as one integrated adaptive system) have characterized the human infant's developmental experiences for well over five million years. While this alone does not prove that separate sleeping arrangements for infants are necessarily detrimental, it does mean that co-sleeping environments should also be considered when questions are raised about infant sleep and infant sleep disorders. Unfortunately, studies of both "normal" infant sleep and the sudden infant death syndrome have universally failed to acknowledge the legitimacy of co-sleeping environments (2–6).

At birth, the human brain is only 25% of its adult weight. Indeed, the human infant is neurologically the least mature primate of all and is subject to the most extensive external regulation and support, for the longest period. This suggests that, for infants to survive, and for human reproductive success to be maximized, natural selection likely favored the co-evolution of highly motivated caregivers on the one hand, alongside highly responsive infants on the other—that is, infants designed to respond to and depend on external parental sensory signals and/or regulatory stimuli. From both an evolutionary and a 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 (5).

Recent laboratory studies showing beneficial physiological effects of mothers' holding their preterm and newborn infants using the "Kangaroo" method of baby care, or skin-to-skin contact (stabilizing temperature and heart rate and reducing apnea and crying), are consistent with this perspective (10). Other studies show that, for other slow-developing primate species, even short-term separation from their mothers induces deleterious physiological consequences in infants, such as loss of skin temperature, cardiac arrhythmias, depressed immune responses, and increased stress involving adrenocorticotrophic hormone release. It is reasonable to propose that natural selection favored infant responsivity to postnatal parental sensory stimuli (whether the infant is asleep or awake) in much the same way that it favored fetal responsivity to, and regulation by, the mother's physiological and or behavioral status prenatally, through fetal-maternal physiological exchanges. It follows that this proposition justifies research into the effects of adult contact on infant sleep physiology.

Insofar as contemporary human infant biology is more conservative and changes much more slowly than cultural attitudes about where infants "should" sleep, I must emphatically disagree with Mitchell and Scragg's suggestion that "co-sleeping has outlived its historical usefulness" (8, p. 389). Recent cultural changes in western societies stressing individualism over interdependence may indeed support this statement; but the question for infants is, has co-sleeping outlived its *biological* usefulness? I think not. As reviewed elsewhere (2), the scientific evidence suggesting negative socioemotional *and* physiological consequences of infants' sleeping socially distant from their parents far exceeds evidence suggesting inherent negative effects of increased contact or proximity. Moreover, not one scientific study has documented the presumed socioemotional, psychological, or physiological benefits of solitary infant sleep, except where "benefits" are defined in terms of parental interests or other cultural values or expectations, or where forms of social sleeping occur under unsafe conditions.

Mother-infant co-sleeping in the laboratory

Our first study consisted of five mother-infant pairs (infant age between 2.5 and 4.5 months) who spent one night in our laboratory sleeping in the same bed while undergoing all-night, simultaneous polysomnography (3). Our second study consisted of three mother-infant pairs who spent three consecutive nights in the laboratory. On the first two nights, they slept in adjacent rooms; on the third night, they slept together in the same bed. Regardless of sleep environment, mothers and infants were simultaneously monitored all night using standard polysomnographic procedures as described below (4–6).

We are now finishing the first year of a three-year study that will eventually include a total of 50 Hispanic mothers and their healthy, breast-feeding, 3-month-old infants. As assessed by sleep logs kept by potential participants for a two-week period before the study,

eligible mother-infant pairs are either routine co-sleepers or routine solitary sleepers. A strict criterion is used to differentiate the two groups. Routine co-sleeping requires co-sleeping for at least four hours per night, five days per week; routine solitary sleeping is defined as co-sleeping (same bed) no more than twice per week for any part of the night. All mothers in the study have normal pregnancies and deliveries, and infants are healthy and have normal developmental histories.

Beginning with an "adaptation night" in the laboratory, mother-infant pairs sleep as they routinely do at home. On one of the next two nights (randomly chosen), the mother and infant sleep again in the routine condition, on the other night, in the non-routine condition. Continuous, all-night polysomnographic and video recordings using infrared lamps are performed nightly. A single polygraph records all standard physiological parameters simultaneously in the mother and infant. Axillary temperature and oxygen saturation are also recorded in the infants.

Electrodes are attached first to the infants, since they retire before mothers at home. Mothers position their infants for sleeping as usual each night with no instructions. Recordings begin when infants retire at their usual time. Mothers retire later, also at their normal bedtimes. They sleep in rooms adjacent to their infants' rooms on solitary-sleeping nights, with the bedroom doors open so that they remain in auditory contact. Mothers respond to infant crying and other distress on an ad lib basis, performing all caregiver interventions themselves each night.

Polygraph recordings are scored for sleep stages in 30-second epochs according to accepted criteria. The Rechtschaffen and Kales (11) system for young adults is used for the mothers, and the scoring system for 3-month-olds developed by Guilleminault and Souquet (12) is used for the infants. Identification of sleep-wake stages in both scoring systems depends on three simultaneous parameters: EEG, EOG, and chin EMG. Five sleep stages are identified in adults: Stage REM plus four stages of non-REM (NREM) sleep delineated as Stages 1, 2, 3, and 4. In the infant, only three stages are defined: Stage REM, Stage 1-2, and Stage 3-4. In the process of data reduction, Stages 1 and 2 in the adult are combined to obtain Stage 1-2, and Stages 3 and 4 are similarly combined for comparability with infant sleep stages.

These epochal systems of sleep-stage scoring assign to each 30-second epoch either wakefulness (W) or one stage of sleep on the basis of the predominant (greater than 50%) sleep or wakefulness pattern occupying the epoch. Although awakenings of 15 seconds or longer that meet these criteria (i.e., epochal awakenings, EWs) are automatically identified by the epochal system, shorter-duration arousals occupying less than 50% of an epoch are not. Because of our interest in all arousal phenomena in sleep, we quantify these transient arousals (TAs) using accepted criteria (13).

Video recordings of all individuals on all nights are hand-scored in real time in their entirety. All observable behaviors are recorded and, where appropriate, their duration measured.

Overview and discussion of selected early findings and their relevance to SIDS research

The most robust and consistent finding under which more specific findings and their potential significance can be discussed is that co-sleeping mothers and infants exhibit overlapping (mutual) arousals, many of which appear to be partner-induced. Unfortunately, not a great deal is known about mutual arousals in co-sleeping environments since arousal phenomena in the laboratory have been studied only among solitary-sleeping infants. Furthermore, clinical models of what constitute "normal" nocturnal sleep-wake patterns of infants are based exclusively on studies of solitary-sleeping infants conducted in the early 1960s, when breast-feeding in the United States was at an all-time low. Bottle-fed infants wake up less frequently than do breast-feeding infants (14), suggesting that widely accepted models of "normal" patterns of awakening are probably not appropriate for either breast-feeding or co-sleeping infants.

The high percentage of mother-infant overlapping arousals may be accounted for multiple factors, not the least of which is their proximity to one another. For example, even though there is enough room in the bed to sleep without touching, the mother-infant pairs analyzed thus far spent between 28% and 99% of the night in direct physical contact ($n = 6$). This means that any kind of sudden sound or movement made by one co-sleeping partner is likely to be sensed by the other, although not all arousals elicit overt responses by the other partner. Moreover, co-sleeping results in mothers' nursing their infants almost three times as frequently as they do when their infants are sleeping in adjacent rooms, just a few feet away. But they do so for a much shorter average duration. It seems that the opportunity to nurse quickly and conveniently, to use nursing as a means of calming a fussy or restless infant, and to unintentionally stimulate infants to "want" to nurse (possibly because of emitted maternal odors (15)) are all factors that could explain the increased frequency of nursing in the co-sleeping environment that, in turn, contributes to a higher number of overlapping mother-infant arousals.

Whether increased nursing sessions in the co-sleeping environment constitute a benefit for the infant is difficult to determine. But one possibility is that the increased sucking and milk digestion that occurs during co-sleeping encourages more Stage 1-2 (light) sleep. It is easier for infants to arouse from lighter sleep than from

deeper (Stage 3-4) sleep, should it be necessary to do so. It is also possible that increased nursing sessions lead to more rapid daily infant weight gain, although future analysis will have to confirm this proposal

Some investigators have suggested that neurological abnormalities may cause an "arousal deficiency" that is manifested as SIDS in some infants (16-18). If the arousal deficiency theory is supported by additional work, our finding that mothers appear to induce different types of arousals, including feeding arousals, is potentially important, especially since, as two investigators remind us, "arousal constitutes a stimulus for breathing" (19).

DiPietro et al (20) compared bottle-fed infants with nursing infants and found that the latter experienced longer heart beat intervals and increased heart rate variation, two physiological characteristics associated with reduced risk of dying of SIDS (21); however, it is not known if these characteristics are a result of inborn or environmental factors. DiPietro et al. do suggest, however, that breast-fed neonates exhibit differences in physiological organization, including "elevated vagal tone". They suggest that previous work "showed a monotonic relation between vagal tone and severity of clinical dysfunction in at-risk neonates as well as lower vagal tone in at-risk infants compared with full term neonates, even when gestational age is corrected ... [H]igher vagal tone is a reflection of better physiological status" (20, p. 471).

Mothers adjust their infant's body position more often while co-sleeping, inducing infant arousals sufficient to cause the infant to shift from one stage of sleep to another, thereby potentially inducing more physiological variability in the infant's sleep experiences than it would experience if sleeping alone. We have speculated elsewhere that increased physiological variability during co-sleeping may facilitate maturational synchrony among developing neurological subsystems that underlie arousals (breathing, heart rate, and motor nuclei); these systems must function in concert to produce protective arousals in response to infant cardiorespiratory crises (2-6).

There is yet another reason why these infant-directed maternal manipulations during co-sleeping may be important. Studies from Great Britain, Norway, Belgium, Australia, New Zealand, Denmark, Ireland, and Tasmania found that placing infants in the supine position for sleep significantly reduces their chances of dying of SIDS (22). While our research cannot contribute directly to this discussion, it can elucidate the relationship between sleep environment and the mother's choice of infant sleep position, as well as the effects that this choice has on infant sleep physiology. For example, the video recordings show that while the infant is always an active participant in its own care in the co-sleeping environment, it cannot feed from its mother's breasts easily, if at all, while lying prone. Interestingly, when placing their infants in cribs for solitary sleep, mothers can, and often do, place them in the prone position; but the same mothers place their infants in the supine or side position when sharing a bed with them.

Our impression is that the supine (or safer) position permits the infant to assert more control over its environment; it can, for example, elicit the mother's attention (e.g., by whacking the blankets), gain access to the nipples when hungry, and turn away from them when satiated. Use of the head, arms, and legs by the infant to communicate needs is especially facilitated by the supine position. Insofar as breast-feeding and co-sleeping evolved in tandem and represent the human evolutionary context of infant sleep, the fact that most of our mothers placed their infants on their backs for most of the night while sleeping next to them suggests that the supine sleep position may well be the more universal (species-wide) position. If so, it should not be surprising that it is safer than the prone position.

Over twenty years ago, Douthitt and Brackbill (23) found that supine-sleeping newborns experienced twice as much motor activity during sleep and more awakenings than did prone-sleeping newborns, and this has been confirmed by Kahn et al. (24) (see below). Since the goal both of parents and of health professionals has always been to promote sleep and not awakenings, these data provided an argument for why infants should sleep prone. We now recognize the risks of prone sleeping, and it is hypothesized (yet still unproven) that some infants who die of SIDS cannot arouse to reinitiate breathing; that is, they may sleep too deeply, in "adult-like sleep", before sufficiently mature arousal mechanisms in the brain are developed (25). On this basis, one can speculate that the supine sleep position might well be safer than the prone position precisely because of the increased arousal and motor activity that accompanies it. A recent study by Kahn et al. (24) lends support to this possibility. They compared the differences between 40 infants who routinely slept supine and 40 infants who routinely slept prone. They found that prone-sleeping infants were associated with a significant increase in sleep duration and decreases in the number and the duration of arousals.

Another consistent finding in which we are interested is that, while co-sleeping, mothers and infants spend a great deal of time in the face-to-face orientation. This raises the possibility that the infant's atmospheric CO_2 is, at times, sufficiently elevated to stimulate infant respiration (26). In addition to examining the actual distance between the mothers' and infants' faces from the video tapes, we measured the CO_2 content of air over a range of distances from women's faces (26). The concentration of CO_2 measured at distances comparable to those that often separate co-sleeping mothers and infants is within the range needed to increase ventilation in young infants, according to steady-state breathing studies. We look forward to reporting these data next year.

We found that, in the co-sleeping environment, mothers continually inspect and attend to, and more frequently (visually) "check out", their infants. These observations elucidate another type of arousal occurring in the co-sleeping but not the solitary-sleep environment. At least 6 to 10 times during the co-sleeping night, mothers lean over and inspect their infants. Often during these periods, mothers reposition their infants' blankets, sometimes repeatedly, as if ventilating. They may stroke, kiss, or whisper to their infants spontaneously. Mothers appear to be ensuring that their infants are safe, comfortable, and not in any apparent distress. Although we cannot say for sure what motivates these infant-directed behaviors, it seems reasonable to speculate that they increase the likelihood of a mother's discovering and intervening to reverse a potentially dangerous condition or situation. As mentioned above, sometimes these activities appear to induce infant arousals at times when there would have been no arousal had the infant been sleeping alone.

We have speculated previously that such "maternal induced infant arousals" *occurring during diverse physiological states* may provide the infant with practice in arousal, and simultaneously reduce the amount of time infants spend in deeper stages of sleep (Stage 3–4), from which it is more difficult to arouse should they confront a respiratory crisis (2–4, 6). In other papers, we have discussed the decline in the total amount of time co-sleeping infants spend in Stage 3–4 of sleep in comparison with when they sleep alone (27). Currently, we are further examining the average duration of Stage 3–4, as well as its temporal distribution in both the solitary and co-sleeping environments.

Some critical comments on the conclusion in the New Zealand Cot Death Study that bed sharing causes SIDS

Bed sharing, or smoking and bed sharing?

We disagree with the conclusion reached by Mitchell (7) and Mitchell and Scragg (8) that bed sharing causes SIDS. It is a puzzling conclusion, since their data show quite clearly that, for non-smoking mothers, "bed sharing was not associated with a significant increased risk" (7, p. 388). They concede that "bed sharing may be protective for a small group of infants" (p. 389) and recognize that the lowest SIDS rates in the world are found in co-sleeping/bed sharing cultures and immigrant subgroups. In fact, the Pacific Islanders had the highest rate of bed sharing in one study (42.2%), yet have the lowest SIDS rates! The authors claim that, absent other known risk factors, "exposure to maternal smoking is required for bed sharing to be associated with an increase risk of SIDS" (p. 389). Yet, in a discussion of the "strength of association", the authors omit this qualification, thereby equating dangerous environments and conditions within which bed sharing can occur, with the act of bed sharing itself. They state: "There appears to be a threefold increased risk associated with bed sharing.... [T]his is the same order as the increased risk seen in prone sleeping position" (p. 388). I believe this statement is misleading.

Certainly, the positive association found between Maori maternal smoking, bed sharing, and increased SIDS risk is important, and justifies a recommendation against Maori bed sharing where mothers smoke. And the data may well apply to the other at-risk populations that Mitchell (7) and Mitchell and Scragg (8) cite where maternal smoking and bed sharing are practiced together, and where multiple known risk factors interact to damage and endanger infants or fetuses. The special characteristics of the populations on which the findings are based do not justify the investigators' sweeping conclusion, namely, that under all circumstances and in all families and cultures, bed sharing, in whatever form it takes, causes, or necessarily increases, the risk of SIDS and should therefore always be advised against. SIDS rates reported by our Japanese colleagues Nishida and Fukui (28) at these meetings make this conclusion, at very least, imprecise; the vast majority of Japanese infants co-sleep with at least one parent in the first year of life, as is true of many other Asian and immigrant cultures, yet their SIDS rates remain the lowest in the world (29–31).

I suggest that a lack of appreciation of the inherent benefits and biological significance of parental contact with and proximity to the infant, as well as cultural judgements, lead the researchers to move beyond the identification of dangerous circumstances that can arise during bed sharing to conclude that bed sharing alone causes SIDS. If, as Mitchell (7) claims, the extremely low rates of SIDS in Asian and immigrant bed sharing cultures can be explained by the *absence* of known risk factors while co-sleeping, one wonders why the *presence* of those same risk factors, rather than bed sharing per se, does not serve as the focus of their conclusion about what is causing increased SIDS risk? The fact that these data are being interpreted in this way, with such certainty and so early, is, I suggest, due to ingrained cultural values, unique to the western industrialized world—values that hold that even under the best of conditions, infant-parent co-sleeping or bed sharing is not desirable, but rather inherently harmful and to be avoided (32).

Bed sharing/co-sleeping is not a unitary phenomenon

Mitchell's (7) and Mitchell and Scragg's (8) conclusions are problematic because, among other things, they treat bed sharing as if it were a unitary, categorically consistent phenomenon practiced the same way across and within all cultures, circumstances, and settings. But studying bed sharing is not as simple as determining if an infant slept prone or supine. Infant characteristics interact with micro-environmental factors relevant to

bed sharing and ultimately affect the infant in his or her setting. To understand this interplay, information must be collected on the physical and or mental characteristics (and numbers and ages) of co-sleepers, the types and qualities of sleeping structures (especially the firmness and make of the mattress or sleep surface), the quantities and constituents of bedding material and objects on which co-sleepers sleep (as well as frequency of cleaning), and the motivations, values, and reasons that determine why infants sleep on or in the bed (33, p 666), not only on the night of death, but also at other times. These are just a few of the important variables that have been insufficiently addressed in studies of bed sharing. These and other variables must be classified and statistically addressed before valid conclusions about either the risks or benefits of bed sharing can be drawn.

Are bed sharing and co-sleeping the same thing? What is bed sharing?

The answers to these questions are not as obvious as they seem. None of us working in this field have yet operationalized or standardized our terms. Suppose, for example, that a mother carries her infant into a room and lies down with the infant to sleep on a hard foam mattress that covers the entire floor. In this context, the floor serves as the "bed"; suppose that this mother lays the infant down on one side of the room, walks to the other side, lies down and, during the next hour or so, while on her back, gradually maneuvers closer and closer to the infant. The unqualified conclusion reached by the New Zealand team suggests that at some point, as the distance separating the mother and the infant decreases, she becomes a risk to her infant—that there is something intrinsic to her association with the infant that increases the likelihood of SIDS. What the New Zealand study cannot address is, at which point in the contact or proximity does her presence become a risk factor, and what mechanisms could be at work here? Also, since they are sleeping on a floor with a hard surface, is this a bed? Is it bed sharing, or co-sleeping? And if it is not a bed, is there still a risk? Furthermore, given that biology specifically designed the infant to lie next to the mother for sleep and nocturnal nursing, and that the infant's survival depends on that proximity and or contact, is it not biologically implausible that something intrinsic to the relationship actually contributes to the infant's dying? If the mother does not smoke, are the answers to these questions the same as they would be if she were a smoker? Is this scenario equivalent to or different from bed sharing among some or all ethnic groups involved in the New Zealand study and the case-control studies used to support the conclusion?

The current public health message conveyed in New Zealand and delivered at this conference is that bed sharing, is bed sharing, is bed sharing. But before we can consider such a proposition, the behavior of bed sharing must be defined operationally and the definition standardized, especially if conclusions influenced by this proposition are supposed to hold cross-culturally. Currently, there is no evidence to suggest that what constitutes "bed sharing" in one culture is necessarily what constitutes it in another. More research is required to describe and distinguish the many different ways parents and infants can sleep close together on the same surface.

Bed sharing with healthy infants in healthy micro-environments is not the same as bed sharing with unhealthy babies in unhealthy environments

The case-control studies that Mitchell and Scragg (8) and Mitchell (7) use to justify their conclusion that bed sharing causes SIDS all involved high-risk populations, where factors such as maternal smoking, young unsupported mothers, poverty, and low education are all salient. Breast-feeding is also underrepresented in these populations. The consequences of bed sharing are likely different for infants whose mothers smoked during their pregnancies than for infants whose mothers did not smoke. Consider also that bed sharing can produce very different biological consequences for infants as changes occur not only in specific features of the bed sharing environment, but also in the infants' developmental status. Both the biological and the social function of parental contact, including co-sleeping, change as a consequence of development and, in cases of sudden death, age at death in relation to bed sharing should be useful in distinguishing, for example, accidental suffocation from different types of true SIDS.

Carpenter and Shaddick's (34) work further elucidates factors in the sleep environment that must be considered in assessing SIDS causation, whether the infant is sleeping alone or socially. They compared SIDS cases with controls and found that pillows were more often used in SIDS cases (51% vs. 29.3%); in the SIDS cases, pillows and mattresses were more often described as soft (34% vs. 12%, and 40.7% vs. 22.5%, respectively). Smialek et al. (35) found that of 492 infant deaths in Wayne County, Michigan, USA, 26% were attributed to unsafe sleeping conditions. Four infants were asphyxiated by plastic mattresses or covers, and another by an older, damaged mattress that did not fit the bed frame. Two infants died alone in an adult bed, while another was wedged between twin beds pushed together.

Although his study remains controversial, Bass (36) found that of the 26 SIDS cases he and his colleagues reviewed after SIDS diagnosis, 13 were rediagnosed as asphyxia. In 8 of the 13, bedding in a crib or bassinet was argued to be responsible, while in 6 cases where the parent slept with the infant at the time of its death maternal obesity, maternal drug use, maternal exhaustion, or illness were implicated. When a positive

association between bed sharing and SIDS is made and conclusions are drawn, these factors must be given attention

SIDS can occur during bed sharing without the co-sleeping partner's having anything to do with the death

SIDS researchers in general, and pathologists in particular, seem to believe that SIDS cannot occur independently of the sleep environment *if* another person (usually a parent) is present in the same bed at the time of death. There is an inclination to suspect that a SIDS death cannot occur independently in the co-sleeping context, but only in the solitary (crib) context. The assumption that co-sleepers have a contributory (i.e., triggering) effect on the occurrence of SIDS but solitude does not, or that the infant must have been overlayed in the co-sleeping environment, illustrates the powerful role that cultural ideologies play in influencing SIDS research strategies and assumptions. Cases of overlaying, accidental suffocation, and other accidents are not SIDS, although in discussions of bed sharing, they invariably become proxies for it, usually to strengthen the argument that bed sharing is inherently dangerous.

Contemporary SIDS medico-historians such as Norvenius (37) are arguing that, during the last three hundred years, mothers were accused unfairly of having accidentally overlayed their infants when, in fact, many of these infants likely died of SIDS. It is particularly ironic that this conclusion coincides with that of the New Zealand study, which provides grounds for re-establishing a maternal contribution to sudden infant death. Where mothers in history did not smoke or did not live in smoke-filled and underventilated rooms, and did not give their infants significant quantities of whiskey or other drugs (opiates), as was the custom to make them sleep (38), it should not be concluded *post facto* that co-sleeping necessarily contributed to infant death.

Similarly, where other risk factors such as maternal smoking are absent, it is both unfair and unjustified to suggest to contemporary mothers who lost their infants to SIDS while co-sleeping that this behavior necessarily contributed to death. If some "types" of SIDS can be expressed under all environmental circumstances, apparently independent of currently identified risk factors, we must expect that co-sleeping should not have to be *completely* or even mostly protective in order for it to be protective for *some* infants. And where it is not protective, we should not conclude because of cultural biases against co-sleeping that it must have somehow contributed to the cause of death.

Bed sharing is vastly underreported in western societies among lower-risk groups, but more likely to be reported among high-risk groups

Studies that examine bed sharing habits, even where SIDS is not involved, are particularly susceptible to recall and reporting biases because bed sharing is so negatively portrayed. It is anything but a neutral subject. In western societies, bed sharing (more commonly called co-sleeping) is discouraged by health professionals, and it is not considered a legitimate sleep-management strategy for infants or children. This is especially true in the countries in which the case-control studies used by Mitchell and Scragg were conducted (Great Britain, the United States, Scotland, Australia, and New Zealand). In these countries, the individuals who value bed sharing, and are thus more willing to report the behavior, are members of minority groups that exhibit multiple SIDS risk factors; furthermore, even when they do not share their beds with their infants, they retain the highest SIDS rates. Individuals from groups with much lower SIDS rates, with whom bed sharing groups are compared, often accept the negative public evaluations of bed sharing and are less likely to admit to this practice if and when they are asked (4).

For example, similar to the Maori in New Zealand, African-Americans in the United States view bed sharing positively and thus are more likely to report the behavior. However, Euro-Americans (like non-Maori New Zealanders of European descent), who as a group exhibit neither comparative, multiple SIDS risk factors nor positive evaluations of bed sharing, are less likely to admit to bed sharing, even if they practice it. These differences in cultural attitudes likely lead to exaggerated, if not artificial, differences in the reported frequencies of bed sharing among the groups being compared. My guess is that among more middle-class, affluent, Caucasian populations, bed sharing is vastly underreported; hence, in case-control studies where socially, politically, and ethnically diverse groups are being compared, the statistics will make bed sharing appear to be more dangerous than it actually is, while its role in protecting some infants will remain hidden. Until co-sleeping or bed sharing is discussed by professionals in a more positive way, it will be difficult to control these methodological problems.

Studying poor African-Americans living in Washington, D.C., Luke (39), whose case-control study of SIDS was used prominently by Mitchell and Scragg to support their conclusion, seems to have anticipated at least one of these methodological difficulties. He warned against the very conclusion that Mitchell and Scragg draw, that is, that bed sharing alone necessarily causes SIDS. He states that "the importance of a single trained and experienced interviewer to define the specific sleeping arrangements of SIDS victims cannot be over stressed. Perfunctory interviewing by questionnaire, for example, is an unacceptable alternative because of the complexities of the personal issue to be addressed" (27, p. 383). To assure conceptual and interpretive validity, both the wording and content of research questions should reflect ethnographic knowledge and sensitivities.

The conclusion that co-sleeping elicits a deadly response from the infant does not make biological sense

To assure biological plausibility, pediatric epidemiological findings should be checked, rechecked, and evaluated against developmental studies, including clinical and anthropological studies of infant biology, socioemotional development studies, psychobiological studies, and evolutionary studies of human infancy and parenting behavior. Mitchell's (7) and Mitchell and Scragg's (8) conclusion that co-sleeping causes SIDS was made in a vacuum; it was based only on epidemiological data. I believe this is a serious shortcoming. The intersection of diverse data should be central to the process of arriving at the most accurate assessment of the findings, and in establishing whether a particular conclusion likely applies universally (i.e., is valid on a species-wide basis), or has limited utility. In this way, the likelihood of simplistic, inaccurate, or, indeed, potentially dangerous conclusions (which can do more harm than good) can be circumvented while, at the same time, the specific micro-environmental factors that have proved risky in all developmental settings can be pinpointed and appropriate public health information provided

How an alliance between pediatric epidemiologists and social scientists might help avert another medical tragedy

The tragic recommendation to place infants prone for sleep underscores the potential advantage of forging a new alliance between epidemiologists and other scientists, including social scientists whose research focuses specifically on the *unique* biobehavioral and developmental characteristics of the human infant. When the prone sleeping position for infants was first recommended, the cultural values in western industrialized societies strongly supported the view that infant-parent co-sleeping was always harmful to psychological development, and bottle-feeding with cow's milk was superior to breast-feeding. Given this cultural ideology, which judged both of these time-tested child care practices to be inappropriate, there was no reason to explore (or even question) how infant sleep position related to the infants' breast-feeding periodically throughout the night as they slept next to their mothers.

Evolutionary studies reveal that co-sleeping and breast-feeding were part of the same adaptive complex; they were inseparable and inevitable. Had we considered optimal infant sleep position in this social and biological context, we might have discovered much sooner that the supine sleep position is more logical and probably safer for infants. For example, the supine but not the prone position permits movement to, and away from, the nipple for feeding, and only while on its back can the infant use its arms and legs to swipe (or kick) successfully at objects (including blankets and sheets) that might fall over its face, obstructing its air passages. Movement, arousal, and control are maximized while infants sleep supine (4, 23, 24). These are all factors that help infants to confront crises during sleep.

Looking ahead

In the past decade, we have witnessed unparalleled development in SIDS research, resulting in quite unexpected findings. The discovery that, merely by placing infants in the supine rather than prone sleep position, SIDS risks could be significantly reduced continues to astonish many SIDS researchers, and has convinced some that the overall environment within which SIDS deficits find expression may be as important as the primary deficits themselves. Before this discovery, if investigators had been asked to prioritize SIDS research areas according to how likely they were to yield clues about reducing SIDS risk quickly and significantly, child care practices—over which both parents and health professionals have control—would not have been ranked very high (except for abstinence from smoking during and after pregnancy). This underscores the necessity of coming to understand both the range of the human infant sleep arrangements, as practiced worldwide, and the kinds of research biases we hold regarding what constitutes "normal" infant sleep.

An evolutionary perspective provides an unbiased starting point for infant sleep and SIDS research precisely because it forces us to consider the species-wide or universal context of infant sleep physiology, rather than how infant sleep is experienced only in our own geographical or cultural settings. To better understand the biology underlying infant sleep, or what factors might cause it to go awry, we must at some point study it in the social or co-sleeping environment within which it was designed and proved itself adaptive—that is, the co-sleeping context. Anthropological data, including cross-cultural, paleontological, and archaeological data, are critical here. We suggest also that, by beginning from an evolutionary perspective, researchers are best able to appreciate the extent to which infant and maternal sleep physiology and behavior are entwined in adaptive ways, and that for the extremely altricial human infant, social care *is* physiological care.

By examining, in detail, ethnic differences in child care practices as they occur in the infant's micro-environment, especially where SIDS rates are extremely low or extremely high (33), we may well find important clues to SIDS etiology. By combining such research with physiological studies of co-sleeping that examine normal sleep biology in the co-sleeping context, we have a chance to understand both the benefits and risks associated with bed sharing. But premature conclusions based on the reduction of complex variables

like bed sharing to simple ones—which is what I believe Mitchell and Scragg have done—ultimately will not serve our goal to reduce SIDS. Without qualifying what *kind* of bed sharing may be dangerous, those authors confuse what is mostly an intrinsically adaptive sleeping arrangement for an inherently maladaptive one. For the future, we need to create a research alliance that brings SIDS epidemiologists together with scientists from other fields whose expertise lies in the social behavior and development of the infant. Such an alliance might have prevented the unqualified recommendation to turn infants prone for sleep, a recommendation made without considering the context within which infant sleep position evolved. Neither I nor my colleagues have ever suggested that sensory contact in the form of co-sleeping can eliminate SIDS, or that it is always practical or safe for all infants and families. In fact, at this point in our research, co-sleeping is to be neither recommended nor advised against. The question is not whether we should co-sleep or not, but rather whether we should study co-sleeping and become more open-minded about its potential role in contributing to infant survival. While much research is needed to prove our hypothesis, we believe that a recognition of the legitimacy of diverse sleeping arrangements for infants, including co-sleeping, is necessary to reaching a complete understanding of SIDS, a disorder for which existing research paradigms have proved inadequate.

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