There is no such thing as infant sleep, there is no such thing as breastfeeding, there is only *breastsleeping*

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Recently Mobbs et al. 2015 describe the need for, and benefits of, immediate and sustained contact, including cosleeping, to establish an appropriate foundation for optimal human infant breastfeeding, neonatal attachment and brain growth. To further support this model, we propose a new concept, 'breastsleeping', aimed to help both resolve the bedsharing debate and to distinguish the significant differences (and associated advantages) of the breastfeeding–bedsharing dyad when compared with the nonbreastfeeding–bedsharing situations, when the combination of breastfeeding–bedsharing is practiced in the absence of all known hazardous factors. Breastfeeding is so physiologically and behaviourally entwined and functionally interdependent with forms of cosleeping that we propose the use of the term breastfeeding to acknowledge the following: (i) the critical role that immediate and sustained maternal contact plays in helping to establish optimal breastfeeding; (ii) the fact that normal, human (species wide) infant sleep can only be derived from studies of breastfeeding dyads because of the ways maternal–infant contact affects the delivery of breastmilk, the milk’s ingestion, the infant’s concomitant and subsequent metabolism and other physiological processes, maternal and infant sleep architecture, including arousal patterns, as well as breastfeeding frequency and prolongation; and (iii) that breastfeeding by mother–infant pairs comprises such vastly different behavioural and physiological characteristics compared with nonbreastfeeding mothers and infants, this dyadic context must be distinguished and given its own epidemiological category and benefits to risk assessment.

Incorporating evolutionary theory and natural selective processes as a powerful explanatory frame, Mobbs et al. (1) revisit a model of what attachment theorist John Bowlby (2) might have called the optimal ‘environment of evolutionary adaptedness’, within which breastfeeding evolved. Ironically, one major weakness in Bowlby’s otherwise polymathic formulation and explanation of ‘attachment’ was his seeming de-emphasis of the role that human breastmilk and breastfeeding delivery actually played in its aetiology. Instead, Bowlby emphasised the more critical consequences of an infant convincing its mother to invest in its survival, with natural selection operating to exaggerate both neonatal facial attributes (big eyes situated on the midline of the face, surrounded by round, nonangular or nonthreatening cheeks) and emotionally attractive, reflexive infantile behavioural responses such as high-pitched cooing infantile gaze, as well as infant affective responses such as terminating cries when soothed, and infant smiling. According to Bowlby, these evolved human infantile traits, which expanded on our related suite of phylogenetic characteristics as primates, coalesced to help motivate mothers (and others) to not only breastfeed and carry but to also protect their infants from predators.

In contrast, Mobbs et al. assign different priorities than did Bowlby to the initial mechanisms promoting attachment and stress the absolute centrality of breastfeeding, and, specifically, they argue for a necessary, sequential unfolding of it. Mobbs et al. argue that human neonates need from the start unhindered and *immediate* sustained maternal contact, including maternal–infant cosleeping, to assure maximum lactogenesis. While many other factors contribute along the way, they posit that the *absence* of unhindered mother–infant contact, from the outset, is a significant hindrance to breastfeeding beyond three months (3).
Indeed, immediate and sustained contact is a prerequisite for what Mobbs et al. call mutual ‘imprinting’, although they use the term in a manner that is distinct from that of ethologist Konrad Lorenz, which reflected canalisation of maternal investment in some mammals and other vertebrates. But, still, they argue for imprinting in a way that facilitates and makes possible (at very least) the neonates’ immediate and instinctive move towards the establishment of oral tactile recognition of the areola around which the infant initiates its first latch, an achievement Mobbs et al. describe as ‘mother in mouth’ or ‘latchment’, an elemental but important step towards attachment.

Accordingly, the neonate’s first latch sets in motion accelerated milk production but also the start of a specific kind of mother–infant sensory and signalling relationship that, over the weeks ahead, likely promotes a particular trajectory of infant neural development, including potentially staving off unnecessary neuronal regression by promoting sensory engagements that enhance neuronal density and interconnectivity (4). We see hints of these possibilities in new path-breaking studies that reveal that breastmilk may assert an organising effect on building ‘types’ of primate brains by significantly enhancing white matter growth compared with formula-fed infants (5). These engagements, which involve practically every sensory modality, are now well documented (6) and provide the scaffolding out of which more complex, emotionally based, cognitively mediated identifications of attachment figures, emerge or, as Mobbs et al. creatively put it in referring specifically to mother–infant attachment, a shift by the infant towards ‘mother in eye’ in addition to ‘mother in mouth’.

Elegant in its simplicity the authors provide a succinct description of oral tactile recognition, which they claim, is achieved through Merkel (tactile) cells having proliferated in the foetus’s first trimester but found later to be more densely distributed widely throughout the infant’s buccal mucosa and exquisitely interconnected with the neonate’s prefrontal sensory cortex. Thus, they add to a list of fundamental characteristics of the neurologically immature human, or ‘exterogestate’ to use Ashley Montagu’s apropos description, whose unfinished brain, central nervous system, immune, respiratory, digestive, thermoregulatory and locomotor systems require continuous contact and proximity to maximise breastfeeding frequency, the foundation piece upon which Mobbs et al.’s model depends. We do, however, urge caution so as not to confuse what amounts to a more labile or flexible bonding process with a simple ‘event’ as if all bets are off after the ‘biological buzzer’ sounds, ending a critical period, after which bonding potential is severely diminished. With that said, we applaud how this model draws on evolutionary theory, with overlaps with developmental systems theory, which is commonly missing, or, at very least underappreciated in Western scientific discourse on identifying more optimal infant care practices and how and why infants respond as they do.

What is at stake is, of course, breastmilk itself and, ideally, lots of it, promoted by an unhindered delivery system (untainted by ‘ decoys’ or pacifiers, or separate sleep spaces). Indeed, breastfeeding mothers find it difficult to keep their babies out of their beds, explicable by the fact that the mothers’ body remains the only environment to which the human neonate–infant is adapted (6). This is a body (both behaviourally and in a physiological sense) that is incredibly responsive to and regulatory of the vulnerable human infant (6) and, assuming a smokeless gestation for the infant, when sober and committed it is ‘value added’ and not an inert lethal weapon over which the mother has no control nor to which her infant offers any feedback. This is a dramatic difference from the portrayals in current ‘Safe to Sleep’ campaigns in the United States, or the studies on which they are based, which analogise mothers to lifeless wooden rolling pins or metal cleavers.

Because bedsharing is known to double and even triple the number of breastfeeds per night (6,7) and the positive relationship between extended breastfeeding and bedsharing is now firmly established, the resultant benefits conferred by the practice are clearly numerous. For example, they potentially include significant protection from a variety of infantile diseases and conditions including SIDS itself, which is breastmilk-dose dependent: that is, the more the breastfeeding, the greater the protection.

Breastfeeding confers significant health benefits to mothers, too. Bartick et al. (8) found that maternal benefits of prolonged breastfeeding are not at all trivial, either. Her data show that suboptimal breastfeeding duration in the U.S. results in nearly 5000 excess cases of breast cancer per year, nearly 14 000 excess heart attacks per year and over 50 000 cases of high blood pressure per year.

While the direct pathways through which breastfeeding protects babies against SIDS are not yet known, recall that with only 24% of its adult brain volume at birth the human neonate is born neurologically the least mature primate mammal of all, the slowest developing and the most dependent on the mother’s body for physiological regulation and support. There is no doubt but that being born so premature was not only a necessity (due to energetic strain on maternal physiology as well as, possibly, birth canal constraints) but also a liability for human infants and their caregivers. Nonetheless, those same liabilities and adaptive challenges, that is keeping our vulnerable and energetically expensive infants alive, produced if not mandated the emergence of a unique suite of human traits such as food sharing, omnivory, empathy, highly invested parenting, contact seeking neonates, cooperation, collaboration, shared care of our young (cooperative breeding) including culture itself, with technological innovation and symbolic and linguistic capacities as its products (6,7,9,10).

More specifically, part of that ‘suite’ of co-evolving traits included maternal and paternal neural and endocrine systems that help to facilitate human parental investment, which, we point out, comes to fruition under a variety of social-ecological conditions and influenced by diverse cultural institutions. However, these evolved systems that enable daytime nurturance are the same pathways through which, even during the most challenging moments, such as
in the middle of the night, exhausted parents (under most circumstances) bring their babies to them (if not already there), to protect, feed, nurture (and, often, to get more sleep) (6,9). Human parental biology and the inclinations that emerge from it persist (and thrive) reflecting the past selective pressures related to our hyper-dependent infants (9,10) despite the fact Western medical organisations are doing everything they can to discourage their night-time expression.

Unfortunately, in Western societies, the question of where infants should sleep appears to be no more easy to answer now than it was a thousand years ago when the Catholic Church through papal ecclesiastical actions advised that infants sleep in their own ‘cradle’ until the age of three years. This ruling was thought necessary due in part to destitute mothers revealing (in the confessional) that they sacrificed by overlying their youngest child to provide minimal support to their other children, as well as because too many infants were dying due to parental drunkenness (11).

Following a series of epidemiological studies showing, especially, a disproportionate number of infant deaths in widely divergent same surface cosleeping environments (e.g. sofa and recliners, adult beds), especially amongst nonbreastfeeding, poor, politically economically marginalised subgroups, population-wide recommendations against any and all bedsharing by the American Academy of Pediatrics in 2005 (12) and again in 2011 (13), were released, as other countries followed suit or formulated similar policies on their own.

In a very recent analysis in Sweden, where rates for SIDS are exceedingly low (two per 10,000 infants), Mollborg et al. (14) found that bedsharing was more commonly associated with SIDS deaths (90% of the sample compared with ~67% associated with SUDI deaths), leading the researchers to confirm Swedish recommendations against any and all bedsharing for infants less than three months of age (15). However, as the authors point out many key social factors including sleep position, pacifier use, and elicit drug or alcohol use, and accurate information on whether breastfeeding at the time of the infants’ death were incomplete or missing, all of which are known to modify SIDS expression (6).

In a related publication, which used the same data set as Mollborg et al. (14), Wennergren et al. (15: p.129) highlighted a controversial meta-analysis by Carpenter et al. (16), which has been used to reinforce recommendations against bedsharing in Sweden and elsewhere. Carpenter et al. conducted individual-level analyses after compiling data from five large population-based, case-control studies of SIDS. Based on these collective data, the authors claim that even in the absence of maternal smoking and/or alcohol consumption, bedsharing–breastfeeding infants remain at higher risk for SIDS (16). It is critical to consider that the study has been highly critiqued on important methodological grounds in part because the five studies represented exceedingly diverse populations, culturally and geographically, with the studies being conducted at different historical time-points. Those complexities are additionally compounded by the difficulties associated with having to impute missing data (to combine the five studies), especially maternal drug and alcohol use. No less than nine significant critiques followed its publication mostly focusing on the validity of the statistics. Questions were raised about the imputation of missing data, particularly that the imputation procedures were based on false assumptions, as well as the analytical approaches (which, themselves, relied on the imputed data), such as known risk factors being left out of the models, precluding the identification of plausible mediator or confounding effects. In the light of these serious methodological issues, several critiques point out that, at minimum, the research design does not justify statements about causality (as opposed to possible confounding) (16).

Legitimate questions can also be raised about the validity of the responses given by case-control families to questions regarding where their infant slept on the reference night, which is a limitation that applies to Carpenter’s analysis as well as other related epidemiological studies. For epidemiological studies, the accuracy of such responses are (of course) of utmost importance in determining the validity of the denominator, the numbers of babies that bedshared and lived, which is a critical foundation piece for producing valid odd ratios. In a previous study in Great Britain, Ball et al. (17) found that when they asked parents about where their baby slept the night before, many families tended to give an answer reflecting where the infant was supposed to sleep, which was in her/his crib, rather than where the baby actually slept (at least for part of the night), which was in the parents’ bed. As her research shows, if the infant began the night in a crib, but later (after the first breastfeed, for example) the infant was relocated to the parental bed, the parents still perceived (and reported) that the baby slept in her/his crib, because the baby was ‘supposed’ to sleep there. Indeed, only after further probing and rephrasing of the original question ‘where did your baby sleep’ did Ball’s research team determine that 40% of the infants whose parents initially reported their babies slept in a crib actually had slept in the parental bed for some or most of the night. We wonder just how many epidemiological findings (related to bedsharing) would be different if, as is a routine practice for psychological surveys, before administering certain ‘hot-button’ questions to the case-control families, validation efforts were undertaken to determine how parents are perceiving, interpreting and responding to questions about sleep location. Moreover, we argue that the fact that for over a decade, parents have been subjected to widely disseminated warnings against bedsharing by authorities including child protective services also makes it more likely that parents are less comfortable acknowledging their bedsharing practices.

For all of these reasons (among others), this contemporary and 15-year-old controversy shows no sign of abating. We suggest that this is unsurprising due to reality that the majority of breastfeeding mothers end up bedsharing (at least intermittently), often unexpectedly (18) and that their
babies seemingly remain at low risk for SIDS. Indeed, sometime during the first three months post-partum, many, if not most, breastfeeding mothers adopt bedsharing (though not necessarily routinely, all night and every night), as it proves to be a successful means initially to solve latching problems and to manage their milk supply (stimulating lactogenesis); but bedsharing also proves to be an effective way to reduce night-time infant crying, to settle babies and for both mother and infants to get more sleep, in addition to finding more emotional satisfaction by way of the stronger attachment such contact and proximity facilitates (6). Thus, research has consistently shown that across Western cultures, breastfeeding and bedsharing are mutually reinforcing. Of course, this has long been the case in many other societies in which neither practice (bedsharing, breastfeeding) or their interrelationship have ever ‘fallen out of favour’ (6). The facilitatory effects of bedsharing apply to both more breastfeeding per night as well as the number of months mothers are able to sustain their breastfeeding (6), thus, supporting Mobbs et al. major contentions.

Given these benefits and the apparent opposition by so many parents as well as by many SIDS researchers, prestigious international organisations, developmental scientists and lactation support communities (6,19,20), the chances of eradicating any and all bedsharing seem highly unlikely, especially given its intimate, biological and functional interconnectivity to breastfeeding, which stands to be negatively impacted. At very least, the possibility of high compliance to an unqualified recommendation against any and all bedsharing (same surface cosleeping). In addition to possibly undermining breastfeeding, such recommendations prevent parents from gaining access to information on minimising bedsharing risks, should parents choose to do so, as millions do (6). Reducing same surface cosleeping (a more generic term than problematic usage of ‘bedsharing’) to a singular risk factor is itself problematic because it is clear that outcomes depend on exactly how the same surface cosleeping is being practiced and by whom (6,20). Given all the ongoing controversies, and lack of professional consensus, certainly new findings from one of the most complete and internally coherent epidemiological studies of SIDS yet conducted strongly suggest that harm reduction approaches have a much higher chance of compliance than do simplistic, negative saturation approaches. Based on a combined individual analysis of two population-based studies from Great Britain of SIDS infants and controls comparable for age and time of last sleep (400 SIDS and 1386 controls from five English health regions), Blair et al. (20) found that in the absence of hazardous factors bedsharing is not a significant risk and after three months of age may well be protective.

Hence, the problem for medical institutions promoting bedsharing eradication is, perhaps, at the root of it, the four to six million years of years of breastfeeding evolution [a term we propose, defined as breastfeeding mothers sharing the same or an adjacent sleep surface, (i.e. cosleeping), with their infants in the absence of all hazardous factors]. Such a successful feeding and sleeping arrangement (humankind’s oldest) will not so easily be subject to cultural nullification in the same way that, say, infant sleep position was, that is stomach sleeping, a behaviour that was never part of a larger evolved biobehavioural, protective complex, such as breastsleeping, for which parents are emotionally and physiologically primed, as well as psychologically invested.

As is true for the thinking that motivated Mobbs et al. to propose their model, it is likewise against a larger evolutionary legacy that we can appreciate the profound need that human infants have for parental contact and why mothers and fathers are thus biologically inclined to provide it (6,9,10).

In sum, we find Mobbs et al.’s. ethological delineation of the simple way in which human neonatal biology finds expression when placed in its appropriate environment quite timely. We say this in the light of the ‘Safe to Sleep’ campaigns, as we mentioned, being imported around the world from the United States, which have, as their centre-piece, an unqualified recommendation against any and all bedsharing (same surface cosleeping).

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We end this piece by calling attention yet again to the importance of evolutionary (biological) processes that serve as a powerful beginning point, as Mobbs et al. and others have so eloquently demonstrated. Life-history theory predicts that because for mothers (and all of us) time and energy are finite how much we invest in any one child is constantly being renegotiated in relationship to all other competing daily demands and responsibilities (9,10). It is within this context that for breastfeeding mothers, the decision to bedshare proves often to be an unexpected ‘no
brainer' explaining why, perhaps, a quiet but seismic shift towards adopting bedsharing in Western cultures, despite medical recommendations against it, is occurring as breast-feeding re-establishes itself in many Western countries as the cultural norm.

In sum, it is because the two are so physiologically and behaviourally entwined and functionally interdependent, and because it may help to resolve what has been a stagnant and nonproductive debate that we propose the term *breastsleeping* as a way to acknowledge the following: 1) the role that sustained maternal contact plays in helping to establish breastfeeding; 2) that normal, healthy, human (species wide) infant sleep is not accurately measurable outside of the breastsleeping context as maternal infant sensory exchanges involved in the elicitation and delivery of breastfeeding, and the ingestion of breastmilk significantly changes infant metabolism, maternal and infant sleep architecture including arousal patterns, as well as breastfeeding frequency reflecting a highly integrated adaptive architecture including the breastfeeding mother–infant dyad exhibits such vastly different behavioural and physiological characteristics compared with the bottle/formula feeding–bedsharing dyad it must be distinguished and given its own epidemiological category and benefits to risk assessment. It is to this fervent purpose that we offer this perspective.

**DISCLOSURE**

No competing financial interests exist.

**References**

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