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Elizabeth H. Peters, Ph.D.

ABSTRACT: Colic is a disorder of early infancy marked by excessive amounts of loud, persistent crying. Lesser amounts of crying are considered normal in infants. Neither the crying of colicky infants nor the baseline crying of normal human infants have any homologue in the vocal behavior of other mammalian infants. This human-specific cry continuum may reflect a human-specific discomfort continuum which is function of the general immaturity of human neonates. Such immaturity may be the result of selection for altricial birth forced by cephalo-pelvic incompatibility during birth.

The medical literature on infant colic includes enough diversity, discrepancy and obscurity to resist comprehensive summary in a few sentences. However, since the more obvious and recurrent features of this discourse may be the ones that yield the most insight under scrutiny, a brief outline of these may be useful:

- 1. Colic in human infants is a *disorder* (a deviation from some conceptualization of "normal"). Its most salient symptom is also its only consistent symptom—loud, prolonged, inconsolable crying (Barr et al. 1992). This crying is a source of stress for caretakers. It is perceived by caretakers to signal discomfort experienced by the infant (Forsyth, 1989; Geertsma & Hyams, 1989)
- 2. Colic exists in relatively *high frequency* in the countries which attempt to measure this statistic. Published estimates suggest that 10-40% of otherwise normal infants have colic (Hide & Guyer, 1982; Illingworth, 1954; Lothe, 1989).
- 3. Colic is developmentally self-limiting. It generally disappears by the time an infant reaches 3-4 months of age (Illingworth, 1954; Taylor, 1957; Weissbluth & Weissbluth, 1991).

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- 4. Although difficult to ignore, colic is also difficult to distinctively diagnose. Prolonged, loud crying is also found in non-colicky infants. Barr et al. (1992) conclude that colicky infants differ principally in the duration of cry bouts although Lester et al. (1992) propose there is a difference in cry quality. A widely accepted definition proposed by Wessel et al. (1954) distinguishes colicky infants from normal infants on the basis of quantitative (but not qualitative) differences in cry behavior.
- 5. An array of suspected "causes" have been proposed and investigated. Lothe (1989) sorts these into the following categories: gastrointestinal; allergenic; hormonal; psychosocial; cerebral immaturity and "miscellaneous". Despite 50 years of discussion in the medical literature, both the *etiology* and the *cure* of infant colic remain *elusive* (Hewson et al., 1987; Hyams et al., 1989; Lothe, 1989; Stahlberg, 1984).

In the on-going dialogue about infant colic, the evolutionary perspective has not been prominent. The current repertoire of proposed causes all share the property of being relatively "proximate" in nature. This paper will explore the value of developing an explanation for infant colic at the level of "ultimate cause" (i.e. an explanation which references natural selection and adaptation over an evolutionary time-scale).

### PROXIMATE CAUSE AND ULTIMATE CAUSE

In a chapter entitled "The dual nature of causation in biology," Goldsmith (1991) suggests that Darwin's most fundamental accomplishment was "to enlarge for all time the concept of scientific explanation." By providing a naturalistic explanation for the adaptative design of organisms, Darwin capitalized on the conceptual potential of considering the expansive time frame we now know as the geological time-scale.

Prior to Darwin, causal relations in biology were established within a time frame that was more in keeping with ordinary human experience. To say that colic is "caused" by an "allergic reaction to cow's milk" or by "parental mismanagement" is to deal with a relatively short-term relationship between one or more precipitating events and their consequences. To such explanations of "proximate cause" Darwin added the complementary kind of explanation that has come to be

known as "ultimate cause." To suggest that organisms who possess some heritable trait survive and reproduce in greater numbers than their counterparts who lack this trait is to reference an adaptive process which may only become apparent over a long time-scale. This paper will introduce a model of directional evolutionary change in which ancestors whose infants born altricial enough to have small heads were at a selective advantage over those whose heads were too large at birth or too small as adults.

It is important to note that explanations which refer to proximate cause-effect relations can and should be embedded in explanations which highlight an evolutionary trajectory. In modern biology, it is not only possible but it is also desireable (in the sense of optimizing clarity) to explain the same phenomenon at multiple levels simultaneously.

Evolutionary explanations are fundamental to modern biology and modern anthropology. In contrast, adaptive ("ultimate cause") explanations are still relatively rare in medical circles. The relevance and the explanatory power of this type of explanation was highlighted when a recent meeting of the American Association for the Advancement of Science featured a special symposium entitled "Evolutionary Medicine" (McKenna, 1993).

#### IS COLIC FOUND ONLY IN HUMAN INFANTS?

There is good reason to suspect that infant colic is a *human-specific* (as well as an age-specific) disorder.

If a pathology can be studied in laboratory animals, scientific investigation can proceed much faster. However, no laboratory animal model of infant colic has ever been found or experimentally produced. Since humans share a recent ancestry (and a good deal of anatomy and physiology) with anthropoid primates, it is logical to expect that these animals would be more likely to show evidence of a similar infant disorder. The primate professional literature (as indexed in a 30-year computerized database by the University of Washington's Primate Information Service) lacks any reference to colic among alloprimate infants.

In an effort to see whether similar expressive behavior (with some probability of similar cause) is present, the literature on primate vocal ontogeny was scrutinized for evidence of something akin to the crying of colicky infants. Such a review reveals that not only is the inconsolable crying of colicky human infants absent but so too is the baseline

"normal" cry behavior of human neonates—the loud, repeated, caretaker-mobilizing vocalizations which are so stereotypically-associated

with human infancy.

Non-human primate neonates are extremely quiet. My own study of vocal behavior in free-ranging rhesus monkeys (Peters, 1983) indicates that neonatal and very young infants rarely emit any vocalizations. When they do, the vocalizations are very short and non-repetitive and the probable exogenous stimulus is easy to identify. Maturing, independently-locomoting infants are much more likely to emit vocalizations than neonates—at first in the form of noisy "aid-enlistment" screams and later (during weaning) as warbled tonal vocalizations. While our general knowledge of the vocal behavior of primate infants is very incomplete, it is unlikely that more research will change the overall impression that neonatal alloprimates vocalize substantially less than neonatal humans.

## IS INFANT COLIC A HUMAN UNIVERSAL?

Lothe's (1989) review of the medical literature (both modern and historical) suggests that reports on infant colic tend to be from countries which share Western, mainstream culture. Native American, Australian aborigine and !Kung San infants do not show the typical symptoms of colic (Brazelton et al., 1969; Konner 1972; Rowley (quoted in Lothe, 1989); Thomas, 1981). On the other hand, Weissbluth & Weissbluth (1991) quote Chinese, Korean, Vietnamese and Japanese terms (e.g., "100 days crying") which suggest the presence of this syndrome in these cultures.

There is no code for colic in the *Outline of World Cultures* (1976) which indexes the ethnographies included in the Human Relations Area Files. Although there is some evidence that anthropologists have neglected the study of human infancy (Peters, 1993), the lack of reference to colic in the ethnographic literature may indicate a relative absence (or at least a lower frequency) of this disorder in non-Western

cultures.

Barr (1990, 1992) used Brazelton's (1962) data on the circadian and developmental patterning of normal infant cry behavior to suggest that there was a continuity in the patterning of normal crying and colicky crying. Both kinds of crying cluster during the evening hours and both exhibit a peak in daily duration during the first few months of life.

When Barr (1990) reviewed Konner's (1976) data on infant development among the !Kung San, he noted that "cry/fret" vocalizations peaked in frequency at about three months of age. Among the foraging !Kung, infant caretaking includes all the behaviors which are usually considered efficacious in eliminating or reducing crying: caretaker contact; constant carrying; continuous breast access; upright posture; rapid response to infant vocalizations. Given the robustness of the cry/fret peak in the face of extreme caretaker solicitude, Barr concluded that the developmental regularity of early infant crying "may represent a species-specific behavioral universal."

If it is reasonable to consider "cry/fret" to be a signal of infant discomfort, there may be a further lesson from this data. If discomfort extreme enough to be expressed in colicky crying is not present in !Kung infants, then some lesser degree of distress seems to follow a similar developmental course. While colic itself may not be universal, a sub-clinical homologue may be. When infant-caretaking patterns depart dramatically from the evolutionary norm (i.e. from continuous application of the discomfort-reduction repertoire which foragers like the !Kung provide), then infant discomfort may increase and colicky crying may express this.

# ARE HUMAN NEONATES MORE IMMATURE AT BIRTH THAN THE NEONATES OF OTHER PRIMATES?

Portmann (1941, 1945 as quoted in Gould, 1977) seems to have been the first to publish the observation that humans have the small litter-size and the slow post-natal growth rate of precocial mammals, yet they deviate from the precocial norm in being extremely helpless at birth. Since human post-natal growth rates resemble the fetal growth rates of other primates, Portmann concluded that "Human growth follows the mammalian norm, but birth occurs much earlier than this norm would imply" (italics mine).

"Exterogestation" is a term coined by Montagu (1961) to refer to the period following birth when human infants function in many ways like a fetus and seem to be completing the same interval of early development that other primates complete *in utero*. Among several markers of fetal status, Montagu noted the extreme motor immaturity of human neonates. He suggested that it takes many months of post-natal development for human infants to achieve the same degree of locomotor independence that other long-gestating mammals have at (or soon after) birth.

Using data from Schultz (1941) and Blackfan (1933), Passingham (1975) compared chimpanzee and human brain weight at four stages of development. He found that chimpanzees have 45.7% of their adult brain weight at birth, while neonatal humans have only 25.5% of the brain weight they will attain as adults. Maturing human infants reach the proportion found in neonatal chimpanzees "around 6 months after birth." From life history data provided by Schultz (1956) and Napier (1967) Passingham observed that the gestation period of apes (gibbon, orangutan, gorilla and chimpanzee) averages 6.48% of the period of bodily immaturity and 1.97% of the total life span. The comparable values for humans (3.64% and 1.04%), imply that gestation is only about half as long in humans as it would be if we followed the hominoid norm. Passingham concludes "If the gestation period were to be relatively as long in man as in the apes, man would be born between 7 and 8 months later than he is."

Holt et al. (1975) plotted the prenatal brain weight/body weight curve in humans and in three species of alloprimates. They found that the high prenatal slope of alloprimates is followed by a flat post-natal slope shortly before or just after birth. In humans, however, the high fetal slope extends well into postnatal ontogeny, resulting in a markedly higher encephalization. Collecting data for a broad range of primate taxa, Harvey and Clutton-Brock (1985) found that alloprimate brain growth resolved into two distinct patterns—either relatively high prenatal or relatively high postnatal brain development. The subfamilies that give birth to relatively large-brained neonates have *low* postnatal brain development (they reduce the high rate of prenatal brain growth soon after birth). Human infants are a "striking exception," maintaining the *fetal* rate of brain growth for about a year after birth.

Trevathan (1987) reviews several other indicators of relative immaturity. These include delayed ossification of the bones of the phalanges, lack of cranial plate ossification and a relative deficit of liver and gastric enzymes in human neonates.

## DID EVOLVING HOMINID ENCEPHALIZATION SELECT FOR EARLIER BIRTH?

Montagu (1961) offers the observation that "...man is born as immaturely as he is because—owing to the great increase in the size of his brain and consequently of his head—if he weren't born when he is, he wouldn't be born at all." Early birth is a consequence of the

evolution of larger brain size at the same time that the pelvic structure necessary for bipedal locomotion imposed an upper limit on the fetal head size which could be accommodated during parturition. By completing after birth much of the brain growth which other primates complete *in utero*, evolving hominids escaped the constraints on adult brain-size which a pelvis shaped for bipedalism would otherwise impose.

Earlier, Portmann (1945 quoted in Gould, 1977) rejected a parturition-related explanation for "precocious birth", in favor of one which emphasized the positive value of the greater stimulation provided by the extrauterine environment. Although earlier exposure to intense and varied stimuli may indeed have consequences for human cognitive development, recent reviewers see this as a fortuitous by-product rather than a primary or secondary selective pressure for earlier birth (Gould, 1977; Trevathan, 1987).

Trevathan (1987) systematically reviews the relationship between the peculiarities of human growth rates, the pelvic constraints imposed by habitual bipedalism and the competing selective pressures shaping gestational length in our species. Her detailed comparison of the birth process in modern humans and closely-related alloprimates makes one conclusion inescapable: birth is much more dangerous for humans than it is for other large-bodied hominoids. Trevathan's data strongly support the hypothesis that cephalo-pelvic size-incompatibility provided a selective pressure for ontogenetically-earlier birth in our recent ancestors.

# IS COLIC A TAG-ALONG CONSEQUENCE OF EARLIER BIRTH?

A number of hypotheses about the etiology of infant colic have implicated immature functioning of the gastrointestinal tract or the central nervous system (Brennemann, 1946; Boon, 1982; Eppinger & Hess, 1915; Spock, 1944; Paradise, 1966). While such hypotheses are consistent with the observation that infant colic is developmentally self-limiting, they do not explain why the neonates of other species (including our closest primate relatives) never show evidence of this disorder.

The transition from fetus to neonate requires a switch from the placental transfer of oxygen, nutrients and waste products to mobilization of the infant's lungs and gastrointestinal tract for these essential life functions. If human neonates switch from uterogestation (and placental nourishment) to exterogestation (and gastrointestinal absorption) at an *earlier* point in general fetal maturation than other primates, it might be instructive to compare the relative maturation of this system in humans and closely-related alloprimates both before and after birth.

If we examine the digestive tract of *fetal* alloprimates when they are at a stage of overall maturity comparable to that of human neonates (perhaps about halfway through gestation?), would this system be able to handle gastrointestinal absorption? Would the alloprimate prenatal system be more vulnerable to malfunctioning than the human neonatal system (suggesting that the human system has undergone selection for *accelerated* maturation as a correlate of earlier birth)? Do contemporary human neonates deviate from the primate developmental norm in the maturation of systems necessary to support life outside the womb (i.e. lungs and gastrointestinal tract) even as they follow the primate fetal norm in other systems (e.g. in skeleto-muscular development and in the rate of brain growth)?

If further investigation supports a model of early birth and accelerated gastrointestinal maturation, it is not too soon to think about the geological timing of its emergence. Paleoanthropologists have already begun to consider the relative ease of birth in Australopithecus and earlier species of *Homo* (Tague and Lovejoy, 1985; Leutenegger 1972, 1973, 1974; Trevathan, 1987). If it is reasonable to conclude that selective pressure for earlier birth emerged relatively recently in the hominid lineage, it would also be reasonable to suspect that adaptation for the early functioning of essential life support systems (like the lungs and the gastrointestinal tract) may be less-than-optimal. That is, selection may not have occurred over a geological interval long enough to optimize present functioning in all (or nearly all) contemporary infants. In a neonatal population with a normal distribution of variation, these systems may be only marginally-functional in individuals at the lower end of the distribution. If infant care-taking practices deviate from the norm under which this adaptive complex evolved, then infants with marginally functioning digestion may be pushed into colic (and perhaps those with marginally-functioning respiration into sleep apnea and SIDS).

In 1961 Ashley Montagu argued eloquently:

<sup>&</sup>quot;... if this interpretation of the gestation period is sound, then it would follow that we are not at present meeting the needs of infants in any-

thing approaching an adequate manner . . . Whereas the marsupial (exterogestate) infant enjoys the protection of its mother's pouch during its period of immaturity, the human infant is afforded no such natural advantage. That is all the more reason why the parental generation in such a species must clearly understand what the immaturity of its infants really means . . . The biological unity, the symbiotic relationship, maintained by mother and conceptus throughout pregnancy does not cease at birth but becomes—indeed, is naturally designed to become—even more intensive and interoperative after birth. . . ." (p. 157).

The time may come when infant colic will be looked upon as one more example of the "diseases of civilization," a syndrome which can be reduced (or even totally avoided) by caretaking practices which are congruent with the special needs of human neonates.

#### SUMMARY

Medical attention to the problem of colic in human infants has been limited to a search for proximate cause. This paper has explored the value of adding an evolutionary perspective and proposed an ultimate cause. Vulnerability to colic may be prevalent in human infants, but absent in other mammals, because modern humans switch from uterogestation (and placental nourishment) to exterogestation (and gastrointestinal absorption) at an earlier point in fetal maturation. Human neonates may be forced to absorb nutrients and excrete wastes with a gastrointestinal tract which is less-than-optimally adapted for its required early functioning. Infant care-taking practices in modern Western cultures may overstress the marginally functioning biology of some infants and produce the more serious discomfort which is signaled by colicky crying.

#### REFERENCES

Barr, R.G., (1990). Carrying, colic, and culture: Crying in context. Paper presented at the Seventh International Conference on Infant Studies, Montréal, Québec, April, 1990.
Barr, R.G., Konner, M., Bakeman, R. and Adamson, L. (1991). Crying in !Kung San infants: A test of the cultural specificity hypothesis. Developmental medicine and child neurology, 33, 601-610.

Barr, R.G., Rotmans, A., Yaremko, J., Leduc, D., Irancoeur, T.E. (1992). The crying of infants with colic: A controlled empirical description. *Pediatrics*, 14-21.

Brazelton, T.B. (1962). Crying in infancy. Infancy, 29, 579-588.

Brazelton, T.B., Robey, J.S. Collier, GA (1969). Infant development in the Zinacanteco Indians of southern Mexico. *Pediatrics*. 44: 274-290.

Geertsma, M., Hyams, J. (1989). Colic-A pain syndrome of infancy? *Pediatric Clinics of North America*, 36(4): 905-919.

Goldsmith, T.H. (1991). The biological roots of human nature: Forging links between evolution and behavior. New York: Oxford University Press.

Gould, S.J. (1977). Ontogeny and phylogeny. Cambridge, MA: Harvard University Press. Harvey, P., Clutton-Brock T. (1985). Life history variations in primates. *Evolution*, 39: 559-581.

Hewson, P., Oberlaid, F., Menahem, S., (1987). Infant colic, distress, and crying. *Clinical Pediatrics*, 26(2): 69-75.

Hide, D.W., Guyer, B.M. (1982). Prevalence of infant colic. Arch. Dis. Child 57: 559-60.
 Holt, A.B., Check, P.B., Mellits, E.D. & Hill, D.E. (1975). Brain size and the relation of the primate to the nonprimate. In D.B. Check (Ed.): Fetal and postnatal cellular growth: Hormones and nutrition. New York: John Wiley.

Hyams, J., Geertsma, M., Etienne, N., Treem, W. (1989). Colonic hydrogen production in infants with colic. *The Journal of Pediatrics*, 115(4): 592–594.

Illingworth, R.S. (1954). Three-Month Colic: Arch. Dis. Child, 29: 165-174.

Kennell, J.H. (1980). Are we midst of a revolution? Am. J. Dis. Child, 134: 303.

Konner. (1976). Maternal care, infant development and behavior among the !Kung. In R. Lee and I. DeVore (Eds.) Kalhari Hunters-Gatherers. Cambridge, MA. Harvard University Press.

Leutennegger, W. (1972). Newborn and pelvic dimensions in Australopitheans Nature, 240: 548-569.

Lothe, L. (1989). Studies on infantile colic with special reference to cow's milk whey protein, macromolecular absorption and regulatory peptides. Department of Pediatrics, University of Lund, Malmö, Sweden.

McKenna, J.J. (1993). Evolutionary medicine. Symposium presented at the Annual Meeting of the American Association for the Advancement of Science.

Montagu, A. (1961). Neonatal and infant immaturity in man. Journal of the American Medical Association, 178: 56-57.

Peters, E.H. (1983). Vocal communication in an introduced colony of feral rhesus monkeys (*Macaca mulatta*). Ph.D. Dissertation, University of Florida.

Peters, E.H. (1993). Human infancy as a vehicle for teaching anthropology. Strategies for teaching the central themes of anthropology. New Delhi: Reliance Press.

Portmann, A. (1941). Die Tragzeiten der Primaten und die Dauer der Schwangerschaft beim Menschen: Ein Problem der vergleichen Biologie. Rev. Suisse Zool, 48: 511-518.

Portmann, A. (1945). Die Ontogenese des Menschen als Problem der Evolutionsforschung. Verh, Schweiz, Naturf. Ges., 125: 44-53.

Schmitt, B.D. (1985). Colic: Excessive crying in newborns. *Clinical Perinatology*, 12: 441-451.

Schultz, A.H. (1956). Postembryonic age changes. Primatologia, 1: 887-964.

Stahlberg, M. (1984). Infantile colic: Occurrences and risk factors. *Pediatrics*, 143: 108–111.

Taylor, W.C. (1957). A study of infantile colic. Canadian Medical Association Journal, 76: 458-461.

Thomas, D.B. (1981). Aetiological associations in infantile colic: An hypothesis. Aust. Paediatr, 17: 292–295.

Trevathan, W. (1987). Human Birth: An Evolutionary Perspective. New York: Aldine De Gruyter.

Wessel, M.A., Cobb, J.C., Jackson, E.B., Harris, G.S., Detwiler, A.C. (1954). Paroxysmal fussing in infancy, sometimes called 'colic', *Pediatrics*, 14: 421-434.