

Rapid *E. Coli* Invasion May Cause Sudden Infant Death

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BETHESDA, Md.—Sudden Unexpected Infant Death Syndrome (SUID) in human infants may have the same pathogenesis as infectious diarrhea (scours) and sudden death of newborn calves.

A strong plea has been made for physicians and pediatric researchers to investigate the probability that acute toxemia resulting from rapid and massive invasion of *Escherichia coli* into the highly absorptive portion of the small intestine may be the death mechanism in the human syndrome, just as it is in the disease syndrome in calves and the young of other mammalian species.

Dr. Robert C. Reisinger, research veterinarian with the National Cancer Institute, told an NIH seminar here that there is a definite model in calf research for the Sudden Unexpected Infant Death Syndrome.

"Instead of studying only viruses and slides of fixed tissues physicians and researchers need to do quantitative studies of the bacteria in the anterior intestinal tracts of these children," Dr. Reisinger urges. He points out that slide pathology will not demonstrate the condition—there are no pathognomonic lesions.

By quantitatively culturing the human infants microbial flora at different appropriate levels of the intestinal tract, it would be possible to establish the role of bacteria in the SUID syndrome, he says.

(Dr. Reisinger commented that the recently published proceedings of a Seattle conference on the causes of sudden deaths in infants [Public Health Service Publication No. 1412] contain only three references to bacterial studies—these were not quantitative, and did not include the upper intestinal tract.)

Concerning the relevance of the calf disease syndrome to Sudden Unexpected Infants Death Syndrome, Dr. Reisinger notes that the peak incidence in humans is comparable to that seen in calves when the difference in maturity of the two species is considered. In calves, the peak incidence occurs while the calf is basically a monogastric animal, on a mainly liquid diet. The human infant is also of course on a liquid diet, and it is not difficult to visualize that under this condition it would be relatively more simple for *E. coli* invasion to occur than in the older child or animal when intestinal contents are more firm, the normal intestinal flora more firmly established, and the entire organism more adapted to various stressors.

In his own calf research, he reports that all of more than 60 lethal cases of scours were associated with overgrowth of *E. coli*; several cases of sudden death, with no scours or diarrhea, showed the same overgrowth of these bacteria. Other young animals, including

rabbits and pigs, show the same susceptibility and response to multiplication and migration of *E. coli* into the upper digestive tract.

Normally, Dr. Reisinger explains, the gram-negative *E. coli* are found in the lower part of the ileum, and in the cecum, large intestine and rectum. The duodenum, jejunum and upper ileum, where most digestion and absorption of nutrients occur, is normally acid, and inhabited by acid-producing lactobacilli and enterococci; *E. coli* cannot long exist in this acid medium.

When the balance between lower and upper bowel is upset, *E. coli* in great numbers invade the upper more absorptive portion, and diarrhea and/or death occurs. It is well-known that *E. coli* do not normally occur in the upper digestive tract of any mammalian species. When these organisms are present in large numbers in this portion of the intestinal tract, absorption of toxins results in varying degrees of illness and, in the young, often in death. Counts of 10^7 or 10^8 coliform bacteria per cubic centimeter in the more absorptive portions of the small intestine have been found associated with this disease syndrome in newborn calves, and in calves up to 30 days of age.

Other Factors

Adverse contributing factors which aid in precipitating the disease in animals include: 1) cold and wet, rapid climatic temperature changes; 2) overcrowding, which causes buildup of particularly pathogenic organisms; 3) subnormal nutritional status, such as avitaminosis A; 4) virus infections; 5) overfeeding, which dilutes out the acid in the gut and also predisposes to vomiting, ballooning of the intestine, and perhaps reverse peristalsis; 6) prolonged interval between birth and ingestion of the first colostrum.

Dr. Reisinger observes that early feeding of colostrum—within 15 minutes of birth—is the most critical factor in preventing the disease in newborn animals. Colostrum must be present in the anterior gut for its *E. coli* antibody to be effective.

Lack of antibody to *E. coli* does not appear to play a clearly defined role in

