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Potential Mechanism of Neonatal Bloodstream Infections from a Mouse Model

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Potential Mechanism of Neonatal Bloodstream Infections from a Mouse Model

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Bloodstream bacterial infections that can result in septic shock are a common cause of death in NICU patients. Improvements to hygienic techniques have failed to lower the rate of sepsis in patients. Recently, we have shown several species of bacteria colonizing the GI tract are able to gain access to the body via goblet cells, an epithelial cell that lines the intestinal tract and secretes mucus. We investigated if bacteria could cross goblet cells in infant mice, and found both commensal bacteria and bacteria isolated from pediatric sepsis patients could cross goblet cells in mice between day 10 and 21 of life. Additionally, we found both bacteria could also colonize organs distant from the gut. Thus, bloodstream infections in infant mice could be initiated from bacteria colonizing the intestine. We then asked what prevented bacteria from crossing the intestine prior to 10 days of life. To answer this, we analyzed levels of EGF, a protective protein secreted in the breast milk of nursing mothers, and counted the number of bacterial colonies present in several mouse organs from day 5 to day 21 of life. We found an inverse correlation between the amount of EGF present in breast milk and number of colonies of bacteria in the infant mice. Therefore, nursing infants may be able to prevent other strains of bacteria from translocating from the intestine. In the NICU, patients often lack access to breast milk, suggesting this may allow them to be susceptible to sepsis. In the future, we hope to utilize a mouse-model to control levels of EGF in the mice while infecting them with sepsis causing bacteria isolated from human patients to analyze the ability of EGF to prevent bacterial translocation from the gut.