

New Therapy May Be Effective Against Bacterial Infections and Sepsis

Sepsis, the systemic inflammatory response to infection, affects more than 700,000 people annually in the U.S. alone and is the leading cause of death in the intensive care unit. Ninety percent of reported cases are attributed to bacterial infections and mortality rates remain at 25% despite high quality supportive care and antibiotic treatment. Innate immune responses are vital to containing bacterial pathogens and recent studies link sepsis with impaired immunity. Antibiotic resistance and an increase in the mortality rate of sepsis patients due to the use of inappropriate antibiotics, as well as the role of the innate immune response in pathogen control highlight the need for new antimicrobial therapies.

Eosinophils are white blood cells whose normal function is to protect the body against parasitic infections. They are also commonly associated with asthma and allergies. Numerous prior studies have noted the presence of Toll-like receptors (TLRs) on the surfaces of eosinophils indicating that they may play a part in recognizing and killing viruses and bacteria. In the study researchers found that isolated mouse eosinophils possessed antibacterial properties against Pseudomonas aeruginosa in vitro. In vivo, transgenic mice demonstrating high levels of eosinophils, showed improved clearance of P. aeruginosa, whereas bacterial clearance was impaired in mice with a congenital eosinophil deficiency suggesting an eosinophil specific effect.

"We provide evidence that mouse eosinophils and eosinphil granules play a beneficial but poorly defined role in innate immune responses to bacterial infections," say the researchers. "Moreover, the data suggest that the administration of eosinophil-derived products may represent a viable adjuvant therapy for septic or bacteremic patients in the intensive care unit."

Adapted from materials provided by American Society for Microbiology, via EurekAlert!, a service of AAAS.

www.sciencedaily.com

Published on: Mon, 30 Nov 2009