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Autophagy mediated clearance of *Burkholderia cenocepacia* in cystic fibrosis

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Burkholderia cenocepacia is the most feared bacteria for cystic fibrosis (CF) patients to acquire due to multi-drug resistance, intracellular survival in macrophages, high septicemia, and increased mortality. Autophagy is a physiologic process that plays a role in eliminating intracellular pathogens and has been shown to be deficient in CF host-pathogen interactions. We have shown the ability of autophagy stimulation with rapamycin to resolve *B. cenocepacia* infection in a murine CF model, but concerns remain over rapamycin use due to drug toxicity. Therefore, human CF peripheral macrophage responses were studied in response to autophagy stimulation with IFN-γ, a readily available inhalational and systemic therapeutic. Basal serum levels of IFN-γ are similar between CF and non-CF patients, however after *B. cenocepacia* infection, there is deficient IFN-γ production in CF. We will demonstrate the ability of IFN-γ to improve CF macrophage clearance of *B. cenocepacia* through sequential increased autophagosome formation, bacterial targeting to autophagosomes through cargo adaptor molecules, and subsequent increased trafficking to autophagolysosomes for degradation. Through this process, we will demonstrate positive effects on downstream inflammatory signaling in cell death in CF patients. Together, our results demonstrate that IFN-γ promotes autophagy mediated clearance of *B. cenocepacia* in human CF macrophages, and may be a novel therapeutic for severe infections with *B. cenocepacia* and other pathogens in CF.

Biography

Benjamin T. Koppis an assistant Professor in the Department of Pediatrics at The Ohio State University and an investigator in the Center for Microbial Pathogenesis at The Research Institute at Nationwide Children's Hospital. He received his M.D. from The Ohio State University, fellowship training in Pediatric Pulmonology from Nationwide Children's Hospital, and trained in Dr. Amal Amer's laboratory. His research is focused on mechanisms of pathogen control and inflammation in cystic fibrosis.

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