

Regulation of IL-25 levels provides protection against *Clostridium difficile* infection



Clostridium difficile infection (CDI) continues to be the most common hospital-acquired disease in the United States, with over 450 000 infections annually. In the recent 12 July 2016 edition of *Cell*, researchers from the University of Virginia have discovered a possible immune-based therapy for this disease. Although IL-25 is typically studied in the context of allergies, it has shown to have pathogenic and protective responses to the gut microbiota environment. However, when patients are infected with CDI, their IL-25 levels are greatly suppressed.

The purpose of this study is to examine the effects of IL-25 restoration on a *C. difficile* infection. When IL-25 levels were replenished, there was a substantial decrease in the mortality and morbidity of mouse models by protecting host tissue and preserving the gut epithelium. It is has these protective characteristics due to its ability to down regulate deleterious cytokine IL-23 during CDI. Most interestingly, IL-25 protective measures were highly dependent on the presence of esinophils. When there was an eosinophil levels were depleted, IL-25 decreased drastically and replenishing of esinophils led to an increase of IL-25. These results indicate that IL-25-mediated eosinophilia reduces mortality from CDI as

well as maintaining a healthy intestinal epithelial during infection. The authors suggest that, “this work may provide targets for future development of microbial or immune-based therapies”.

[Buonomo, E. et al, 2016. Microbiota-Regulated IL-25 Increases Eosinophil Number to Provide Protection during Clostridium difficile Infection. Cell Reports.](#)