Sara Dann, J Infect Dis Ther 2018, Volume 6
DOI: 10.4172/2332-0877-C2-040

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Polymicrobial bacterial infection increases host susceptibility to intestinal inflammation

Disease induced by Clostridium difficile infection (CDI) is generally viewed as "monomicrobial" being dominated by the virulence factors of CDI alone. However, co-infections may occur but their significance in CDI is unknown. Fecal specimens from pediatric patients (2-18 years) were screened using BioFire FilmArray GI Panel which detects 22 enteric pathogens. Of 357 patients, 88% had antibiotic-associated diarrhea. Based on toxin PCR, 50% were diagnosed with non-recurrent CDI (nCDI), 8% with recurrent CDI (rCDI), and 30% were C. difficile toxin negative (AAD). Patients without GI symptoms served as controls. FilmArray identified additional pathogens in 31.1% of patients with primary CDI; 64.5% with rCDI; 49.5% with AAD; and 11.9% controls. Enteropathogenic E. coli (EPEC) and rotavirus were significant co-infections in rCDI compared with nCDI ($p<0.05$). In a murine co-infection model, rotavirus improved clinical symptoms; whereas, co-infection with Citrobacter rodentium, a model of EPEC, resulted in greater disease and mortality than singly infected mice ($p<0.05$). Four weeks post-infection, co-infected mice showed significant intestinal inflammation that was not present in singly infected mice ($p<0.05$), which correlated with prolonged bacterial shedding and toxin production. Mortality in co-infected mice was associated with reductions in early response chemokines involved in the recruitment of protective innate immune cells. Administration of innate cytokine IL-22 protected co-infected mice from death compared to controls ($p<0.05$). Taken together, co-infections can exert differential clinical outcomes in CDI. Notably, co-infection with EPEC may place CDI patients at greater risk of disease recurrence because of pathogen-induced impairment in protective innate immunity against C. Difficile.

Biography

Sara Dann research focuses on understanding the interaction of enteric pathogens with the host mucosal immune system. Her goals are aimed at defining the role of common parasites, such as Giardia and Cryptosporidium, in intestinal inflammation and the involvement of innate immunity in this process. She is currently studying how dendritic cells and other innate cells initiate immune responses while maintaining intestinal tolerance. Understanding these processes will allow her to dissect the mechanisms involved in microbial-triggered colitis in genetically-susceptible hosts. Her findings might have very direct implications for the design of improved therapeutic and preventive strategies for the treatment of IBD.

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