PROPERDIN DEFICIENCY DOES NOT IMPACT THE MOUSE RESPONSE TO DSS-INDUCED COLITIS DESPITE DIFFERENCES IN COLONIC MICROBIOME


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Background: The role of complement in colitis is only beginning to be understood. We reported that properdin deficient mice (P\textsuperscript{KO}) have increased susceptibility to infectious colitis and to piroxicam-provoked colitis when combined with IL-10 deficiency. Here we examined the PKO strain’s response to chemical colitis, including their colon microbiome.

Aims: The aim was to determine whether properdin deficiency impacted the intestinal microbiome and response to DSS.

Methods: Second generation offspring from P\textsuperscript{KO} X C57BL/6 wildtype (WT) matings were used. Dextran sulfate sodium (DSS) was added to their water for 5 days, then groups of mice were killed either 1 (acute) or up to 5 days (recovery) later. The animals’ weights were recorded and stool collected and frozen. At necropsy their colons were extracted, measured, a scraping collected, and the remainder prepared for histology or cultured overnight for secreted mediators. DNA was isolated from stool and mucosal scrapes and the 16S rRNA gene was amplified and sequenced for microbiome analysis.

Results: All mice lost weight and became inflamed with no significant difference between strains in any measure of pathology or anaphylatoxin levels. This was despite a significant difference in the colon microbiome of healthy mice of the two strains, and the colitis resulting in significant changes in the microbiome of both strains. Interestingly, a greater change was detected in mucosal scrapes but not feces of WT compared to PKO mice.

Conclusions: We conclude that properdin does not play a role in chemical-induced colitis despite the mice hosting a different microbiome. Moreover, our results underscore how models of colitis may have different mechanisms including the relationship between complement and the microbiome.

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