Complement control of Klebsiella pneumoniae colonization and dissemination

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Klebsiella pneumoniae (Kpn), is a leading cause of opportunistic healthcare-associated infections, which are complicated by the presence of extensively drug resistant strains. The complement system is one of the first lines of host defense against pathogens. Despite its importance, the scale of activity within the gastrointestinal tract (GI) has not been fully determined. We aimed to dissect the role of complement components, C3 and C4, in the context of *Kpn* GI burden and systemic spread. Our data indicates C3 plays a pivotal role controlling *Kpn* burden. Nearly all $C3^{-/-}$ mice succumb to intragastric challenge, with high GI bacterial burden within the first few days of infection. Depletion of circulating C3 in wild type mice using cobra venom factor did not alter GI bacterial burden relative to control mice but significantly reduced survival, suggesting C3 controls *Kpn* growth in the GI tract whereas circulating C3 is critical for controlling dissemination. $C4^{-/-}$ mice similarly exhibit high levels of GI burden, but the majority of mice survive infection. Taken together, our data support C3/C4-dependent pathways predominantly control *Kpn* GI burden, whereas a C3-dependent (alternative) pathway may be critical for control of systemic dissemination in the naïve state.

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