Role of NKT Cells during *Chlamydia Trachomatis* Mouse Pneumonitis Lung Infection in Mice

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Abstract: *Chlamydia trachomatis* is an obligate intracellular pathogen that causes a variety of diseases, including trachoma, pelvic inflammatory diseases, infertility and pneumonia. Although Th-1 type cytokines have been demonstrated to be crucial in the host defense against *C.trachomatis* infection, the role of innate immune system, especially in mucosal immunity such as lung and genital tract, remains largely obscure. We investigated the role of invariant NKT (iNKT) cells in *C. trachomatis* lung infection. Jα18-/- gene knock out (KO) mice, which lack iNKT cells, and C57BL/6J wild type mice were infected intratracheally with *C.muridarum*. Compared to iNKT KO mice, wild type mice revealed significantly severe body weight loss, increased chlamydial in vivo growth and severe lung pathological changes. Systemic Chlamydia dissemination was also prominent in wild type mice. FACS analyses of lung infiltrating cells revealed that $CD11b^{+}Ly6G^{+}$ neutrophils and $CD11b^{+}Ly6C^{+}$ inflammatory macrophages greatly increased in wild type mice than in iNKT KO mice after infection. We found that increased mRNA MIP-2 and TNFα in lung tissues of WT mice but not in NKT-/- mice, suggesting a critical role of NKT cells in recruitment of neutrophils in the lung. Significantly higher levels of IL-4 and IL-13 were also found in lung homogenates of wild type mice with concomitant induction of Arginase-1 gene expression which is known to express in alternatively activated macrophages. Together, these results contribute to the understanding of how iNKT cells exacerbate *C.trachomatis* lung infection.

Key words: **Natural killer T cells, CD1d, Chlamydia trachomatis, lung mucosal immunity, macrophages**