Distinct DC subsets regulate adaptive Th1 and 2 responses during Trichuris muris infection

Low- and high-dose infections with the murine large intestinal nematode *Trichuris muris* are associated with induction of adaptive Th1 and Th2 responses, respectively, in mesenteric lymph nodes (MLN). Classical dendritic cells (cDC) accumulate in the large intestinal mucosa and MLN upon *T. muris* infection, yet their role in driving adaptive responses to infection remains largely unknown. We performed low- and high-dose *T. muris* infections of mice deficient in defined cDC subsets to investigate their role in induction of adaptive immune responses. Mice lacking IRF4-dependent cDC failed to clear a high-dose infection and displayed impaired Th2 responses. Conversely, mice lacking IRF8-dependent cDC cleared a low-dose infection and displayed an impaired Th1 response while increased production of Th2 cytokines. Finally, mice lacking both IRF4- and IRF8-dependent cDC were able to generate a Th2 response and clear a low-dose infection. Collectively, these results suggest that IRF4- and IRF8-dependent cDC act antagonistically during *T. muris* infection, and demonstrate that intestinal Th2 responses can be generated towards *T. muris* in the absence of IRF4-dependent cDC.

**General information**
Publication status: Published
Organisations: National Veterinary Institute, Mucosal Immunology, Lund University
Contributors: Demiri, M., Müller-Luda, K., Agace, W. W., Svensson Frej, M.
Publication date: 2017
Peer-reviewed: Yes

**Publication information**
Journal: Parasite Immunology
Volume: 39
Issue number: 10
Article number: e12458
ISSN (Print): 0141-9838
Ratings:
BFI (2017): BFI-level 1
Scopus rating (2017): CiteScore 2.56 SJR 1.173 SNIP 0.947
Web of Science (2017): Impact factor 2.836
Web of Science (2017): Indexed yes
Original language: English
Keywords: Dendritic cells, Infection, Intestine, Mesenteric lymph nodes, Parasite, T helper cells
DOIs:
10.1111/pim.12458
Source: FindIt
Source ID: 2390962153
Research output: Contribution to journal › Journal article – Annual report year: 2017 › Research › peer-review