Commentary
Volume 12:1, 2022

Journal of Blood & Lymph

ISSN: 2165-7831 Open Access

Brief Note on Immune Defense against Pathogenic Infection

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Description

Monocytes are a type of leukocyte that plays a variety of important and diverse roles in the host's immune response to pathogen infection. There are two types of murine monocytes: Ly6C^{hi} cells and CX3CR1^{hi} cells. Ly6C^{hi} monocytes express CCR2 but not CX3CR1. In response to inflammation, Ly6C^{hi} monocytes exit the Bone Marrow (BM) in a CCR2-dependent manner and migrate to infection sites. Ly6C^{hi} inflammatory monocytes can differentiate into macrophages or Dendritic Cells (DCs) and perform phagocytosis, cytokine secretion, antigen transport, and T cell priming. Monocytes expressing CX3CR1 have low levels of Ly6C and CCR2. In the intestinal lamina propria, CX3CR1^{hi} monocytes patrol the luminal endothelium and can differentiate into macrophages. Furthermore, Ly6C^{hi} monocytes recruited to the infected intestinal lamina propria can develop into CX3CR1-negative inflammatory DCs.

In bacterial infections, CCR2^{+} Ly6C^{hi} inflammatory monocytes have been assigned a variety of functions. During systemic Listeria monocytogenes infection, inflammatory monocytes can differentiate into Tip-DCs (Tumour Necrosis Factor alpha [TNF-] and inducible Nitric Oxide Synthase [iNOS]-producing DCs), which are important for innate host defence. *In situ* imaging revealed that IMs are recruited to L Monocytogenes infection foci in the spleen and liver. Tip-DCs, on the other hand, were not required to elicit CD8^{+} or CD4^{+} T cell responses in mice infected with L monocytogenes. IMs were not required to control initial bacterial growth in the lungs in a Mycobacterium tuberculosis aerosol infection model, but they were essential for priming a CD4^{+} T cell response. In this role, IMs transported M. tuberculosis from the lung to the lymph nodes, where DCs primed the CD4^{+} T cell response. As a result, during bacterial infection of mice, IMs can play distinct and important roles in both the innate and adaptive phases of immunity.

The delivery of T3SS effectors into phagocytes by *Y. pseudotuberculosis* provides antigen-presenting cells with peptides that can be presented to CD8^{+} T cells *via* Major Histocompatibility Complex Class I(MHCI). YopE(YopE69-77) from. *Y pseudotuberculosis* contains an immunodominant H-2Kb-restricted CD8^{+} T cell epitope located N terminal to the GAP domain. Primary *Y. Pseudotuberculosis* infection of C57BL/6 mice results in a dominant CD8^{+}T cell response to the YopE69-77 epitope in intraepithelial lymphocytes, the lamina propria, MLN, spleen, and liver.

Naive mice vaccinated with a YopE69-77 peptide epitope are partially protected against subsequent lethal intestinal infection, indicating that preexisting YopE69-77-specific CD8^{+} T cells can provide measurable immunity. YopE69-77-specific CD8^{+} T cells are produced as a result of vaccination or during primary Y. TNF- and gamma interferon (IFN) are produced during pseudotuberculosis infection, and these cytokines are thought to mediate the protective effect. We previously used i.v. infection of mice with attenuated *Y. Pseudotuberculosis* to investigate the features of YopE and the host immune response that are important for the production of large numbers of YopE69-77-specific CD8^{+} T cells. Because we discovered that YopE catalytic activity was not required for the large YopE69-77-specific CD8^{+} T cell response, we used an attenuated yopE GAP mutant in these studies. When infected with a Y, CCR2$^{-/-}$ mice, which are defective in the recruitment of IMs from bone marrow, had a significant defect in the production of YopE69-77-specific CD8^{+} T cells. YopE GAP mutant in pseudotuberculosis furthermore, when infected with the yopE GAP mutant, CCR2$^{-/-}$ mice were more susceptible to lethal infection than wild-type controls, indicating that IMs are important for the control of *Y. Pseudotuberculosis* in addition to their role in the production of YopE69-77-specific CD8^{+} T cells. To expand on the previously described findings, we looked into the role of IMs in immune responses during the natural oral route of infection with wild-type *Y. Pseudotuberculosis*. Surprisingly, we discovered that infected CCR2/mice were not more susceptible to lethality from oral infection, but instead retained body weight better starting at day 4 postinfection, cleared *Y. Pseudotuberculosis* from MLNs more quickly, and had less mesenteric lymphadenopathy at day 14 postinfection compared to CCR2$^{+/+}$ controls. Thus, IMs appeared to contribute to the persistence of *Y. pseudotuberculosis* and lymphadenopathy in MLNs.

Green Fluorescent Protein-Positive (GFP^{+} IMs were recruited to the periphery of neutrophil-rich Yersinia-containing pyogranulomas as early as day 3 postinfection, according to *in situ* imaging of MLNs and spleens from CCR2-GFP mice. GFP^{+} IMs colocalized in MLNs with YopE69-77-specific CD8^{+} T cells, CD11c^{+} cells, and Ly6G^{+} neutrophils, implying that IM-derived DCs prime adaptive responses in Yersinia pyogranulomas. As a result, CCR2$^{-/-}$ mice had fewer YopE69-77-specific CD8^{+} T cells, fewer CD4^{+} T cells and B cells, and lower serum antibody responses to Y. Antigens of pseudotuberculosis on day 14 after

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Received: 03-Feb-2022, Manuscript No. JBL-22- 56508; **Editor assigned**: 05-Feb-2022, Pre QC No JBL-22- 56508 (PQ); **Reviewed:** 17-Feb-2022, QC No JBL-22-56508; **Accepted:** 19-Feb-2022, Manuscript No. JBL-22- 56508(A); **Published**: 26-Feb-2022, DOI: 10.37421/ 2165-7831.2022.12.279

infection. These findings indicate that IMs recruited to Yersinia pyogranulomas promote lymphadenopathy and bacterial persistence in MLNs while also promoting adaptive immune responses during oral infection. $CCR2^{-/-}$ mice with weakened adaptive immunity may be protected from oral Y lethality. In the absence of IMs, *Y. pseudotuberculosis* infection is mitigated in part by decreased mesenteric lymphadenopathy and increased innate responses.

The immune system is a complex network of cells and molecules that work together to protect the host from pathogenic microorganisms and exogenous noxious agents found in our environment. However, host tissue destruction may occur if the immune response is insufficient due to intrinsic or extrinsic mechanisms in cell function, or if there is hyper-responsiveness due to dysfunctional regulatory mechanisms. The pathogenesis of periodontal disease is clearly inflammatory, and as such, it has a close relationship with the immune system.

How to cite this article: Ghimire, abishkar. "Brief Note on Immune Defense against Pathogenic Infection ." *J Blood Lymph 12* (2022) : 279.