Research Highlight

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IN BRIEF

B cells: Protective role of innate-like B cells in sepsis

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This study describes a protective immune role for a previously uncharacterized population of innate-like B cells. When mice were systemically treated with lipopolysaccharide (LPS) or infected with Escherichia coli, cells that produced granulocyte–macrophage colony-stimulating factor (GM-CSF) accumulated in the spleen.

Surprisingly, most of these cells were IgM+ B cells, and transfer studies showed that they develop from innate-like B-1 cells. In response to LPS, peritoneal B-1 cells proliferated, migrated to the spleen and gave rise to the GM-CSF+ B cell population. Development of the GM-CSF+ B cells depended on BAFFR, TLR4 and MYD88, and these cells were retained in the spleen by the integrins VLA4 and LFA1. In a model of sepsis, mice with a B cell-restricted deficiency in GM-CSF showed increased neutrophil infiltration to the peritoneum. However, these neutrophils had impaired phagocytic activity, and the mice experienced a severe cytokine storm and died. This suggests that GM-CSF-producing B cells contribute to bacterial clearance by promoting neutrophil phagocytic functions.

References and links

ORIGINAL RESEARCH PAPER


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