

NF- κ B2/p100 deficiency impairs immune responses to T-cell-independent type 2 antigens.

Krljanac B, Weih D, Jacobsen ID, Hu D, Koliesnik I, Reppe K, Witzenrath M, Weih F (2014) NF- κ B2/p100 deficiency impairs immune responses to T-cell-independent type 2 antigens. *Eur J Immunol* 44(3), 662-672.

[Details](#)



Abstract

Formation of the splenic marginal zone (MZ) depends on the alternative NF- κ B signaling pathway. Recently, we reported that unrestricted activation of this pathway in NF- κ B2/p100-deficient (p100(-/-)) knock-in mice alters the phenotype of MZ stroma and B cells. Here, we show that lack of the p100 inhibitor resulted in an expansion of both MZ B and peritoneal B-1 cells. However, these cells failed to generate proliferating blasts in response to T-cell-independent type 2 (TI-2) Ags, correlating with dampened IgM and absent IgG3 responses. This phenotype was in part due to increased activity of the NF- κ B subunit RelB. Moreover, p100(-/-) $\rightarrow$ B6 BM chimeras were more susceptible to infection by encapsulated *Streptococcus pneumoniae* bacteria, pathogens that induce TI-2 responses. In contrast to the TI-2 defect, p100 deficiency did not impair immune responses to the TI-1 Ag LPS and p100(-/-) MZ B cells showed normal Ag transportation into B-cell follicles. Furthermore, p100(-/-) MZ B and B-1 cells failed to respond to TI-2 Ags in the presence of WT accessory cells. Thus, NF- κ B2/p100 deficiency caused a predominant B-cell-intrinsic TI-2 defect that could largely be attributed to impaired proliferation of plasmablasts.

Importantly, p100 was also necessary for efficient defense against clinically relevant TI-2 pathogens.

Beteiligte Forschungseinheiten

[Mikrobielle Immunologie Ilse Jacobsen](#) [Mehr erfahren](#)

Leibniz-HKI-Autor*innen



Ilse Denise Jacobsen

[Details](#)

Identifier

doi: 10.1002/eji.201343484

PMID: 24242887