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PTP1B is a negative regulator in the host defense against *Pseudomonas aeruginosa* infection

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Pseudomonas aeruginosa is a major opportunistic pathogen in immune-compromised individuals. Toll-like receptors (TLRs) contribute to innate immunity against *P. aeruginosa* through activation of transcription factors IRF7/IRF3 and NFκB. However, mechanisms involved in the regulation of *P. aeruginosa*-induced TLR pathway activation remain incompletely defined. Here, we demonstrate that protein tyrosine phosphatase-1B (PTP1B) is a critical negative regulator in *P. aeruginosa* infection. PTP1B-deficient mice display greatly enhanced bacterial clearance which is accompanied with increased neutrophil infiltration and cytokine production. Interestingly, PTP1B-deficiency mainly up-regulates the production of IFN-stimulated response elements (ISRE)-regulated cytokines and chemokines including CCL5 (RANTES), CXCL10 (IP-10) and IFN-β production. Further studies reveal that PTP1B-deficiency leads to increased IRF7 activation. Importantly, PTP1B is physically associated with IRF7 in dendritic cells. These findings demonstrate a novel regulatory mechanism of the immune response to *P. aeruginosa* infection through PTP1B-IRF7 interaction. This novel PTP1B-IRF7-ISRE pathway may have broader implications inTLR-mediated innate immunity.

Biography

Tong-Jun Lin is working as a Professor in Department of Microbiology and Immunology. His research interests include signaling mechanisms in allergy, host defense mechanisms against bacterial infection, and immune response in cancer development. His work is interdisciplinary, collaborative and geared both to contributing to the academic literature and to developing immunological therapeutic approaches in inflammation.

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