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Myeloid KLF2 regulate host response to infection

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Abstract

Inflammation is characterized by recruitment of myeloid cells to the site of injury. Macrophages derived from circulating monocytes are plastic in nature and can modulate their response to local microenvironment. These activated macrophages are critical in eliminating source of inflammation in acute inflammatory events such as bacterial infection. However, this activation of myeloid cells must be under exquisite control to prevent uncontrolled inflammation. Indeed, macrophage activation is an exquisitely robust biological response that involves transcriptional alterations in gene expression affecting a substantial part of the cellular genome. The transcriptional modules that drive this response fashion the phagocytes with a multipronged armamentarium against invading microorganisms that includes the elaboration of numerous antimicrobial peptides, cytokines, chemokines, and reactive nitrogen and oxygen species. Recent studies from our group have identified that Kruppel-like transcription factor 2 as a potent regulator of myeloid cell activation *in vivo*. Exposure of myeloid cells to hypoxia and/or bacterial endotoxins reduced KLF2 expression while inducing hypoxia inducible factor-1α (HIF-1α). Myeloid KLF2 was found to be a potent inhibitor of nuclear factor-kappaB (NF-□B)-dependent HIF-1α transcription and, consequently, a critical determinant of outcome in models of polymicrobial infection and endotoxemia. Collectively, these observations identify KLF2 as a tonic repressor of myeloid cell activation and an essential regulator of the innate immune system.

Biography

Ganapati H. Mahabaleshwar is an assistant professor of Medicine at Case Western Reserve University School of Medicine. Dr. Mahabaleshwar earned his Ph.D. in Biotechnology from the Pune University, Pune, India. He completed post-doctoral fellowships at the Cleveland Clinic and Case Western Reserve University. His laboratory studies transcriptional regulation of innate immune cell response to chronic and acute inflammatory stimuli.