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Highlighted Article

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HIGHLIGHTED ARTICLE

Frontline Science: Targeted expression of a dominant-negative high mobility group A1 transgene improves outcome in sepsis

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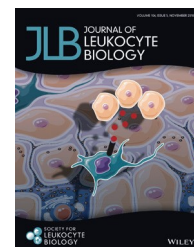
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Abstract
High mobility group (HMG) proteins are a family of architectural transcription factors, with HMG1A playing a role in the regulation of genes involved in promoting systemic inflammatory responses. We speculated that blocking HMG1A-mediated pathways might improve outcomes from sepsis. To investigate HMG1A further, we developed genetically modified mice expressing a dominant-negative HMG1A transgene (HMG1A^{DN}) targeted to the vasculature. In HMG1A^{DN} transgenic (TG) mice, endogenous HMG1A is present, but its function is decreased due to the mutant transgene. These mice allowed us to specifically study the importance of HMG1A not only during a purely inflammatory insult of endotoxemia, but also during microbial sepsis induced by the implantation of a bacterial catheter into the peritoneum. We found that the HMG1A^{DN} transgene was able to improve survival in TG and not wild-type (WT) littermate mice, and the mutant transgene was able to interact with transcription factors, such as NF- κ B, but was not able to bind DNA. TG mice exhibited attenuated hyperinflammatory responses to endotoxemia, and less mortality in microbial sepsis. Moreover, TG mice had a reduced inflammatory response during sepsis, with decreased macrophage and neutrophil infiltration into tissues, which was associated with reduced expression of proinflammatory cytokines, chemokines, and matrix metalloproteinases. Collectively, these data suggest that a targeted expression of a dominant-negative HMG1A transgene is able to improve outcomes in models of endotoxemia and microbial sepsis, in part by modulating the immune response and suggest a novel modifier pathway to target therapeutics in sepsis.

KEYWORDS
architectural transcription factor, chemokines, immune response, transgenic mice

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