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Circulating histone-induced acute lung injury

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Although intra-nuclear histones play essential roles in DNA packaging and gene regulation, released histones following extensive cell or organ damage are toxic to pathogens but also to host hematopoietic, endothelial and epithelial cells. Cellular toxicity mainly results from direct membrane binding and resultant calcium influx with our work showing that this can directly trigger neutrophil MPO release and NETosis. In patients with severe trauma and sepsis, we found that high circulating histone levels correlated significantly to the incidence of acute lung injury (ALI) as well as markers of endothelial damage and coagulation activation. Using histone-infusion mouse models we showed ALI with oedema, neutrophil congestion, NETs and thrombus formation, which thereby impair pulmonary microcirculation as indicated by pressure increase and even enlargement of right ventricle in extreme conditions. As the lungs are the predominant sites of neutrophil margination and alveolar neutrophil infiltration is the hallmark of ALI, histone-induced neutrophil cogestion, MPO release and NETs formation may provide an explanation as to why lungs are more susceptible to histone toxicity than other organs.

Biography

Guozheng Wang has both medical and biological backgrounds. He practiced internal medicine in China for 12 years and has been performing full time biomedical research in UK Universities (Cambridge, Oxford and Liverpool) for over 20 years.

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