

Accepted Manuscript

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PII: S0945-053X(17)30407-9
DOI: doi:[10.1016/j.matbio.2017.11.013](https://doi.org/10.1016/j.matbio.2017.11.013)
Reference: MATBIO 1381

To appear in: *Matrix Biology*

Received date: 15 November 2017
Revised date: 30 November 2017
Accepted date: 30 November 2017



Please cite this article as: Dhana, Ermanila, Ludwig-Portugall, Isis, Kurts, Christian, Role of immune cells in crystal-induced kidney fibrosis, *Matrix Biology* (2017), doi:[10.1016/j.matbio.2017.11.013](https://doi.org/10.1016/j.matbio.2017.11.013)

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Role of immune cells in crystal-induced kidney fibrosis

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Abstract

Chronic kidney diseases can lead to kidney fibrosis, which can be considered a futile attempt of tissue healing to replace functional kidney tissue with connective tissue, basically forming a scar. Chronic inflammation is a frequent cause of kidney fibrosis. Classical as well as recently discovered immune cell subsets and their molecular mediators have been intensively investigated for their contribution to kidney fibrosis and their potential as therapeutic targets. Here we review the current knowledge about the role of immune cells in crystal-induced renal fibrosis.

Introduction

Fibrotic diseases are a major health problem worldwide with increasing prevalence due to the modern lifestyle. The various types of chronic kidney disease (CKD), which affect 8-16% of the global population, can all lead to fibrosis [1]. Fibrosis results from an imbalance between synthesis and degradation of extracellular matrix (ECM). The composition of this matrix affects the structure of glomeruli and the tubular interstitium in the kidney, thereby contributing to the loss of kidney function [2]. A frequent cause of fibrogenesis is chronic inflammation, which may result from different causes. Ischemia-reperfusion injury (IRI) results from temporarily restricting and the restoring

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