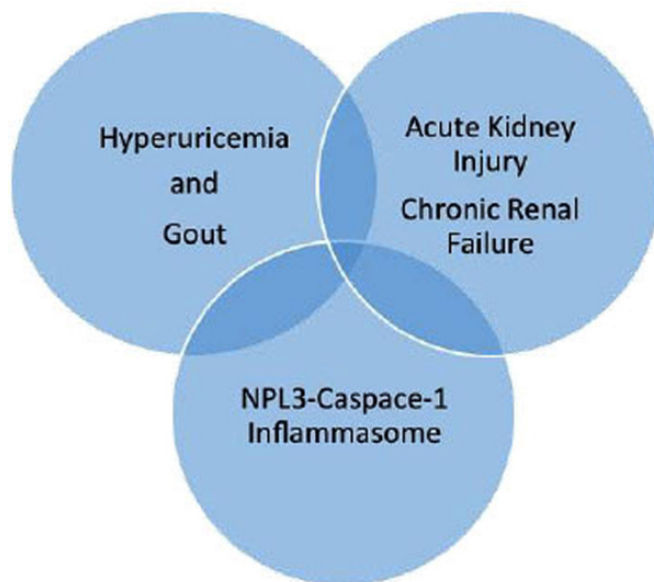


# Uric acid, gout and kidney disease: The chicken or the egg?

28 March 2016



Hyperuricemia and gout are commonly associated with both acute chronic kidney disease. Common to both conditions is stimulation of the innate immune system with activation of the NLRP3 inflammasome, generation of IL-1 $\beta$  and IL-18, and release of cytokines and chemokines. Credit: Vandana Dua Niyar, MD, Bentham Science Publishers

The increasing prevalence of both gout and chronic kidney disease has led to a growing interest in the association between hyperuricemia (an abnormally high level of uric acid in the blood) and kidney disease.

A new thematic issue of *The Open Urology & Nephrology Journal*, titled 'Current Perspectives in Hyperuricemia, Gout and the Kidney,' reports on the interplay of various factors, particularly the role of the kidney in [uric acid](#) excretion on the one hand, and the possible impact of hyperuricemia on progression of renal disease on the other. The common patho-physiological link appears to be the chronic, low-grade, systemic inflammation that is

intrinsic to both conditions, and that may explain some of the perplexing observations noted in these clinical conditions.

This thematic issue discusses the effect of the activation of the innate immune system, through stimulation of the NLRP3 inflammasome, leading to the subsequent generation of interleukins and the release of cytokines and chemokines, and how these factors interact in the complex interplay between hyperuricemia, [gout](#) and [kidney](#) disease.

Additionally, with the recent updates in clinical management guidelines for acute and chronic gout, and given that there are special considerations in specific patient populations, articles in the issue incorporate recommendations from three different medical perspectives: the primary care physician, the rheumatologist and the nephrologist.

**More information:** William F. Finn. Kidney Disease and Gout: The Role of the Innate Immune System, *The Open Urology & Nephrology Journal* (2016). [DOI: 10.2174/1874303X01609010012](https://doi.org/10.2174/1874303X01609010012)

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