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## The role of soluble uric acid and uric acid crystals in chronic kidney disease

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Uric acid (UA) and chronic kidney disease (CKD) have a bidirectional relationship. The development of CKD has been associated with increased UA levels due to decreased urinary excretion of UA and resultant hyperuricemia, a major risk factor for gouty arthritis. Mild CKD often leads to an increased prevalence of gouty arthritis, as prospective observational studies have demonstrated, whereas advanced CKD and end-stage renal disease (ESRD) leads to a drop in the prevalence despite massive hyperuricemia. Conversely, there is evidence that gout and associated hyperuricemia may independently impair kidney function. Hyperuricemia in the presence or absence of gout, the use of gout medications, and the deposition of UA microcrystals in the medullary interstitium can potentially be harmful to the kidney, increase the risk for developing CKD, and are progressive factors for worsening CKD. However, these clinical data are conflicting and currently, there is no ideal animal model available to investigate the molecular effects of UA and gout in CKD in more detail, which raises the following unanswered questions: What is chicken-and-egg, CKD causes hyperuricemia but does hyperuricemia also lead to the progression of CKD? and by what mechanism? Which further raises the question: do CKD patients profit from uricostatic pharmacotherapy? What are the molecular mechanisms behind an increased prevalence of gouty arhtritis in mild CKD and a drop-in prevalence in advanced CKD? Does hyperuricemia affect UA crystal-induced inflammation by priming inflammatory cells, and is therefore responsible for the lower risk of acute gouty arthritis with advanced CKD and ESRD? These questions will be addressed in the oral presentation with supportive clinical data and an in *vivo* animal model.

### Biography

Stefanie Steiger has studied Bioengineering at the Martin-Luther University Halle, Germany from 2000 until 2006 followed by a working contract at Boehringer Ingelheim for two years. In 2008, she completed her PhD in Medical Science at Malaghan Institute of Medical Research, Victoria University Wellington, New Zealand, and two years of Post-doctoral fellowship. She moved to University of Munich, Germany in 2014, where she established her own group. Her research focus is the bidirectional impact of uric acid and chronic kidney disease.

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