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Comment on: Gout: an independent risk factor for all-cause and cardiovascular mortality: reply

Sir, We acknowledge the comments of Mascitelli et al. [1] regarding our recent publication [2]. As an epidemiological study, our recent study cannot provide the underlying mechanism resulting in different mortality impact between gout and hyperuricaemia. We concur that inflammation is the underlying distinction between gout and hyperuricaemia. As low-grade inflammation has been demonstrated to be of importance in the development of atherosclerosis and subsequent cardiovascular events [3, 4], higher mortality risk of gout in contrast to pure hyperuricaemia is conceivable. The recent finding that monosodium urate crystal is a trigger of inflammasome [5], which is the pivotal molecular complex in the maturation of IL-1, can also explain the presence of inflammation in gout, but not pure hyperuricaemia. Whether iron overload is related to the difference is subject to further investigation.

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