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## Response to Letter by Proctor

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# Letters to the Editor

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## Response to Letter by Proctor

### Response:

Dr Proctor<sup>1</sup> raises several important issues which concur with Amoro and colleagues' conclusion<sup>2</sup> that the potential benefits of elevated serum uric acid are independent of the likely relationship between chronically elevated serum uric acid and cardiovascular disease. Thanks to Amoro et al, there is now data from both the basic science and clinical research arenas to suggest that infusion of uric acid after stroke could reduce free radical-mediated injury, a potential treatment for an area sorely in need of an advance. However, as discussed, data exist to support uric acid reduction as a preventative strategy in those at risk of atherosclerotic disease.

It seems unlikely, however plausible, that explanations for a circumstantial beneficial or detrimental effect of uric acid may be that these differences will not lead to confusion in wider nonspecialist circles. Such confusion could ultimately hinder adoption of these strategies in either research or clinical circles—"you want me to administer uric acid, but I thought uric acid was bad for you?" As such, we welcome the attention given to this area in recent issues of *Stroke*.

Proctor also highlights evidence suggesting that uric acid itself may mediate oxidative stress-related damage, rather than this being a consequence of its mechanism of generation (xanthine oxidase activity). Again, there is evidence for (some of which he highlights) and against this hypothesis. A series of elegant

studies by Struthers et al<sup>3</sup> suggest that the beneficial effects of allopurinol on a variety of measures of endothelial function relate to a reduction in xanthine oxidase activity, and not uric acid reduction while other data suggest uric acid reduction is important.<sup>4</sup> Clearly, more studies are needed to disentangle causal mechanisms so we can better direct evaluation of potential therapies.

## Disclosures

None.

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