The management of hyponatremia in HIV disease

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Dear Editor,

We thank Musso and colleagues for their interest in our work [1]. They report evidence of a hitherto unreported signal for potential tubular dysfunction and impaired water handling in patients with HIV disease that is independent of anti-retroviral therapy and glomerular filtration rate [2]. Extensive mechanistic studies have suggested direct invasion of tubular epithelial cells by HIV virus with both in vitro and in vivo evidence of direct pathogenicity (see review) [3]. Notably, this invasion has been reported in the absence of detectable viremia [4]. Their study, in our opinion, may mark the first physiological documentation of these effects.

We would, however, like to raise the following caveat. The use of creatinine-based estimations may be significantly affected by creatinine generation (i.e., muscle mass), specifically in the HIV population [5]. The measurement of renal function (i.e. GFR) in these patients should be validated using a more specific non-creatinine assay to truly

ascribe this impaired handling to a tubular effect that is independent of GFR.

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