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(Accepted for publication August 14, 2012.)

Hemorrhagic Shock and Acute Kidney Injury Model

To the Editor:

We have read with interest the review by Yap *et al.*[1] on acute kidney injury and extra renal organ dysfunction. We agree with their comments on the limitations of previously described models based on nephrotoxicants to explore acute kidney injury in animals. Surprisingly the authors wrote that ""severe and prolonged hypotension in rats does not typically induce renal injury and is therefore not suitable for use as a "single insult" animal model."" Indeed, in a recent experimental study in mice, we have shown that a 2- hr pressure control hemorrhagic shock in mice induces prolonged decrease in glomerular filtration rate, histological lesions, and molecular hypoxic impact.[2] Moreover, 3 weeks after injury, sparse renal fibrosis and persistent tubular function abnormalities are present. In many respects and despite the limits of such anthropomorphism, this single-insult model reproduces closely both functional and histologic features of human ischemic acute kidney injury. This work and others sustain the potential interest of such a model.[3]

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(Accepted for publication August 15, 2012.)

In Reply:

We thank Mayeur *et al.* for their interest in our review on acute kidney injury and extra renal organ dysfunction.[1] As stated in our review, "single-insult" kidney injury models including complete renal ischemia, nephrotoxins, nephrectomy, or renal hypoperfusion do not completely recapitulate the multifactorial causes of clinical acute kidney injury. Single-insult models of kidney injury, however, have been invaluable in demonstrating the effects of acute kidney injury-mediated remote and multiorgan dysfunction.[2] We agree with Mayeur *et al.* that the hemorrhage and resuscitation model is used to produce multiorgan injury as well