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Role of Mast Cells in Renal Fibrosis

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To the Editor

Summers et al[1] recently published their observations that mast cells are crucial to renal fibrosis induced by ureteral obstruction. This work confirmed our report showing that mast cells are required for the development of renal fibrosis in the rodent unilateral ureteral obstruction (UUO) model[2]. Our manuscript was published online September 28th, 2011. We would like to take this opportunity to expand on this role of mast cells in renal fibrosis, and advance a hypothesis for the underlying mechanism. In view of the fact that mast cells are found in close proximity to fibroblasts in UUO kidneys, we hypothesize that mast cell mediators released during degranulation are responsible for fibroblast proliferation and activation ultimately leading to fibrosis[3]. Recent findings from our lab demonstrate that kidney fibroblasts express the ANG II AT_1 receptor (R) and the histamine H_1R subtypes. Addition of ANG II and histamine to kidney fibroblasts in culture, promote proliferation, TGF-βsynthesis and collagen production[3]. These results, along with the findings of Veerappan et al[2] and Summers et al[1] suggest that release of mast cell mediators like renin (ANG II) and histamine, provide a mechanism that couples mast cell degranulation to fibroblast activation with the ensuing fibrosis. Targeting of mast cells and their products may represent novel therapeutic targets for preventing renal fibrosis.

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