

Macrophage COX-2 prevents diabetic nephropathy progression

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(HealthDay)—Macrophage cyclooxygenase-2 (COX-2) deletion is

associated with progression of diabetic nephropathy (DN), according to an experimental study published online Nov. 4 issue of *Diabetes*.

Xin Wang, from the Vanderbilt University School of Medicine in Nashville, Tenn., and colleagues examined the effects of macrophage COX-2 on development of DN in a mouse model of type 1 diabetes.

The researchers found that cultured macrophages with deletion of COX-2 exhibited a proinflammatory "M1" phenotype, with higher inducible nitric oxide synthase and NF- κ B levels, but lower levels of interleukin-4R α . Mice with COX-2 deletion in hematopoietic [cells](#) or macrophages developed severe DN compared with corresponding wild type diabetic mice, as indicated by increased albuminuria; fibrosis; and renal infiltration of T cells, neutrophils, and macrophages. Diabetic kidneys with macrophage COX-2 deletion had more infiltration of macrophages, but fewer renal "M2" [macrophages](#) (reparative phenotype); they also had increased ER stress and decreased numbers of podocytes. Results were similar in diabetic [mice](#) with macrophage E4 deletion.

"These studies have demonstrated an important but unexpected role for macrophage COX-2/PGE2/EP4 signaling to lessen progression of [diabetic kidney disease](#), unlike the pathogenic effects of increased COX-2 expression in intrinsic renal cells," the authors write.

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