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# The role of the ErbB receptor family in progressive kidney disease and metabolic syndrome

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The Epidermal Growth Factor Receptor (EGFR), a member of the family of ErbB receptors, can be activated by a family of ligands. EGFR and its ligands are expressed in a variety of cell types, including myeloid origin in kidney. Persistent EGFR activation contributes to the development of tubular interstitial fibrosis in type I and type diabetes. Mice with either intrinsic EGFR activation (Dsk5 mice) or overexpression of human HB-EGF in proximal tubule develop spontaneous tubular interstitial fibrosis. In high fat diet-induced obesity, mice with EGFR deletion in myeloid cells develop less fibrosis in epididymal fat. EGFR itself is also highly expressed in myofibroblasts in patients with kidney fibrosis and in mouse fibrotic model. Mice with selective deletion of EGFR in fibroblasts develop less fibrosis in UUO, ischemic AKI, folic acid and adenine nephropathy. snRNAseq and flow cytometry and migration analysis demonstrate that EGFR activation early after insults leads to pericyte/fibroblast migration and proliferation, and then TGF-β and other profibrotic factor transform fibroblasts into myofibroblasts with subsequently increased extracellular matrix.

**Keywords:** EGFR, diabetic nephropathy , fibrosis, acute kidney injury , chronic kidney disease