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effacement due to the presence of accumulated extracellular matrix and inflammatory cells. Circulating pro-inflammatory monocyte/macrophage- and T-cell-derived cytokines along with local resident inflammatory cells are thought to play a crucial role in the pathogenesis of glomerulosclerosis. In view of the central role of B cells in regulating immunity, this review addresses the pro-inflammatory role of these cells in FSGS. We reviewed the published literature on the role of circulating and resident B cells in the pathogenesis of glomerulosclerosis. The various paracrine and autocrine effector cytokines and chemokines, which have been shown to be regulated by B cells in vitro and in vivo, are highlighted. The potential role of B cells in the recruitment of inflammatory cells to the glomeruli is described. Evidence

is provided for the autocrine regulation of B-cell survival by monocyte-derived tumour necrosis factor-alpha. The involvement of B cells in the production of antibodies may serve as an alternate mechanism in autoantibody-mediated glomerulonephritis. The role of B cells in specific forms of glomerulosclerosis, such as lupus nephritis and membranous nephropathy, is also discussed. B cells are highly capable of regulating the functions of other immune cells via multiple mechanisms. These cells may play a crucial role in the initiation and progression of autoimmune-mediated renal diseases, such as FSGS and I 6d1f23a050

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