**Table S1.** Influences of kidney disease progression by interfering with immune and non-immune cell metabolic pathways in literature

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| --- | --- | --- | --- | --- | --- | --- |
| **Human/Mouse** | **Disease model** | **Cells** | **Treatment** | **Disrupted metabolic pathways** | **Renal Outcomes** | **Ref.** |
| Mouse | IgAN | DCs | Overexpression IDO | Tryptophan  metabolic | Alleviate injury | (27) |
| Mouse | UUO | Macrophages | Dichloroacetate/  shikonin | Glycolysis | Alleviate fibrosis | (44) |
| Mouse | UUO | NK cells | mTOR inhibitors | Glycolysis | Alleviate fibrosis | (49,50) |
| BALB/C mice | SLE | Th1/Th2/Th17/DCs | rapamycin | Glycolysis | Alleviate injury | (63) |
| Mouse | UUO | Fibroblasts | shikonin and  2-deoxyglucose | Glycolysis | Alleviate fibrosis | (119) |
| Mouse | UUO | proximal TECs | Tsc1 | Glycolysis | Alleviate fibrosis | (99) |
| Mouse | Folate-induced injury model/UUO | TECs | fenofibrate | FAO | Alleviate fibrosis |  |

IgAN, IgA nephropathy; DCs, dendritic cells; IDO, indoleamine 2,3-dioxygenase; l, Tsc1, tuberous sclerosis complex 1; TECs, tubular epithelial cells; FAO, fatty acid oxidation.