

# ImmunoTools IT-Box-Cy55M-Award 2013



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### Role of inflammatory cytokines and chemokines in glomerular disease.

Glomerular injury (Glomerulosclerosis) results in Chronic Kidney Disease (CKD) which may lead to End Stage Renal Disease (ESRD). Focal segmental glomerulosclerosis (FSGS) is one of the most common forms of acquired glomerular disease leading to ESRD.

It has been known that phenotype switch of macrophages from M1 (inflammatory) to M2 (Regenerative) is important in the development of FSGS. But so far the exact mechanism for the phenotype switch during FSGS and the exact roles of this phenotype switch are not known. We seek to understand this phenomenon during the development and progression of FSGS.

We are interested in studying different factors which are responsible for this phenotype switch e.g. rmCSF-1, rmIL-25 and others. In *in-vitro* studies, we isolate the bone marrow cells and differentiate them into macrophages in the presence of rmM-CSF. These are then converted into either M1 inflammatory phenotype in the presence of rm-IFN $\gamma$ , LPS or M2 regenerative phenotype in the presence of rmIL-4, rmIL-1 $\beta$ , rmIL-10, rmTGF $\beta$  etc. These cells will then be co-cultured with podocytes and mesangial cells to study their effect.

We will also inject the *in-vitro* derived M1 and M2 macrophages in *in-vivo* murine models of FSGS, to study their effects on the inflammatory and repair phase of the disease. We are also interested in studying the effect of different cytokines secreted by macrophages e.g. rmIL-1 $\alpha$ , rmIL-6, rmIL-22, rmIP-10, CXCL10, rmMCP-1, rm\_MIP-1, rmTNF $\alpha$  etc. This will be studied using both *in-vitro* as well as *in-vivo* models.

This allows us to dissect the functional role of the different macrophage phenotype during the progression and development of FSGS.

### ImmunoTools IT-Box-Cy55M for Santosh Kumar includes 55 recombinant mouse cytokines

rm EGF, rm Eotaxin / CCL11, rm FGF-a / FGF-1, rm FGF-b / FGF-2, rm FGF-8, rm Flt3L / CD135, rm G-CSF, rm GM-CSF, rm GRO-a / CXCL1, rm GRO-b / CXCL2, rm IFN $\gamma$ , rm IL-1 $\alpha$ , rm IL-1 $\beta$ , rm IL-2, rmIL-3, rm IL-4, rm IL-5, rm IL-6, rm IL-7, rm IL-9, rm IL-10, rm IL-11, rm IL-13, rm IL-15, rm IL-16, rm IL-17A, rm IL-17C, rm IL-17F, rm IL-19, rm IL-20, rm IL-21, rm IL-22, rm IL-25 / IL-17E, rm IL-27, rm IL-31, rm IL-33, rm IP-10 / CXCL10, rm LIF, rm MCP1 / CCL2, rm M-CSF, rm MIP-1 $\alpha$  / CCL3, rm MIP-1 $\beta$  /

CCL4, rm MIP3 $\alpha$  / CCL20, rm MIP3 $\beta$  / CCL19, rm NGF-beta, rm PDGF-AA, rm PDGF-BB,  
rm RANTES / CCL5, rm sCD40L / CD154, rm SCF, rm SDF-1 $\alpha$  / CXCL12a, rm SDF-1 $\beta$  /  
CXCL12b, rm TNF $\alpha$ , rm TPO, rm VEGF [DETAILS](#)