MolecularMedicine

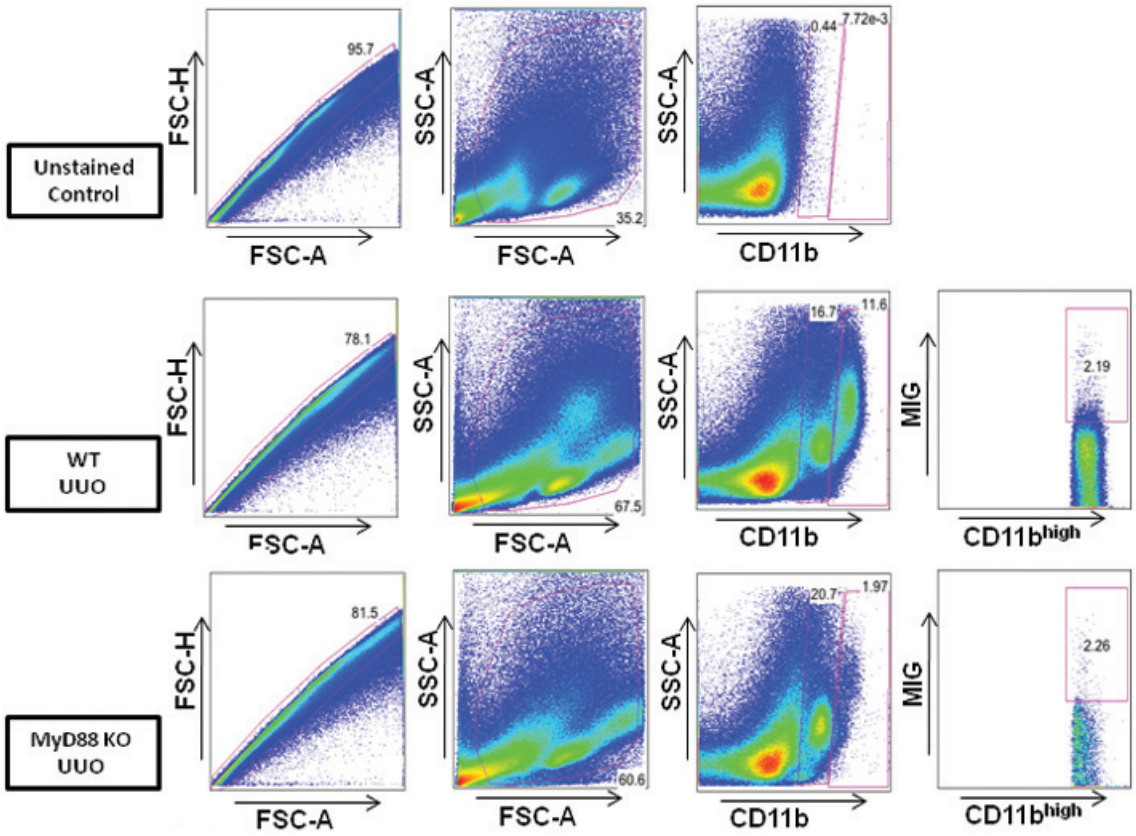
Supplemental Data

MyD88 Signaling Pathway Is Involved in Renal Fibrosis by Favoring a T_H2 Immune Response and Activating Alternative M2 Macrophages

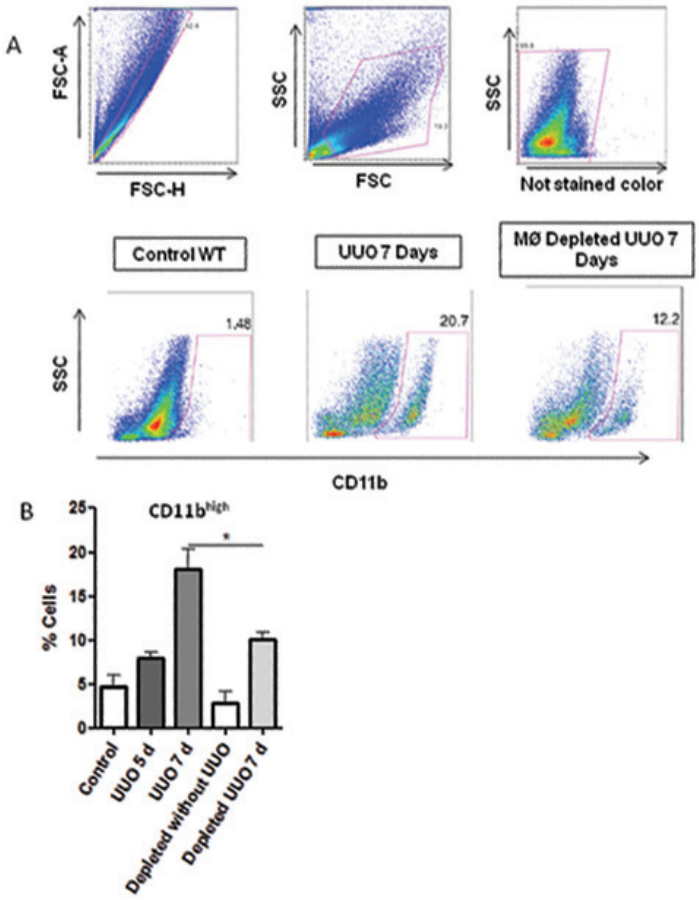
Tarcio Teodoro Braga,[1] Matheus Correa-Costa,[1] Yuri Felipe Souza Guise,[1] Angela Castoldi,[2] Cassiano Donizetti de Oliveira,[2] Meire Ioshie Hyane,[1] Marcos Antonio Cenedeze,[2] Simone Aparecida Teixeira,[3] Marcelo Nicolas Muscara,[3] Katia Regina Perez,[4] Iolanda Midea Cuccovia,[4] Alvaro Pacheco-Silva,[2] Giselle Martins Gonçalves,[1] and Niels Olsen Saraiva Camara[1,2]

Online address: http://www.molmed.org

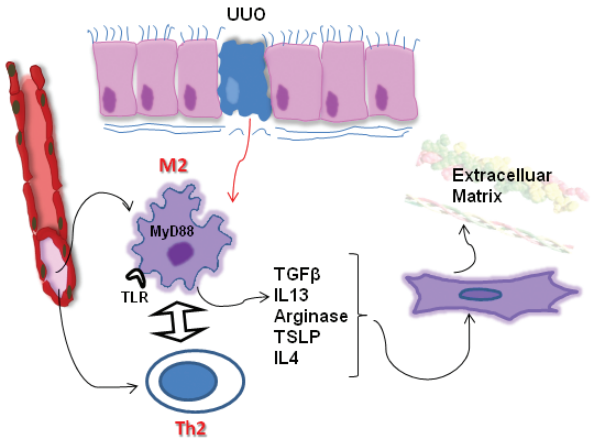
The Feinstein Institute for Medical Research North Shore LIJ



Supplementary Figure S1. Gate strategy of control non-staining and CD11b$^+$ cells infiltrating kidneys at 7 d post-surgery indicated in figure 3 and figure 4. Percentage of CD11b^{high} cells expressing MIG is used as example.



Supplementary Figure S2. Renal CD11b^{high} infiltrating cells in control, after 7 d after UUO in non-depleted and after clodronate liposomes administration (A). Gate strategy and control non-staining are represented in the up panel. Percentage of CD11b^{high} cells (B) at 7 d post-surgery. MØ -Macrophages. *p<0.05.



Supplementary Figure S3. Working model of renal fibrosis formation. Injured signals, released after the death of cells after urinary obstruction flow, lead to the recruitment and to the activation of M2 macrophages as well as to stimulation of a Th2 immune response, in a MyD88-dependent manner. This process leads to extracellular matrix deposition and culminates in fibrosis formation.