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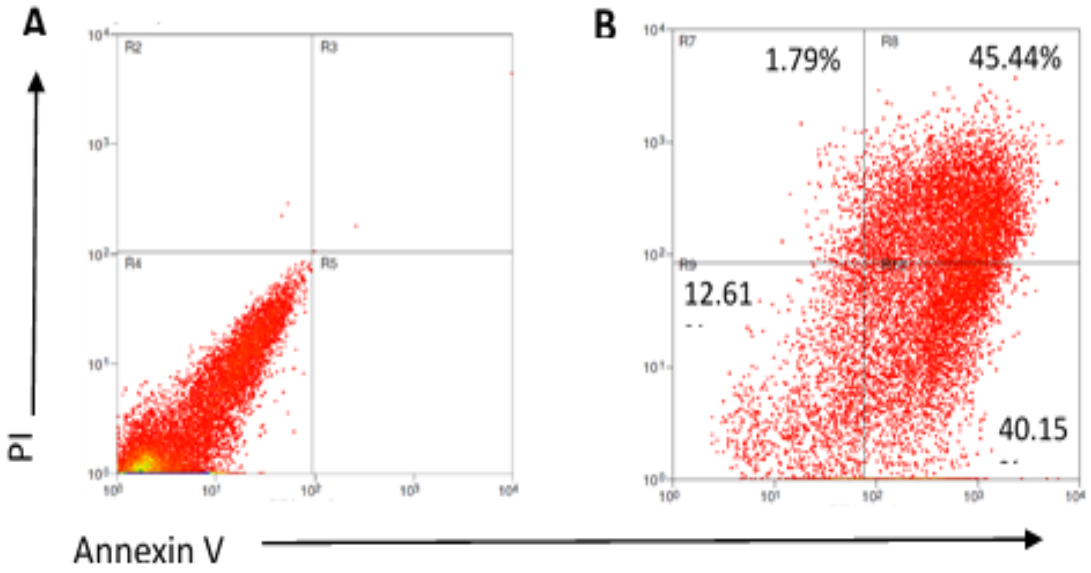
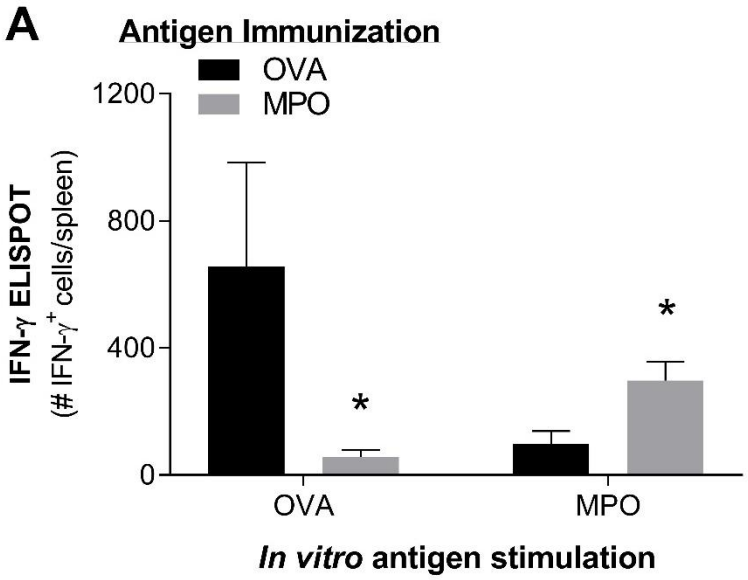
Supplementary Figure Legends

Supplementary Figure 1.



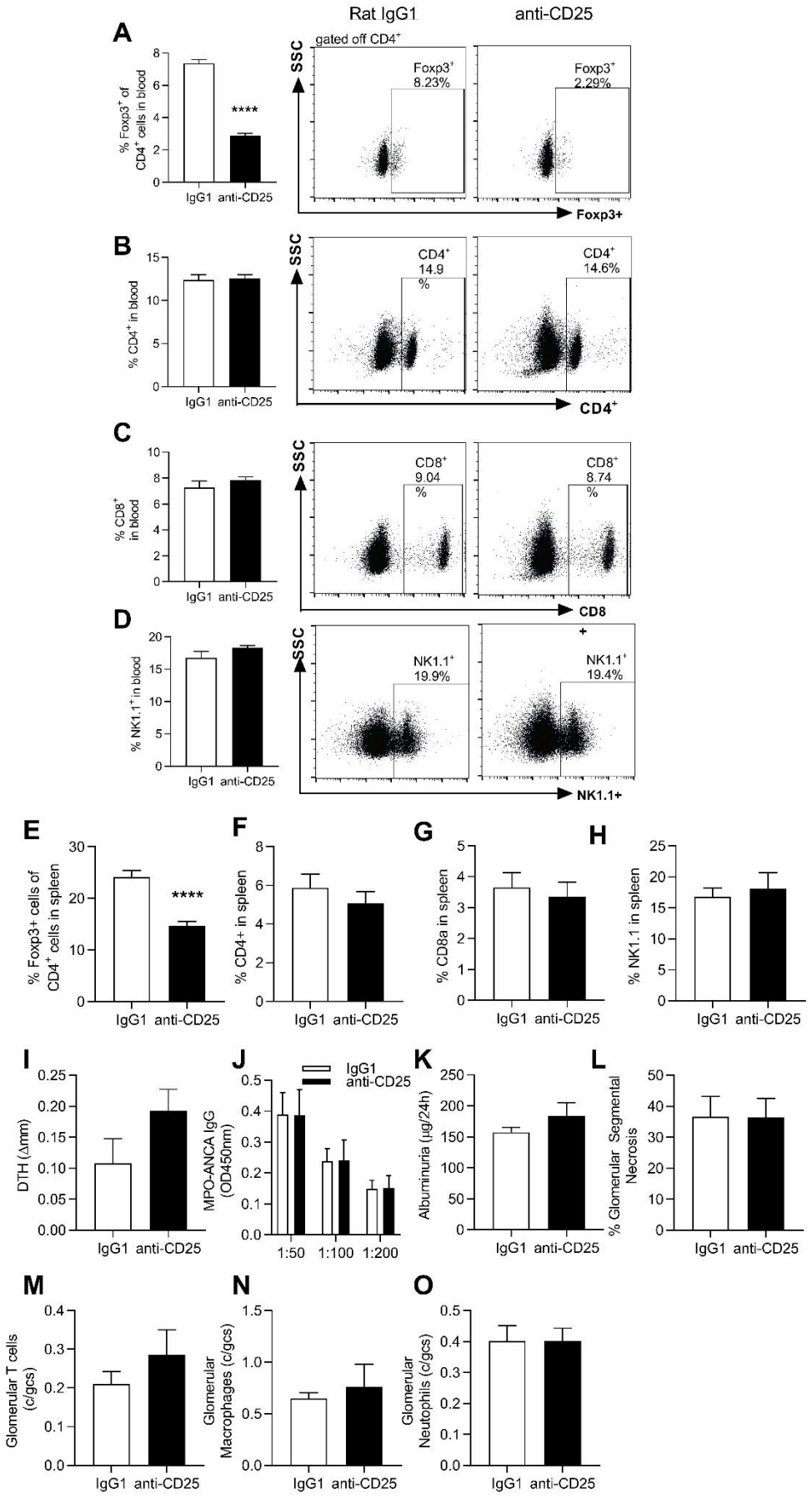
Supplementary 1: ECDI induces >70% splenocyte apoptosis. In order to quantify the induction of apoptosis by treatment with ECDI, donor splenocytes were treated with ECDI for 1hr shaking at 4°C and later incubated at 37°C for 4hrs. Samples were taken of either untreated splenocytes or 4hrs post treatment. Cells were stained with Annexin V and PI to assess apoptosis and cell death. Compared with untreated control, ECDI treatment significantly increased the percentage of apoptotic splenocytes 4hrs post incubation with cells showing two distinctive phenotypes of being early apoptotic ($Annexin^{+}PI^{-}$, 40.15%) and late apoptotic ($Annexin^{+}PI^{+}$, 45.44%).

Supplementary Figure 2.



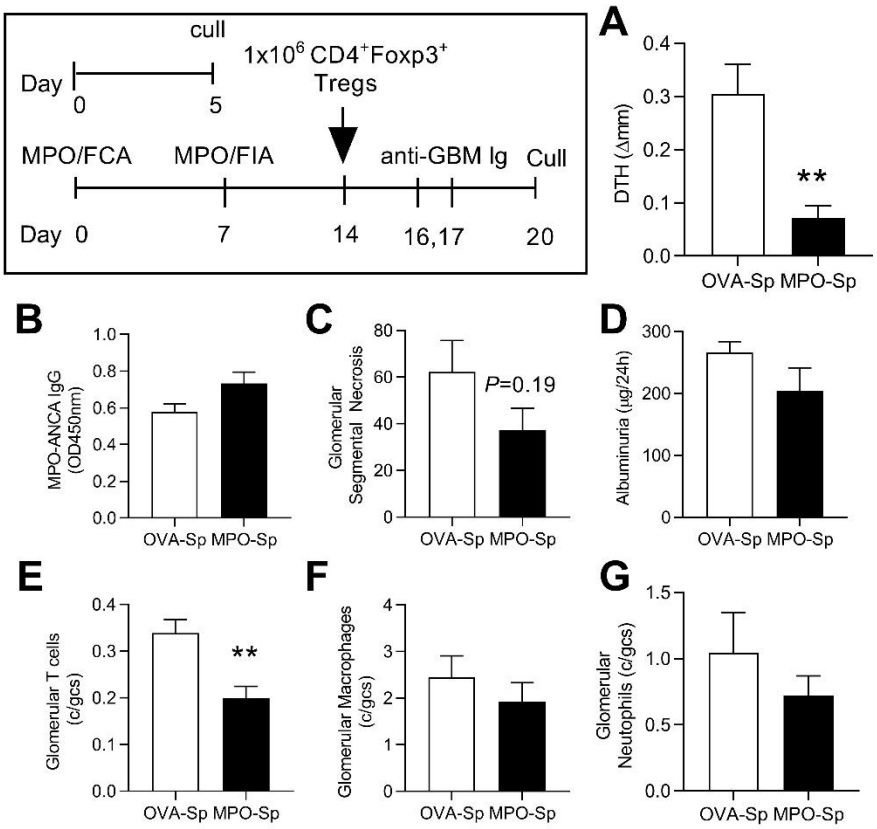
Supplementary 2: Splenic antigen specific recall immune responses. 10 day antigen specific immune responses were measured from spleens of WT mice immunized with either $OVA_{323-339}$ or $MPO_{409-428}$ (*n*=8/group). Frequency of anti-MPO or anti-OVA effector responses assessed using IFN-γ ELISPOT. $OVA_{323-339}$ immunized mice made strong IFN-γ responses against OVA and not MPO, while mice immunized with $MPO_{409-428}$ specifically reacted against MPO. Error bars represent mean ± SEM with statistical analysis by Unpaired *t*-test **P<0.05.*

Supplementary Figure 3.



Supplementary 3. Anti-CD25 mAb depletes Tregs and pretreatment of anti-CD25 mAb does not aggravate anti-MPO GN. Seven days post CD25 depletion, blood was profiled to determine the extent of CD25 depletion. Anti-CD25 mAb depleted (*n*=7) only $CD4^+Foxp3^+$ Tregs and did not affect the frequency of $CD4^+$, $CD8^+$ and NK cells (A-D) compared to isotype rat IgG1 control (*n*=6). Sustained Treg depletion was observed in spleens of anti-CD25 mAb treated mice compared to control at termination of experiment (E). No difference in the frequency of splenic $CD4^+$, $CD8^+$ and NK cells was observed between groups (F-H). Pre-treatment of anti-CD25 mAb did not affect the severity of anti-MPO autoimmunity or GN (I-O). Error bars represent mean ± SEM with statistical analysis by Unpaired *t*-test **** *P<0.0001*.

Supplementary Figure 4.



Supplementary 4: Adoptive transfer of $CD4^+Foxp3^+$ Tregs from MPO-Sp treated mice does not transfer tolerance to recipient mice with established anti-MPO autoimmunity. $CD4^+Foxp3^+$ Tregs isolated from MPO-Sp treated mice (*n=6)* and adoptively transferred to recipient mice with anti-MPO autoimmunity was able to attenuate cellular anti-MPO autoimmunity as measured by MPO specific DTH footpad swelling (A) but not humoral anti-MPO IgG titres (B) compared to mice receiving Tregs from OVA-Sp treated mice (*n*=6). No difference between groups was observed in the frequency of glomerular segmental necrosis and albuminuria (C-D). Mice receiving Tregs from MPO-Sp treated mice significantly reduced the numbers of infiltrating glomerular $CD4^+$ T cells but not macrophages and neutrophils compared to mice receiving Tregs from OVA-Sp treated mice (E-G) Error bars represent mean ± SEM with statistical analysis by Unpaired *t*-test ***P<0.01*.