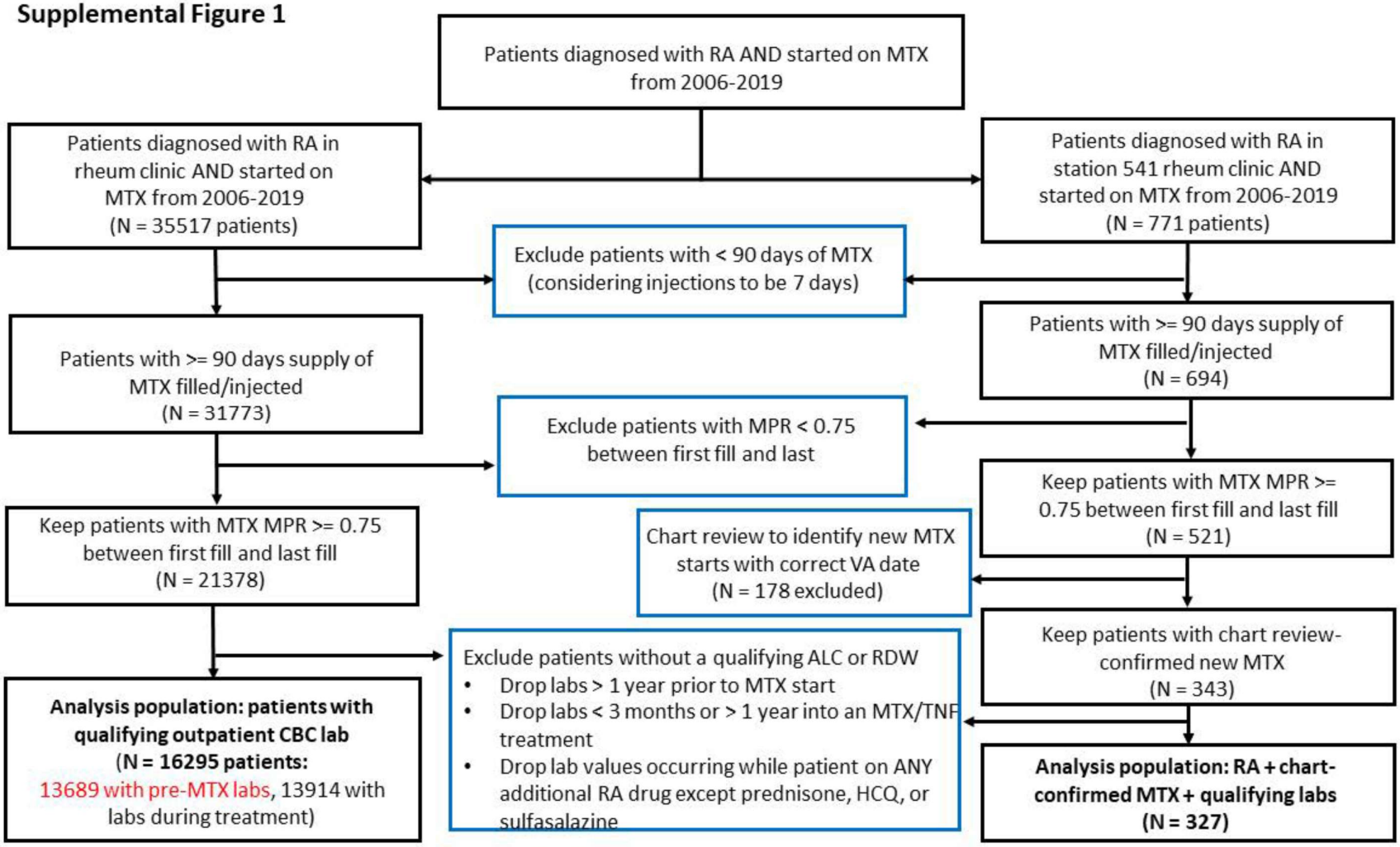
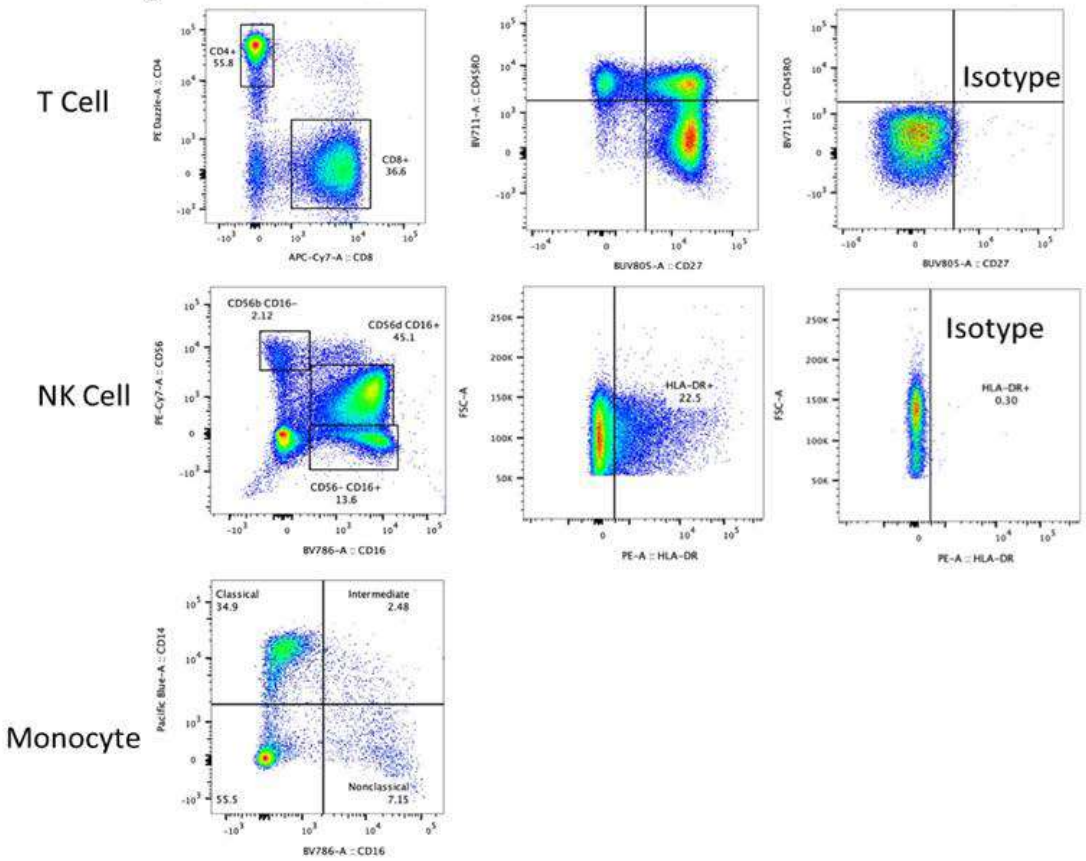
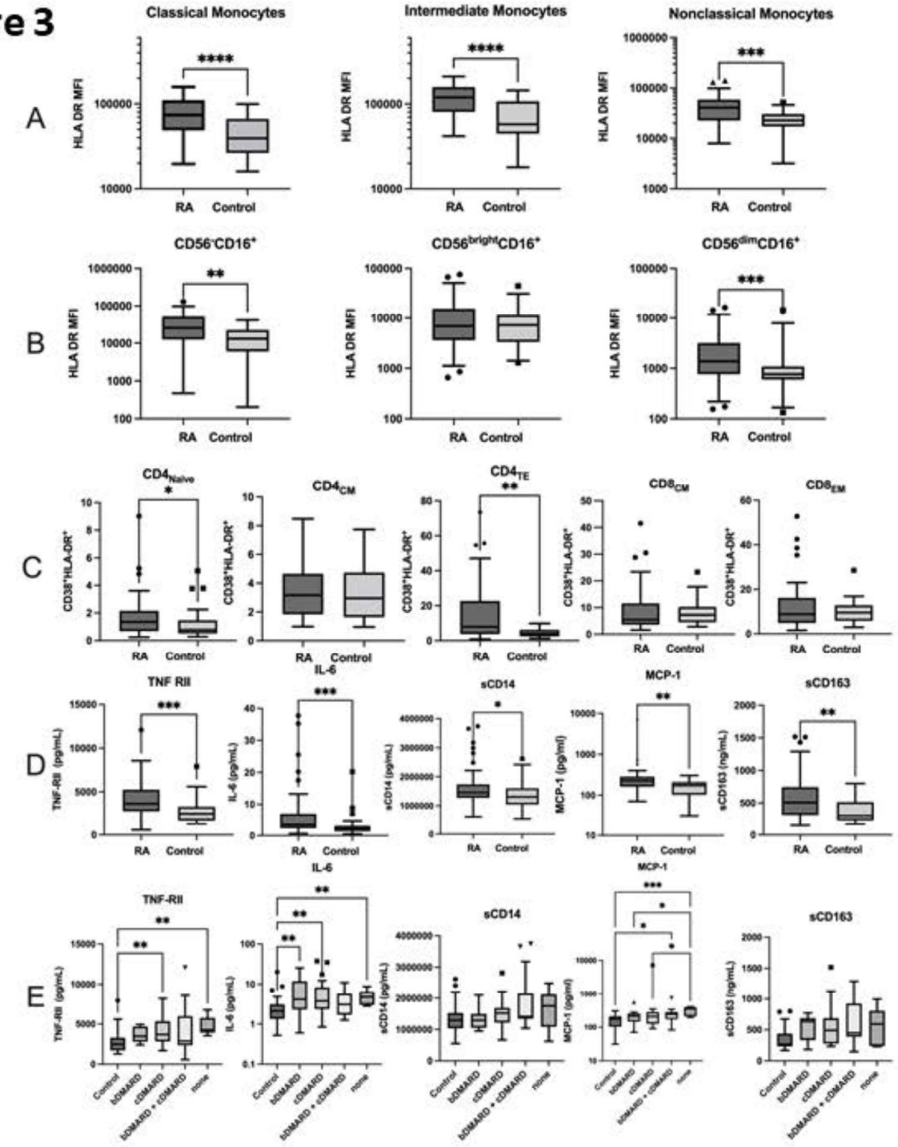
Supplemental Figure 1



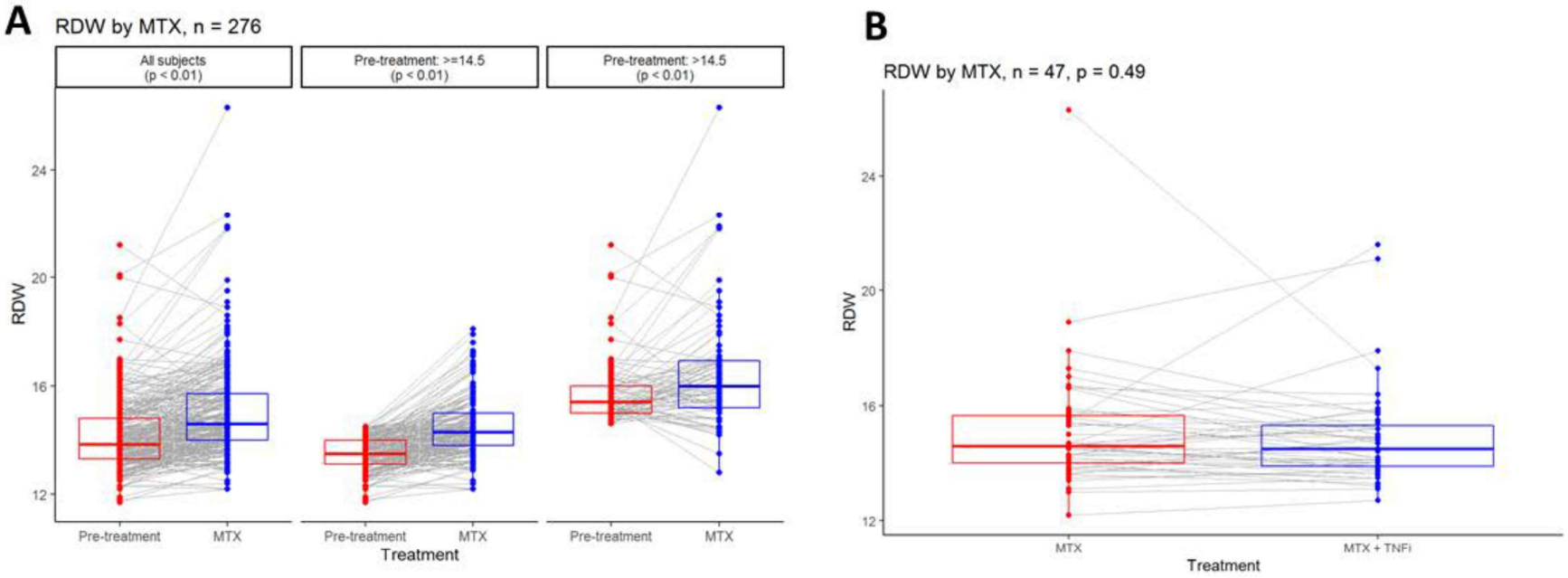
Supplemental Figure 2



Supplemental figure 3



Supplemental Figure 4



SUPPLEMENTARY MATERIALS

Supplemental Figure 1. Local and National RA Cohorts: Consort diagram of local and national RA cohort inclusion/exclusion criteria and methodology.

Supplemental Figure 2: Flow cytometry gating strategy. Peripheral blood mononuclear cells (PBMCs) were labeled with anti-CD14-Pacific Blue, CD16-BV786, HLA-DR-PE, CD142-FITC, CD3-Per-CP, CD56-PE/CY7, CD19-AF700, CD38-APC, CD86-BV395, CD4-PE-Dazzle-594, CD8-APC-CY7, CD80-BV605, CD27-BUV805, CD45RO-BV711, Live-Dead-BV510. Flow cytometric analysis was performed on a BD LSRFortessa (BD Biosciences, San Jose, CA). Compensation was performed using single antibody labeled compensation beads; Live/Dead^{TM} Fixable Aqua Dead cell stain-labeled Amine Reactive compensation beads (Life Technologies Corporation, Eugene, Oregon) and BD^{TM} CompBeads (BD Biosciences, San Jose, CA) for cell-surface marker antibodies and analyzed with FACS DIVA software on the BD LSRFortessa Population-based gating strategy was used to determine live and singlets cells, $CD3^{+}CD19^{-}$T cells were divided into $CD4^{+}$ and $CD8^{+}$ subsets and thereafter $CD4^{+}$ T cell subsets were defined by CD27 and CD45RO expression: naïve ($CD27^{+}CD45RO^{-}$), central memory (CM, $CD27^{+}CD45RO^{+}$), effector memory (EM, $CD27^{-}CD45RO^{+}$), and terminal effector (TE, $CD27^{-}CD45RO^{-}$. All T cell subsets were further defined for expression of CD38 and/or HLA-DR based on isotype gating. $CD3^{-}CD19^{+}$ B cells were further defined for CD80 and CD86 expression based on isotype gating. $CD3^{-}CD19^{-}$ cells were population gated into $CD14^{-}$ cells. The $CD3^{-}CD19^{-}CD14^{-}$ NK cells were further divided by population-based gating into $CD56^{bright}CD16^{-}$, $CD56^{dim}CD16^{+}$, and $CD56^{-}CD16^{+}$ subsets. These subsets were further defined for their expression of HLA-DR based on isotype gating. $CD3^{-}CD19^{-}$ cells were population gated into HLA DR+ cells. The $CD3^{-}CD19^{-}HLA\text{-}DR^{+}$ cells were further characterized as monocyte subsets based on population based gating of $CD14^{+}CD16^{-}$ (classical monocytes), $CD14^{+}CD16^{+}$ (intermediate monocytes), and $CD14^{-}CD16^{+}$ (non-classical monocytes). These subsets were defined by their

expression of HLA DR MFI and CD142 (tissue factor), CD80, and CD86 based on isotype gating.

Shown a Rheumatoid Arthritis donor sample for CD3+CD16- T cells utilizing population gating for CD4+ and CD8+ distinction and isotype gating for CD4+ and CD8+ T cell subsets. CD3-CD19-CD14- NK cell subsets using population gating for subsets and isotype gating for HLA-DR expression. CD3-CD19-HLADR+ Monocyte subsets done with population gating.

Supplemental figure 3. Soluble and cellular markers of inflammation are elevated in persons with RA, and in some cases levels vary by RA treatment. Panel A: Monocyte subset (Classical, Intermediate, and Nonclassaical) HLA DR expression as mean fluorescence intensity (MFI). **Panel B**: NK cell subset HLA DR expression (mfi). **Panel C**: CD4 and CD8 cell subset HLADR/CD38 coexpression (%). **Panel D**: soluble markers (TNFR2, IL6, sCD14, MCP1, sCD163) shown for control and RA groups. **Panel E**: soluble marker expression shown by RA treatment status.

Supplemental Figure 4. RDW increases after start of MTX therapy in both those with low and high baseline RDW, while RDW remains similar upon addition of TNF blocker therapy to those on MTX. 4A: RDW before and after start of MTX therapy. 4B: RDW before and after TNF blocker therapy in patients on MTX.