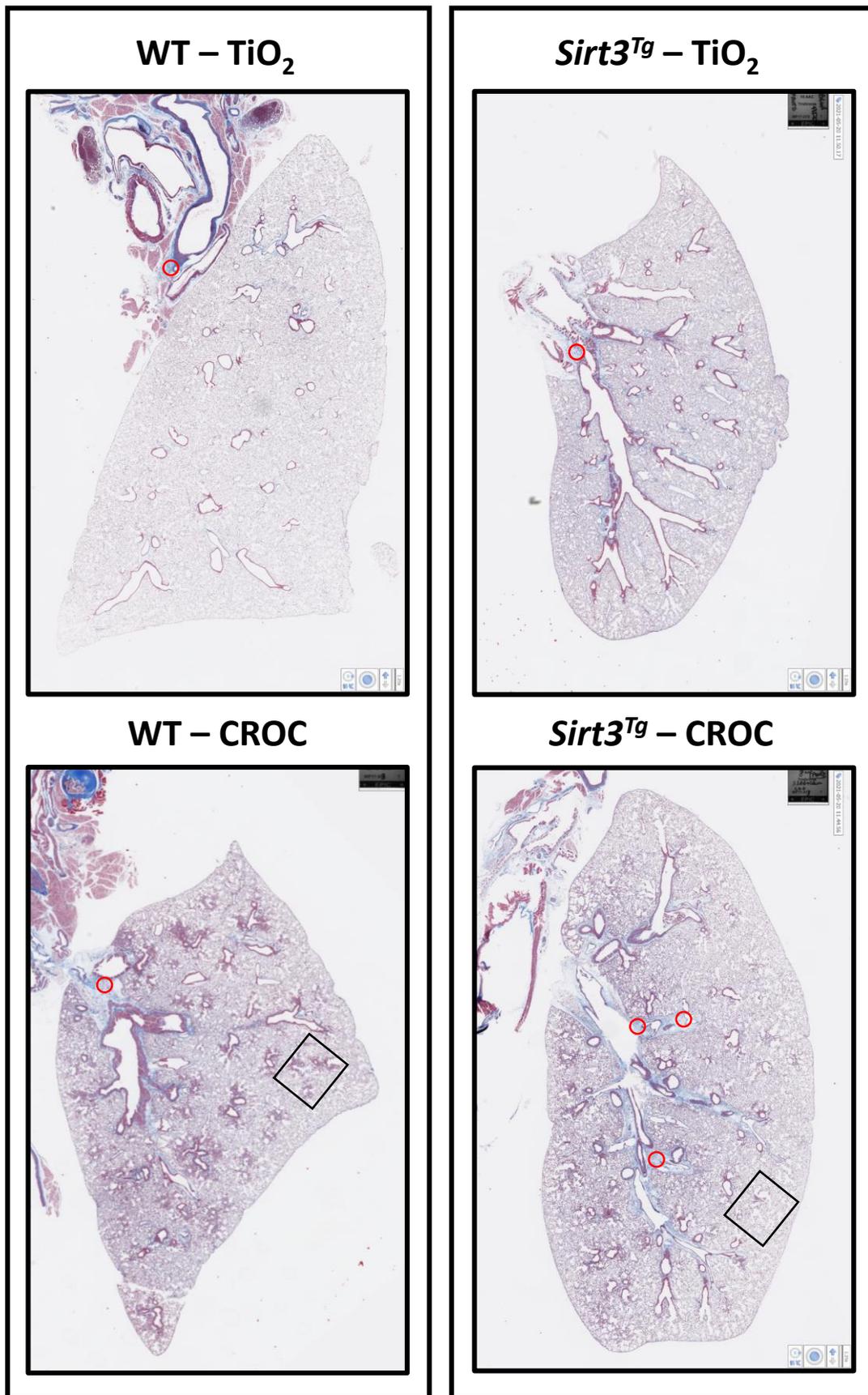
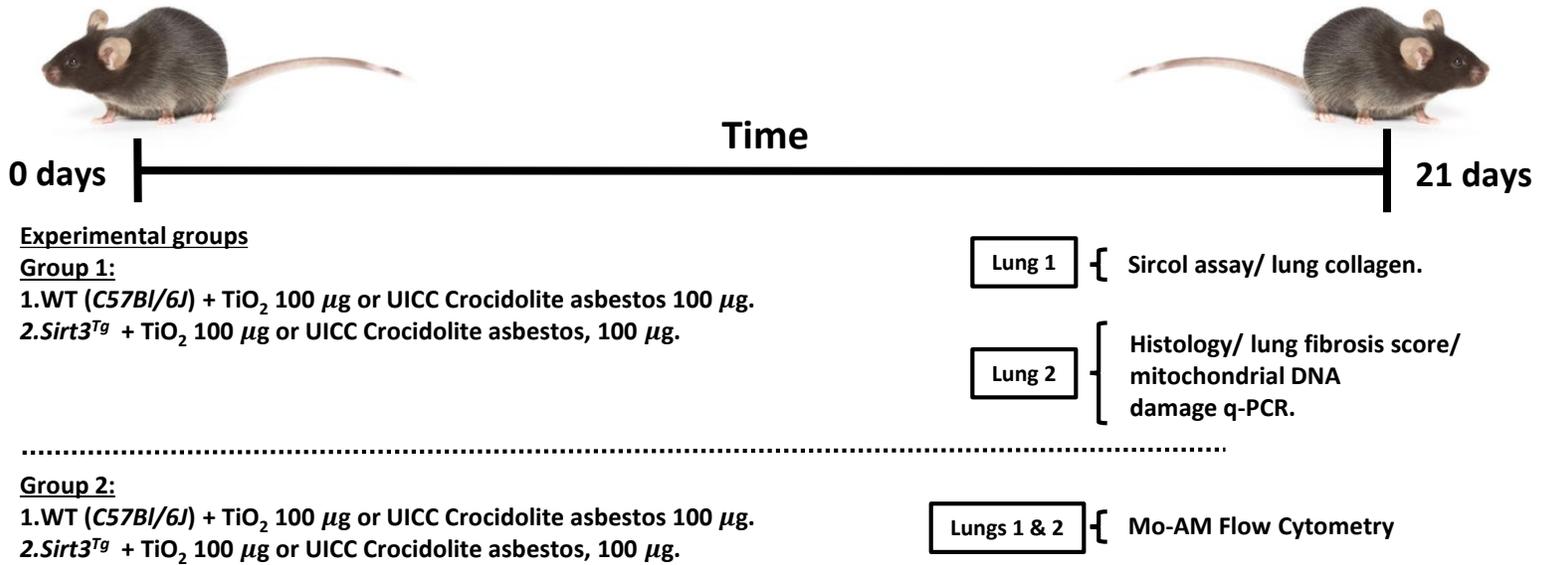


Supplementary Figure S1. *Sirt3^{Tg}* mice are protected against asbestos-induced pulmonary fibrosis. Whole lung images. Fibrotic bronchoalveolar duct junction area in Figure 1 (Black square). Airway wall collagen (red circles). Magnification = 1.25X

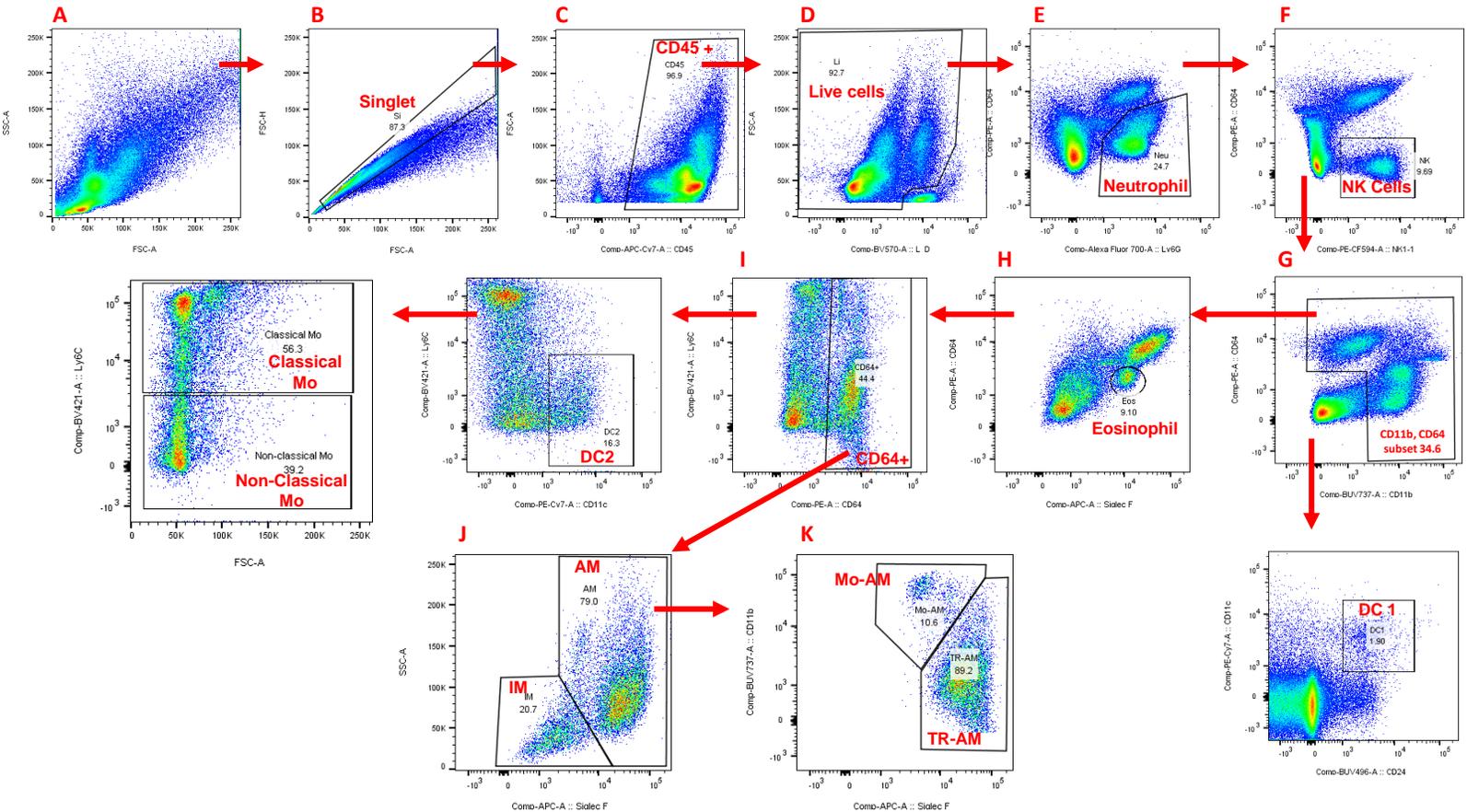


Supplementary Figure S2.

(A) Experimental Design.



(B) Flow gating strategy. Twenty-one days after exposure to 100ug TiO₂ or Crocidolite asbestos, lungs were harvested from WT (C57Bl/6J) or Sirt3Tg mice, digested the lung enzymatically to generate a single-cell suspension, and stained with the panel of antibodies described in 4.13 of the Methods section of this manuscript. After the exclusion of doublets and debris (Panel B), live immune cells were identified by CD45 and live/dead staining (Panel C and D). A sequential gating strategy was used to identify populations expressing specific markers: neutrophils (Ly6G⁺), NK cells (NK1.4⁺), eosinophils (CD11b⁺ CD64⁻ Siglec F⁺), interstitial macrophages (CD64⁺ Siglec F⁻), and alveolar macrophages (CD64⁺ Siglec F⁺) (Panel E-J). Alveolar macrophages (AMs) were categorized as tissue-resident AMs (TR-AM) and monocyte-derived AMs (Mo-AM), based on the expression levels of CD11b and Siglec F: TR-AM (Siglec F^{high} CD11b^{low}) and Mo-AM (Siglec F^{low} CD11b^{high}) (Panel K). Please see refs 41 and 42 for details.



Supplementary Figure S3. *Sirt3* protein expression is silenced MLE cells treated with *Sirt3* siRNA. SIRT3 ~ 28 kDa; GAPDH ~37kDa (Panel A), N=3.

