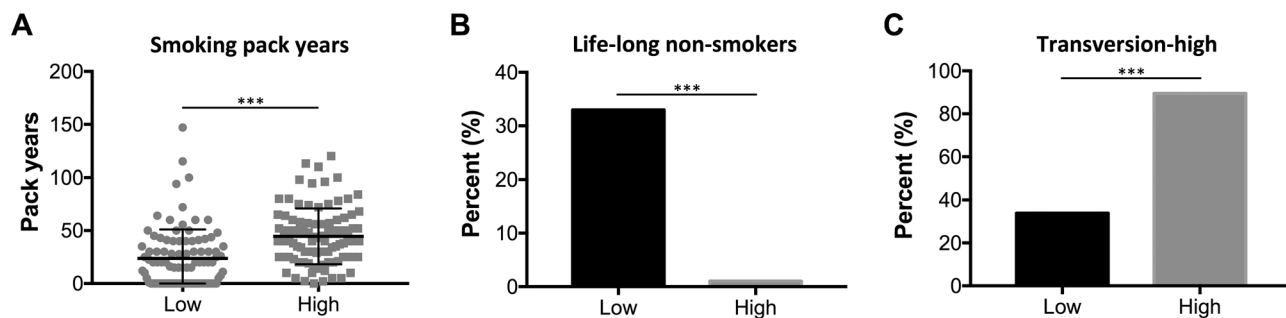
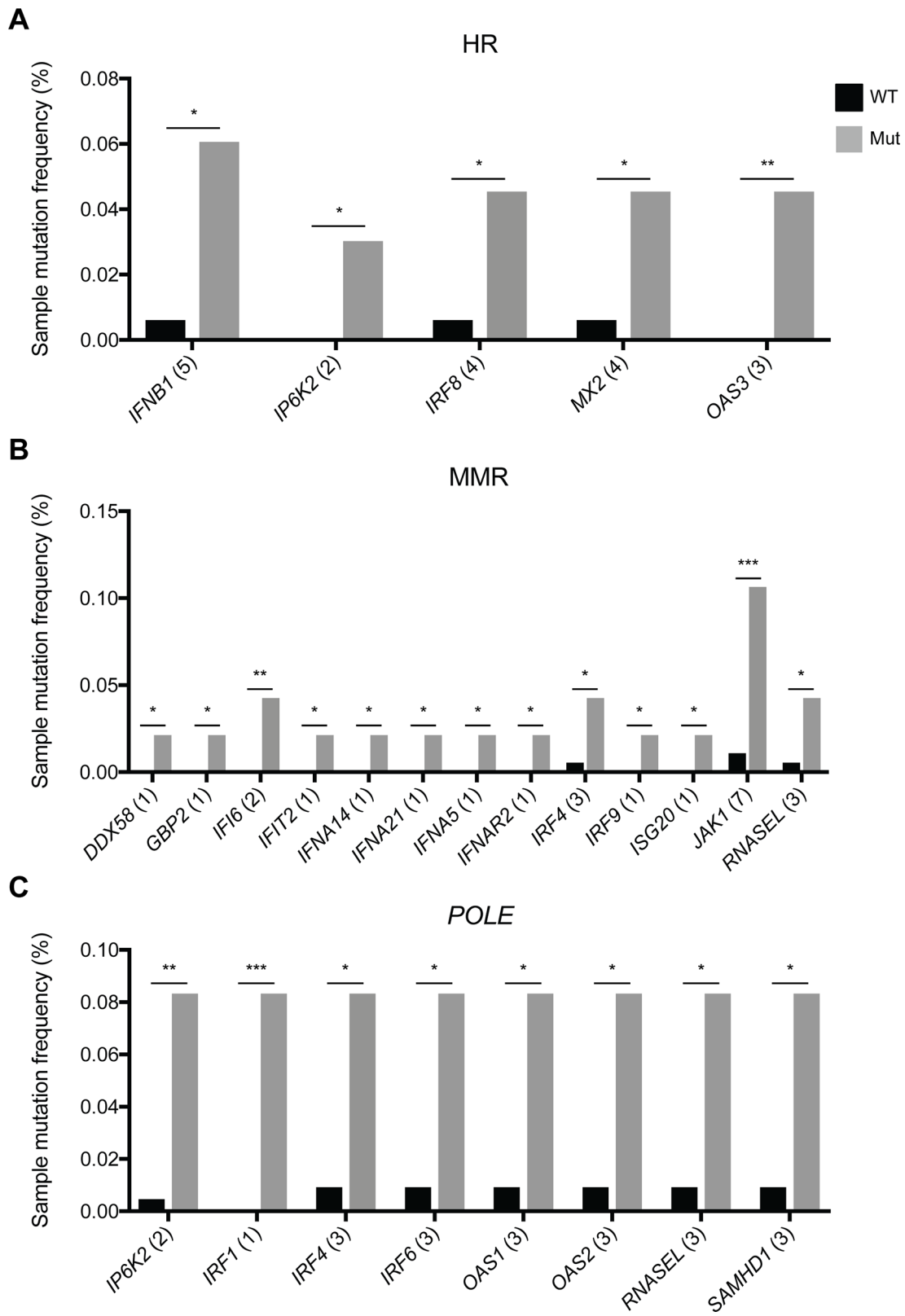


Mutations in DNA repair genes are associated with increased neo-antigen load and activated T cell infiltration in lung adenocarcinoma

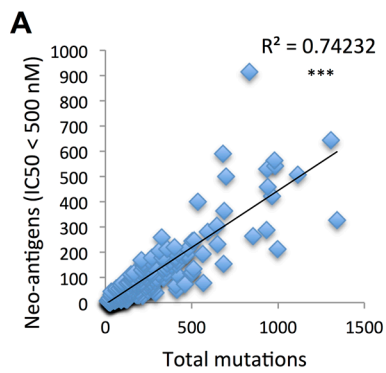
SUPPLEMENTARY MATERIALS



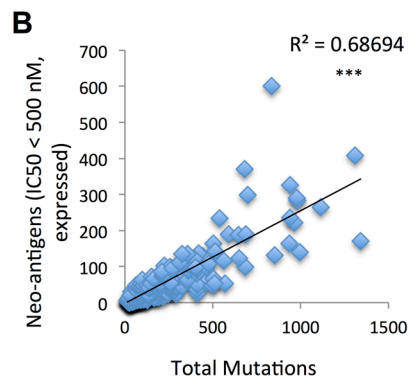
Supplementary Figure 1: Tumor mutational burden is increased in smokers and contains increased transversions. High and low total mutational burden tumors classified by (A) patient smoking pack years (results are presented as mean \pm SEM), (B) percent of life-long non-smokers, and (C) percent of transversion-high tumors. *** $P < 0.001$.



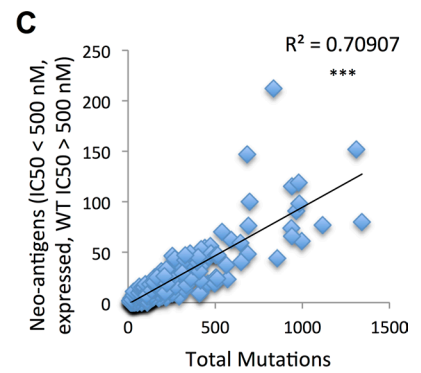
Supplementary Figure 2: DNA repair mutations are linked to increased frequencies of type I IFN gene mutations. The mutation frequency of all type I IFN signaling pathway genes (GO: 0060337) with mutation data from the TCGA dataset were analyzed in samples with WT or mutated (A) HR genes, (B) MMR genes, or (C) *POLE*. All genes with significantly altered mutation frequencies are displayed. Numbers in parenthesis indicate the number of samples which contained a mutation in each gene. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.



Median neo-antigen count = 59



Median neo-antigen count = 38



Median neo-antigen count = 12

Supplementary Figure 3: Filters used in predicting neo-antigen load. Correlation between neo-antigen load and total mutation count as defined by (A) predicted MHC binding affinity (IC₅₀ < 500 nM), (B) predicted MHC binding affinity (IC₅₀ < 500 nM) and positive gene expression, and (C) predicted MHC binding affinity (IC₅₀ < 500 nM), positive gene expression, and non-mutated parental peptide predicted MHC binding affinity (IC₅₀ > 500 nM). *** $P < 0.001$.