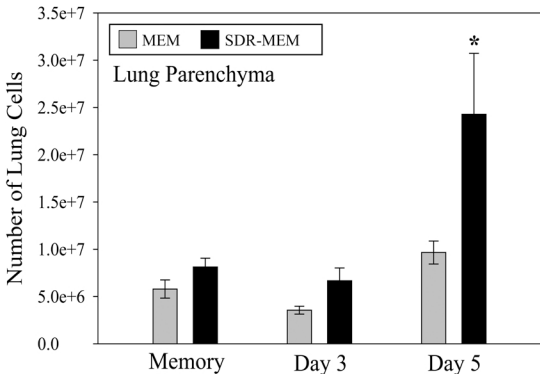
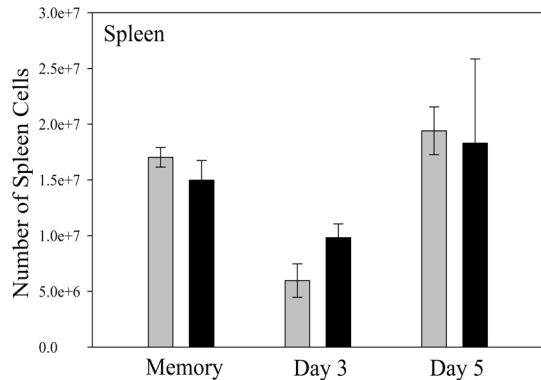
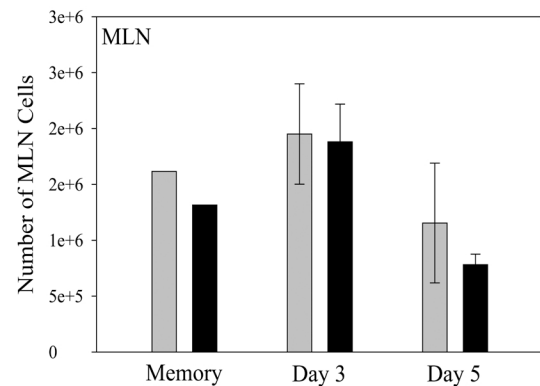


Supplementary Figure 1. SDR increased the size of the lung CD8⁺ T cell population during A/PR/8/34 re-challenge. Mice were subjected to SDR and primary influenza infection as described. Flow cytometry data for a resting memory timepoint and days 3 and 5 after re-infection with A/PR/8/34 virus is shown. In A-C, bars represent group mean +/- SEM; n=4-6 per group. In D and the memory timepoint of C, the bars represent the measured value for the pooled sample by group each day. ANOVA indicated group-wise differences in the lung parenchyma (A) by stress, day post-infection, and stress*day, all p<0.05. No differences were detected in the spleen or MLN samples.*SDR>MEM, p<0.05 by individual t-test.

Supplementary Figure 2. SDR altered cellularity and function of lung parenchymal cells during resting memory after X-31 priming. (A) Mouse lungs were sampled 2-3 months following resolution of a primary X-31 infection. When lung CD8⁺T cells were stimulated ex-vivo with NP366-74 peptide and stained for intra-cellular IFN γ production, there was a significant increase in the number of IFN γ -producing NP₃₆₆₋₇₄ cells isolated from SDR-MEM lungs when compared with MEM lung cells during resting memory, but not during A/PR/8 challenge. (B) Lung gene expression was assessed using semi-quantitative real-time PCR during resting memory and challenge infection with A/PR/8/34 influenza virus. Bars indicate group mean \pm SEM. Statistical comparisons were done using non-parametric 95% confidence intervals.

A.**B.****C.****D.**