

Figure S1: Nuclear accumulation and recruitment of AID-GFP to DNA damage sites

A) Representative false-color images of AID-GFP accumulation in the nucleus after laser microirradiation in HeLaKyoto cells. **B)** Quantification of AID accumulation in the nucleus. **C)** Representative images of AID-GFP recruitment to laser-induced DNA damage sites in the presence of the nuclear export inhibitor LMB (20 ng/ml, 6 h pre-treatment). **D)** Quantification of the results shown in C. Data of 44 (B) and 26 (D) cells from two (D) or three (B) independent experiments are shown as mean \pm SEM. Microirradiation was performed using a spinning disk microscope. Scale bar, 5 μ m. RFU = Relative fluorescence units.

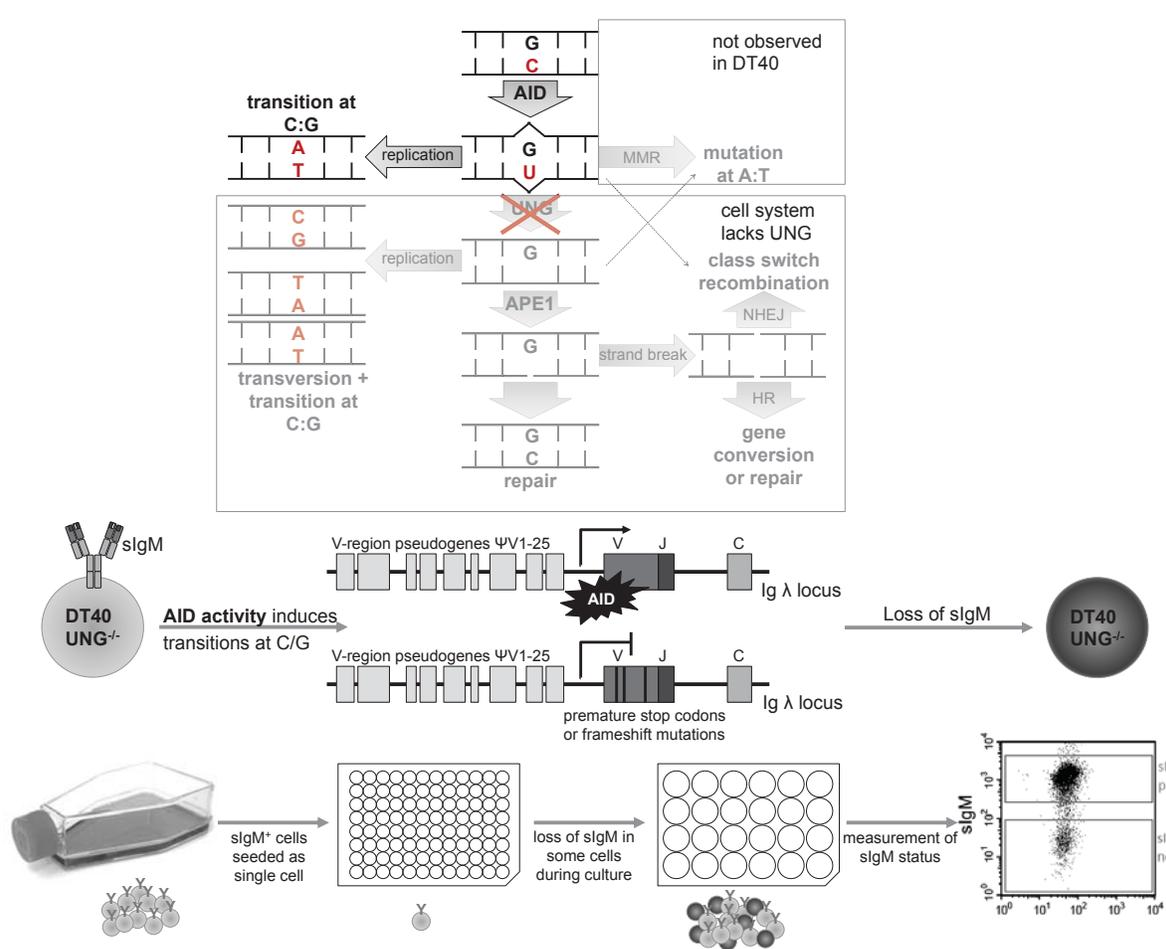
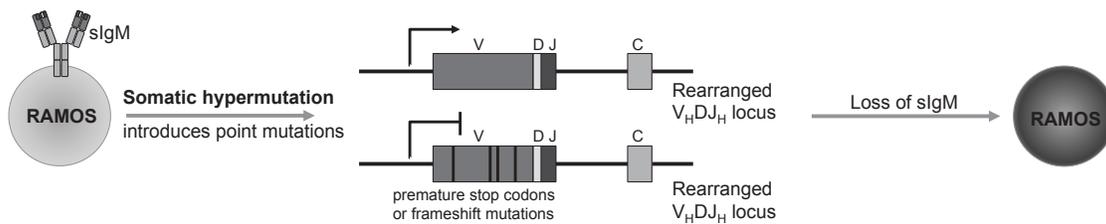
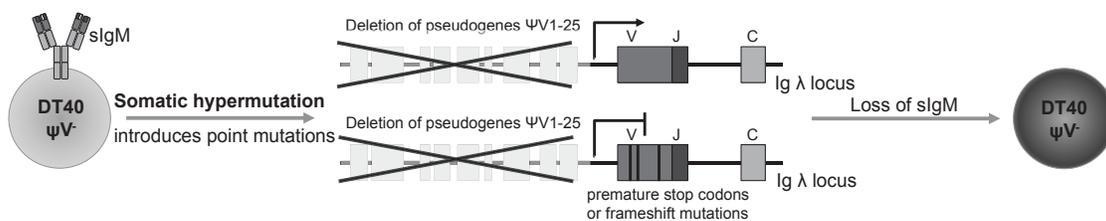
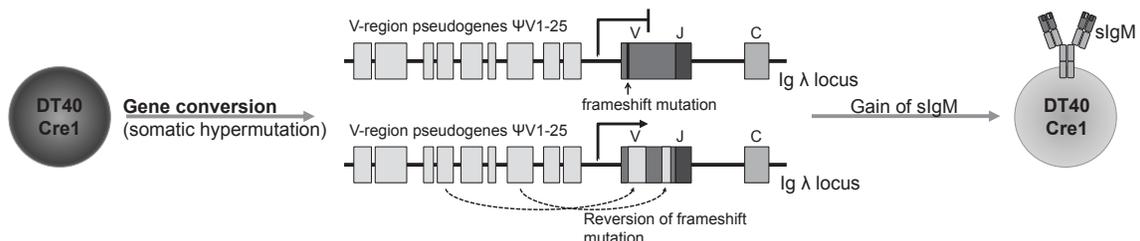
A**B****C****D**

Figure S2: Systems for AID activity in DT40 and Ig diversification in RAMOS or DT40

A) System for the analysis of AID activity in DT40UNG^{-/-} cells. Inactivation of UNG blocks Ig gene conversion and only allows transition mutations in Ig genes (due to the lack of an A:T mutator in DT40 cells), which are indicative of AID activity. Some of these mutations lead to surface Ig loss, which can be quantified by FACS measurements of multiple individual single cell clones that were initially slg⁺ and lose slg upon culture. **B)** System for measurement of somatic hypermutation in RAMOS. Mutations in Ig genes may lead to slg loss that was quantified as described in A). **C)** System for measurement of hypermutation in DT40 ψ V⁻ cells. Deletion of the pseudogenes required for Ig gene conversion leads to a switch to somatic hypermutation. **D)** System for measurement of Ig gene conversion in DT40Cre1 cells. Repair of a frameshift mutation in the original cells by AID-induced homologous recombination with upstream pseudogenes leads to slg gain.

