Supporting Information

Kracker et al. 10.1073/pnas.1012591108

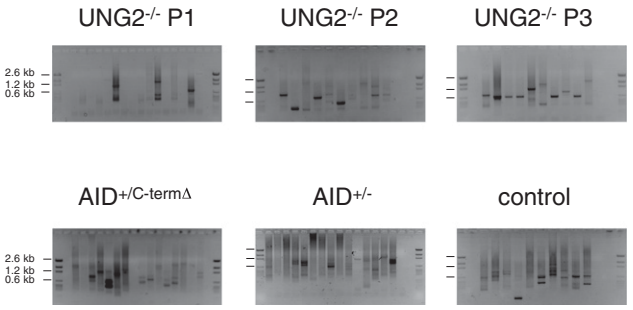

Fig. S1. PCR amplification of Sμ–Sα fragments. Shown are representative agarose gels for all UNG2$^{-/-}$ patients (with identification numbers corresponding to those in ref. 1) and from one patient each from the AID$^{+/-}$, AID$^{+/C\text{-term}\Delta}$, and control groups. Molecular weight marker (pGEM; Promega) was loaded on the first lane and last lane of each gel. The sizes of 2.6, 1.2, and 0.6 kb are indicated on the left side of each gel.

1. Imai K, et al. (2003) Human uracil-DNA glycosylase deficiency associated with profoundly impaired immunoglobulin class-switch recombination. *Nat Immunol* 4:1023–1028.

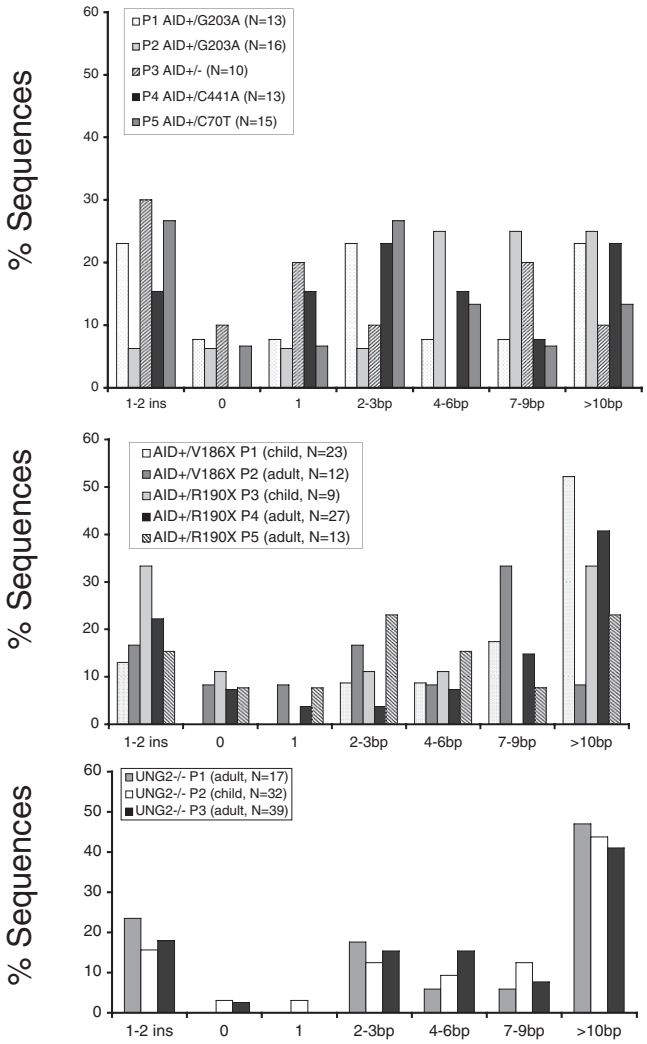

Fig. S2. Characterization of Sμ–Sα recombination junctions per individual. The bar graphs show perfectly matched microhomology at junctions of PCR products cloned from the indicated patients. (*Top*) Sμ–Sα junctions of the $AID^{+/-}$ patients. The "–" indicates that in this patient, the complete coding region of the *AICDA* gene is deleted. (*Middle*) Sμ–Sα junctions of the $AID^{+/C\text{-}term\Delta}$ patients. (*Bottom*) Sμ–Sα junctions of the $UNG2^{-/-}$ patients. $UNG2^{-/-}$ patient identification numbers correspond to those in ref. 1.