Supplementary Figure 5. AID induction and *Trp53* mutation frequencies in wild type mice after *H. pylori* infection. (a, b) Wild-type C57BL/6J mice were challenged with $1.5 \times 10^7$ cfu *cagPAI*-positive *H. pylori* (TN2GF4) as described previously. AID expression and *Trp53* mutation frequencies in stomach were determined 1, 2, and 40 weeks after *H. pylori* infection. (a) Lack of AID immunoreactivity in mouse gastric mucosa without *H. pylori* infection (left panel) and AID immunostaining appeared in mouse gastric mucosa 1, 2 and 40 weeks after *H. pylori* oral infection (right panel) are shown. Scale bar, 200µm. (b) The mutation frequencies in the *Trp53* gene between exons 2 and 11 derived from gastric specimens of mice without *H. pylori* infection (control) and those 1, 2 and 40 weeks after *H. pylori* infection are shown. (c) Genomic DNA was extracted from wild-type (control) and 52-week-old AID Tg mouse stomach. *Trp53* mutation frequencies in exons 1, 7 and 8 in each tissue sample are shown. (b, c) Number of mutated clones per total clones examined and number of mutated bases per total base pairs sequenced are shown.

**Preparation of anti-AID monoclonal antibody.** A synthetic peptide from carboxy-terminal 14 residues of mouse AID with an amino-terminal extra cysteine (CEVDDLDRDAFRMLGF) was conjugated with keyhole limpet hemocyanin and used for immunization of rats. After five immunizations with two-week intervals, bone marrow cells were collected and fused with P3U1 mouse myeloma cells by conventional procedure. Fused cells were selected by cultivation in HAT medium. Five clones producing AID-specific antibodies were obtained after screening by enzyme-linked immunosorbent assay and western blot. Two clones that yielded indistinguishably good results were named as MAID-1 and MAID-2, the former of which was used in this study. MAID-1 hybridoma cells were injected into peritoneal cavity of nude mice. Developed ascites were collected for purification of antibody.