Figure S1: Removal of the uracil by UNG. For optimization of UNG treatment, 5' [γ-^{32}P]-labeled U-substrates (U16G16, D1D1C and D2D2C) were incubated with various amounts of UNG and 1 ng of human recombinant APE1 protein. The endonucleolytic cleavage product was then monitored on a polyacrylamide urea denaturing gel. Complete removal of uracil from the various DNA substrates was achieved using ≥2,9 units of UNG (corresponding to lane 4 for each substrate). Arrows show the full-length substrate; arrowheads show the incision product.

Figure S2. Repair of control random substrate is modulated by BER stoichiometry. Left panel. Radioincorporation experiment showing the time course of repair of the control U16G16 substrate under striatal (st) and cerebellar (cb) repair conditions, with or without 1 mM ATP supplementation. The full-length repaired products (FL) are indicated with bold arrows (➡) and +1nt intermediate products are shown. Right panel. Graph representing repair efficiencies of the control U16G16 susbstrate.

Figure S3. Radioincorporation experiments showing the time course of repair of the control substrate U16G16 when changing the stoichiometries of LIG1 and LIG3. The substrate was incubated with a mixture of BER proteins reflecting the stoichiometry in the cerebellum of HD mice, except that the concentrations of LIG1 and LIG3 were that measured in the striatum (lanes 2), or, conversely, with a mixture of BER proteins reflecting the stoichiometry in the striatum, except that the concentrations of LIG1 and LIG3 were that found in the cerebellum (lanes 4). As controls, the substrate was also incubated with mixtures of BER proteins reflecting the levels in the cerebellum (lanes 1) and striatum (lanes 3).