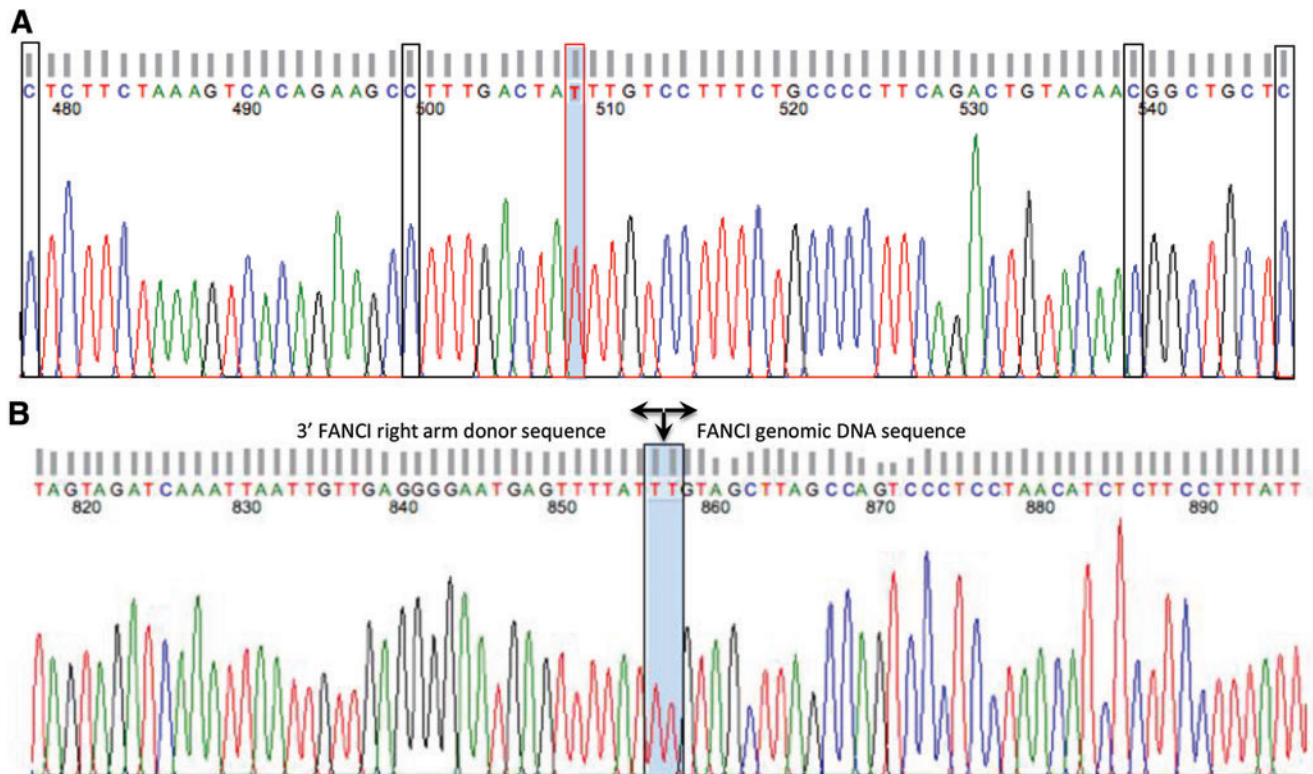
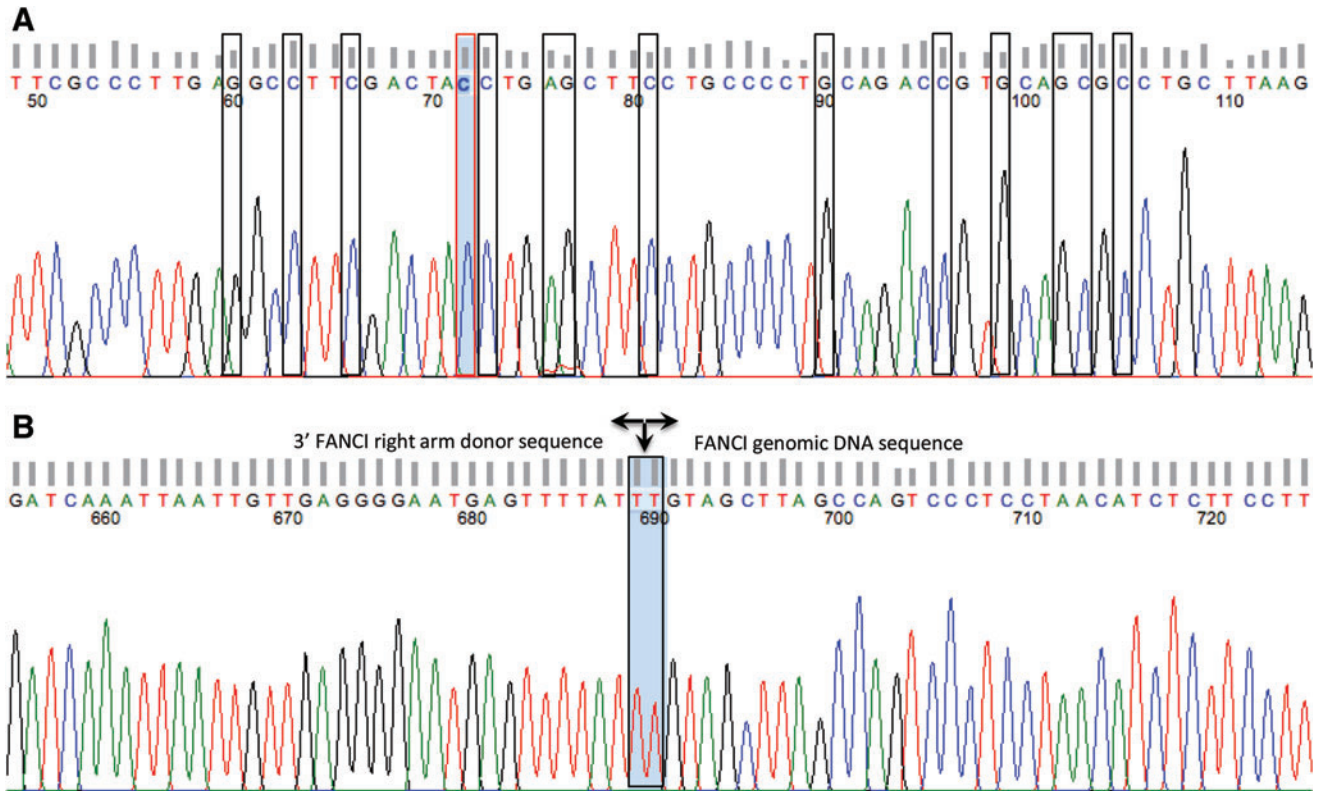


SUPPLEMENTARY FIG. S5. CRISPR/Cas9 mediated homologous recombination. FANCI primary fibroblast cell gene targeting. Cells were targeted with a plasmid HDR template and a bulk population of drug-resistant cells were obtained and HDR was detectable only at the cDNA level. The *shaded letters* indicate the targeted base for reversion/correction. *Boxed* bases show donor-derived silent polymorphisms. CRISPR, clustered, regularly interspaced short palindromic repeats.



SUPPLEMENTARY FIG. S6. FANCI iPSC cell homologous recombination using a puromycin-based donor. PCR detection and Sanger sequence analysis of gene modification. **(A)** iPSC gene correction showing restoration of proper sequence (*shaded base*) and donor-derived polymorphisms (*boxed sequences*). **(B)** Donor and adjacent genomic locus junction is marked by the *shaded sequence* from an “inside-out” PCR with primers within the puromycin gene and the FANCI locus.



SUPPLEMENTARY FIG. S7. Exogenous marker sequence free donor mediated HDR in FANCI iPSC. HDR in FANCI iPSC with selection by mitomycin C. *Shaded letters* show the corrected DNA base. **(A)** Sequences marked by boxes are unique polymorphic bases introduced into the respective donor sequence. Note: in this donor format the 1461 T>A mutation is restored to the proper tyrosine AA sequence by incorporation of a cytosine rather than an thymidine as is shown in Supplementary Fig. 6. **(B)** The *shaded bases* indicate the junction between exogenous donor and endogenous sequence.