

Appendix A. Supplementary Data

DNA polymerase β -dependent cell survival independent of XRCC1 expression

Julie K. Horton, Natalie R. Gassman, Brittany B. Dunigan, Donna F. Stefanick and Samuel H. Wilson.

Laboratory of Structural Biology, NIEHS, National Institutes of Health, Research Triangle Park, NC 27709, USA

Figures S1-S3

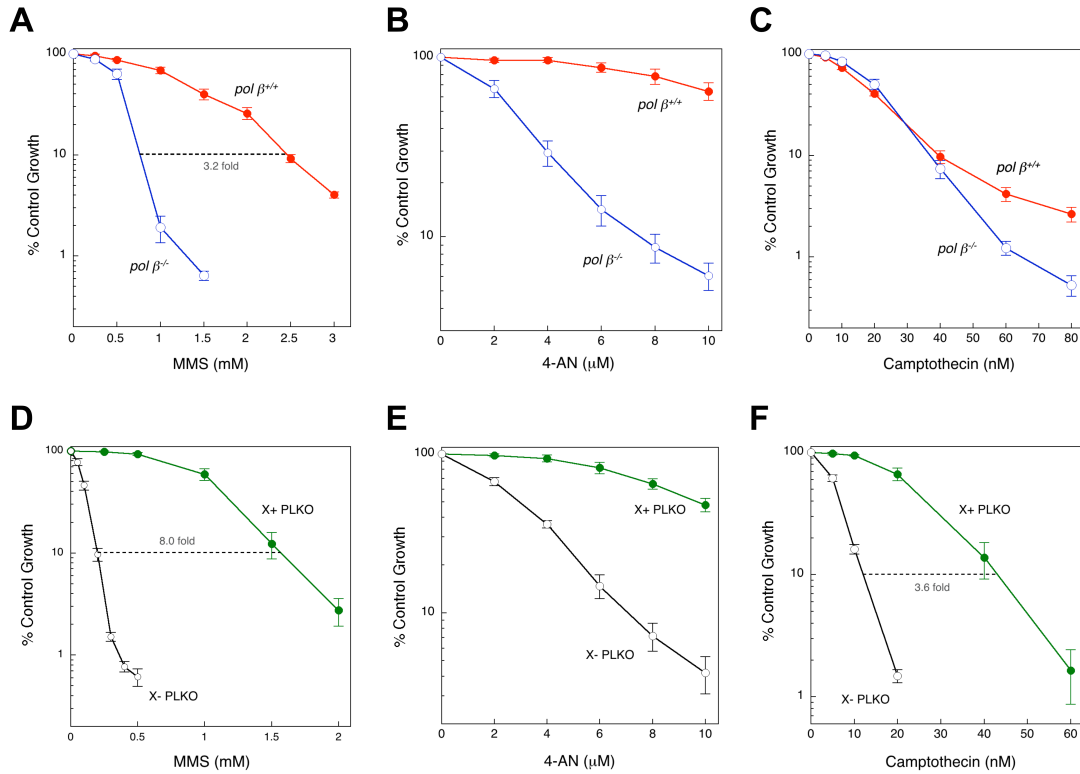


Fig. S1 – Comparison of hypersensitivity phenotypes of *pol β^{-/-}* and *Xrcc1^{-/-}* (X- PLKO) cell lines compared with their respective wild type (*pol β^{+/+}* and X+ PLKO). Pol β cells are shown in panels A-C, and XRCC1 in panels D-F. Cytotoxicity of MMS (Panels A and D), 4-AN (B and E) and camptothecin (C and F). Sensitivity was determined by growth inhibition assays described in Section 2. Plotted are mean ± SEM values obtained from at least 3 independent experiments.

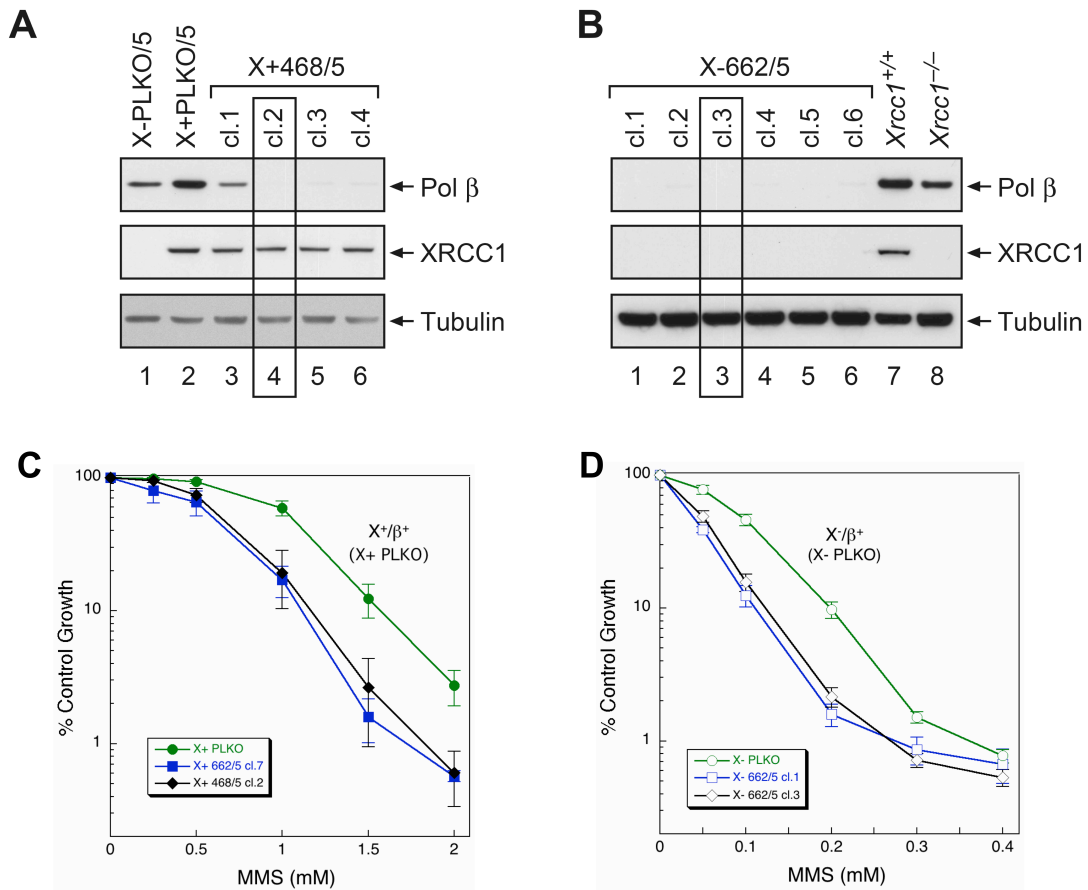


Fig. S2 – Selection of alternate pol β^{kd} clones (A) X+468/5 cl.2 and (B) X-662/5 cl.3 by Western blotting as described in Fig. 1. (C) MMS hypersensitivity of X+/ β^{kd} clones (X+ 662/5 cl.7 and X+ 468/5 cl. 2) compared with control X+ PLKO. (B) MMS hypersensitivity of X-/ β^{kd} clones (X- 662/5 cl.1 and X- 662/5 cl. 3) compared with control X- PLKO. Plotted are mean \pm SEM values obtained from 5 independent experiments.

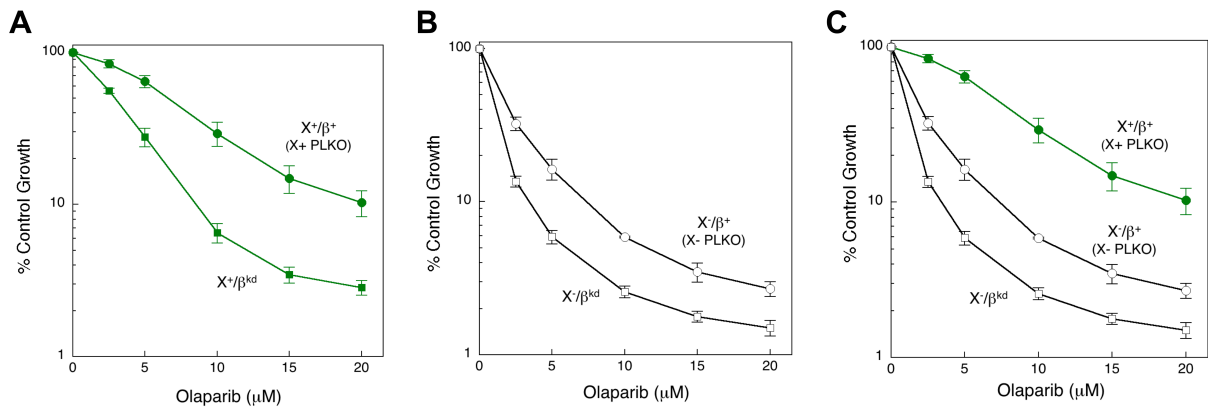


Fig. S3 – Olaparib hypersensitivity of pol β^{kd} clones. Clones selected as shown in Figure 1 were treated continuously with a range of concentrations of olaparib as described in Section 2. (A) Hypersensitivity of X⁺/ β^{kd} compared with control X⁺ PLKO. (B) Hypersensitivity of X⁻/ β^{kd} compared with control X⁻ PLKO. (C) Olaparib hypersensitivity of X⁻ PLKO and X⁻/ β^{kd} compared with X⁺ PLKO. Plotted are mean \pm SEM values obtained from 5 independent experiments.