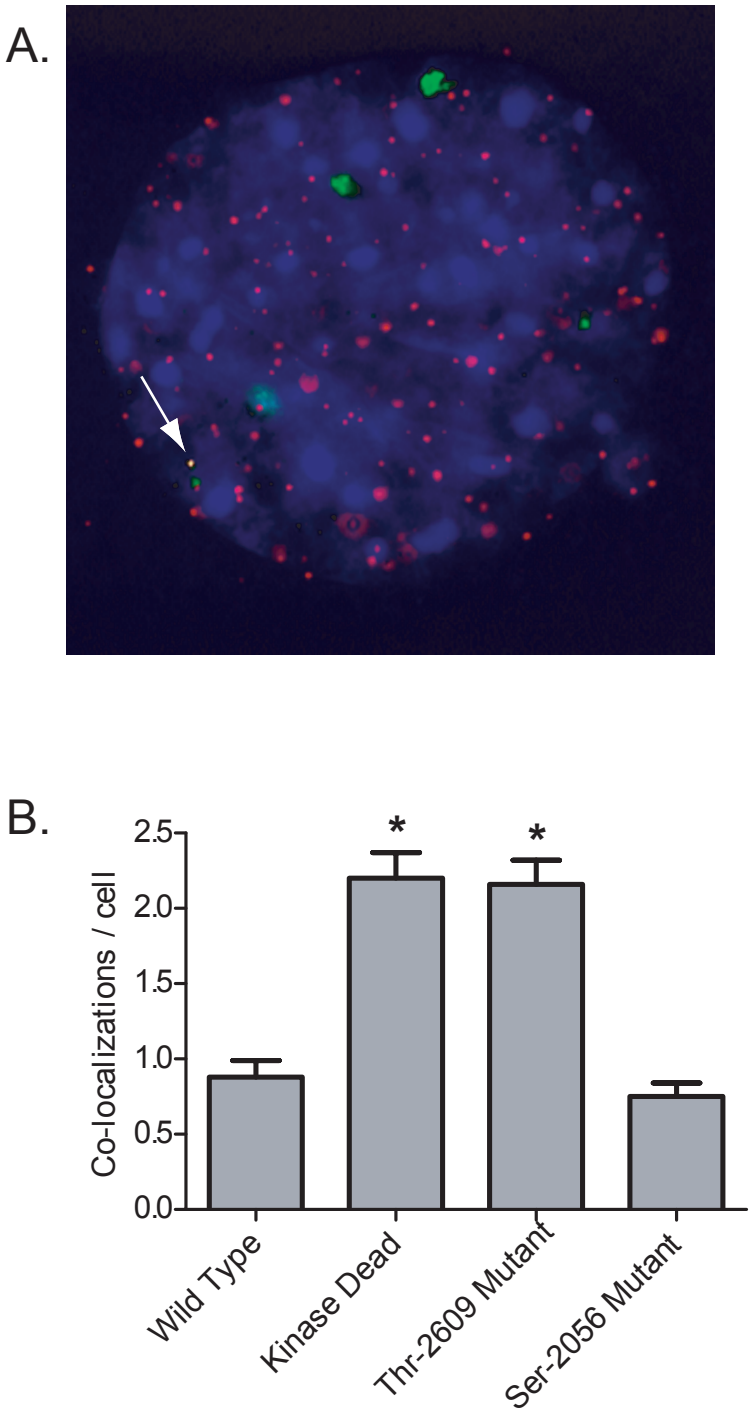

Supplemental Figure 1. Telomere-induced foci (TIF) in DNA-PKcs auto-phosphorylation mutants. TIFs form when dysfunctional telomeres trigger a DNA damage response, resulting in the recruitment of DNA repair proteins such as ATM and 53BP1 and the telomere-specific phosphorylation of H2AX to form γ–H2AX, a common marker for DNA DSBs. Thus, telomere FISH followed by immunostaining for γ–H2AX reveals the presence of uncapped telomeres at sites where telomeres and γ–H2AX signals co-localize. (A) An unirradiated cell expressing the Thr-2609 mutation exhibits a TIF. (B) Levels of telomere dysfunction as measured by TIF formation were nearly identical in the kinase dead and Thr-2609 mutants, consistent with our cytogenetic analysis (see Figure 3). Mutations in the Ser-2056 cluster of sites had little effect on telomere dysfunction indicating its non-essential role in telomere end-processing. (*$p < 0.05$ versus WT by student's t-test)