

Figure W1. Validation of the MPP assay. To check the accuracy of the procedure, we first needed to verify that the MPP assay was able to detect known *hTERT* transcription factors. To this end, we transfected HeLa cells with pCR3-USF2a, a pCMV plasmid that encodes bHLH USF2a, which acts as a heterodimer with USF1 in regulating *hTERT* promoter activity [8,9]. NEs were prepared from HeLa cells transfected with pCR3-USF2a or with the corresponding empty vector, and MPP was carried out as described in the Materials and Methods section. For both USF2a-overexpressing cells and control cells, the protein complexes eluted from the hCP or the BpX control DNA fragments were purified and subjected to Western blot analysis with antibodies recognizing either USF2a or USF1 isoforms. This figure shows that pCR3-USF2a transfection allowed the detection of both USF1 and USF2, whereas no signal was obtained either with the control lysate or with the BpX control DNA fragment. The MPP assay thus made it possible to detect known specific *hTERT* promoter partners that included not only the overexpressed USF2a product but also one of its cognate endogenous partners, USF1. We therefore used the same method to test whether Tax expression could modify the *hTERT* promoter proteome (Figure 1A).

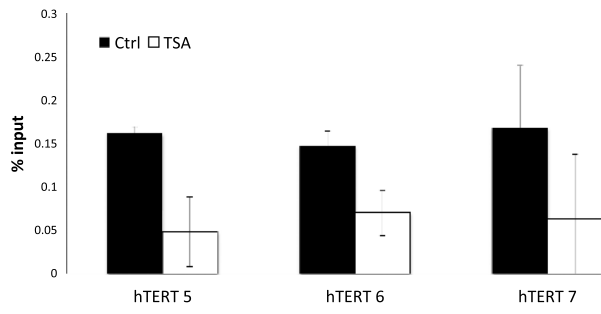


Figure W2. TSA treatment reduces DEK occupancy on the *hTERT* promoter. qChIP analysis of DEK association with the *hTERT* promoter was carried out as described in the Materials and Methods section. Results (means \pm SDs) are representative of triplicate experiments.