Figure S1 Scoumanne et al., 2010

3 h

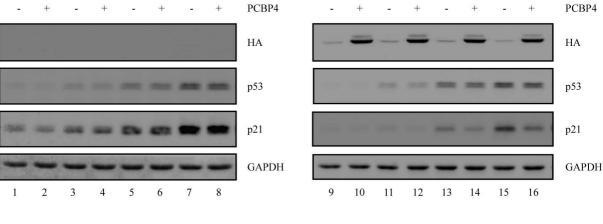
RKO-pTR-13

6 h

12 h

PCBP4, p53, p21 and GAPDH were detected by Western blot analysis.

CPT



RKO-PCBP4-HA-2

6 h

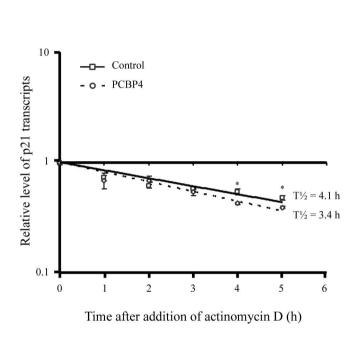
12 h

CPT

3 h

Figure S1. PCBP4 prevents the induction of p21 by DNA damage in RKO cells. Cell extracts were prepared from RKO-pTR-13 (left panel) and RKO-PCBP4-HA-2 (right panel) cells uninduced (-) or induced (+) to express PCBP4 for 2 days, and then untreated or treated with 200 nM CPT for 3, 6 or 12 h. Levels of HA-tagged

Figure S2 Scoumanne et al., 2010



MCF7 cells that were uninduced or induced to express PCBP4 for 2 days, and then treated with 2 μ g/ml actinomycin D for 0, 1, 2, 3, 4 or 5 h. Upon normalization to GAPDH transcript levels, the mean \pm S.D. from triplicate samples was plotted and the rel-

ative half-life of p21 transcript was calculated. *, p < 0.05.

Figure S2. PCBP4 expression decreases p21 mRNA half-life in MCF7 cells. Levels of p21 and GAPDH transcripts were measured by quantitative RT-PCR on cDNA samples from

Figure S3 Scoumanne et al., 2010

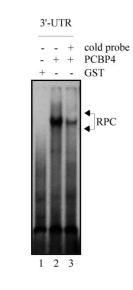


Figure S3. PCBP4 binds specifically to the 3'-UTR of p21. Competition REMSA assay was performed by adding an excess amount of unlabeled full-length p21 3'-UTR probe

to compete the binding of PCBP4 to radiolabeled p21 probe.