Expanded View Figures

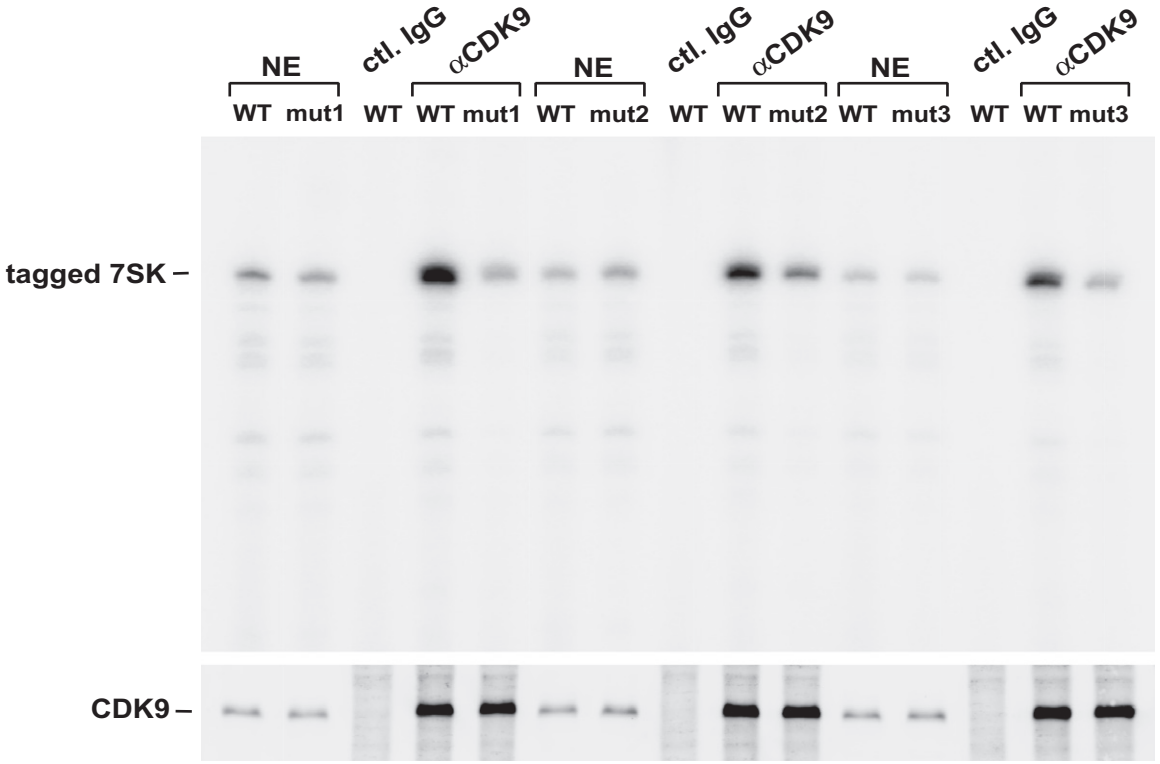

Figure EV1. Mutations at or around U250 in 7SK RNA prevent endogenous CDK9 from binding.
Tagged WT or mutant 7SK (mut1, mut2, and mut3) were transfected separately into HeLa cells. Anti-CDK9 IPs were analyzed by immunoblotting to detect CDK9 and primer extension for the bound 7SK RNA.

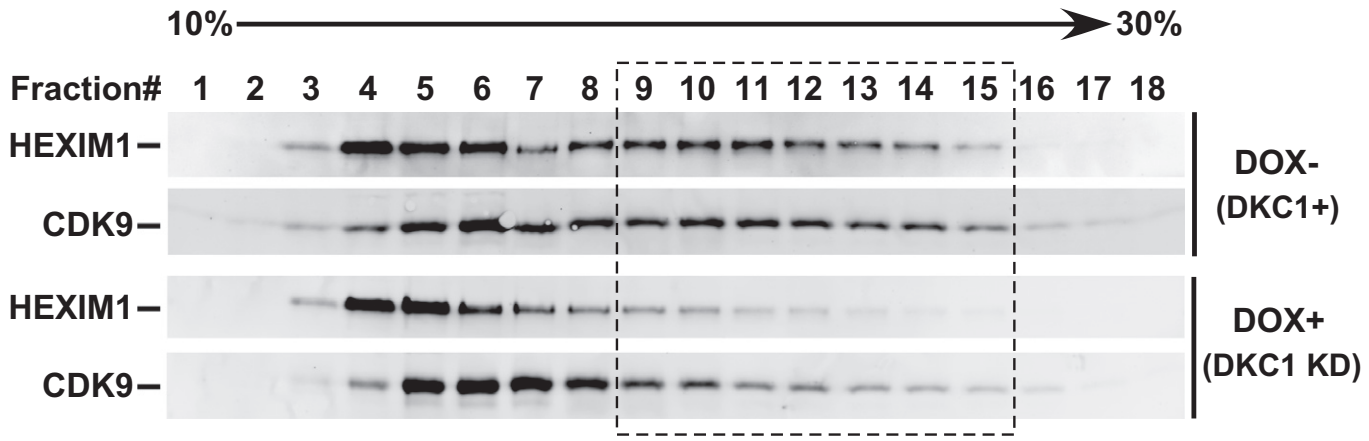

Figure EV2. Glycerol gradient analysis of DKC1 KD-induced 7SK RNP disruption.
NEs from inducible DKC1 KD clone 1 before and after the DOX-induced shDKC1 expression were analyzed by sedimentation through a 10–30% glycerol gradient. HEXIM1 and CDK9 in collected fractions were detected by immunoblotting. The dashed box highlights the fractions in which the levels of the two proteins drop upon KD.

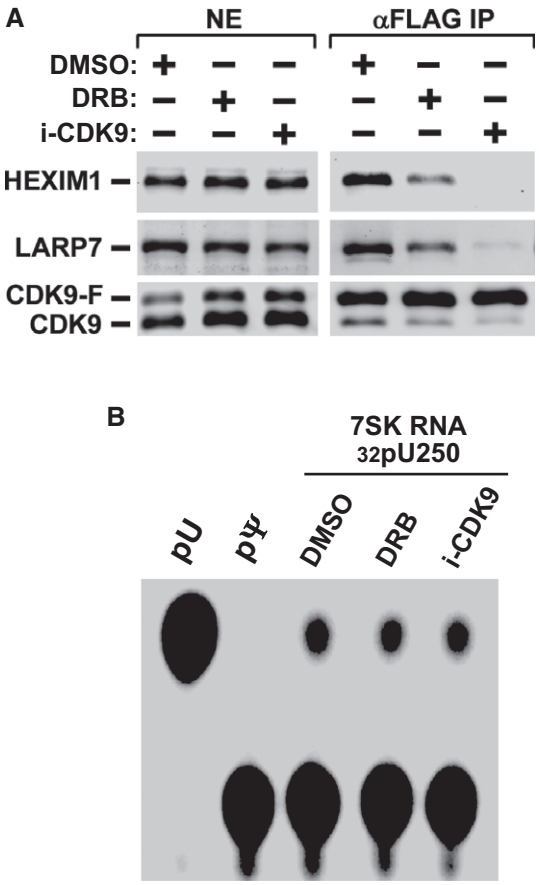

Figure EV3. Drug-induced 7SK RNP destruction does not change Ψ250 level in 7SK RNA.

A F1C2 cells expressing CDK9-F were treated with the indicated drugs or DMSO. Anti-Flag IP from NEs were analyzed by immunoblotting.

B Total RNA was purified from the treated cells, subjected to the procedure described in Fig 1D, and analyzed by 1D-TLC.

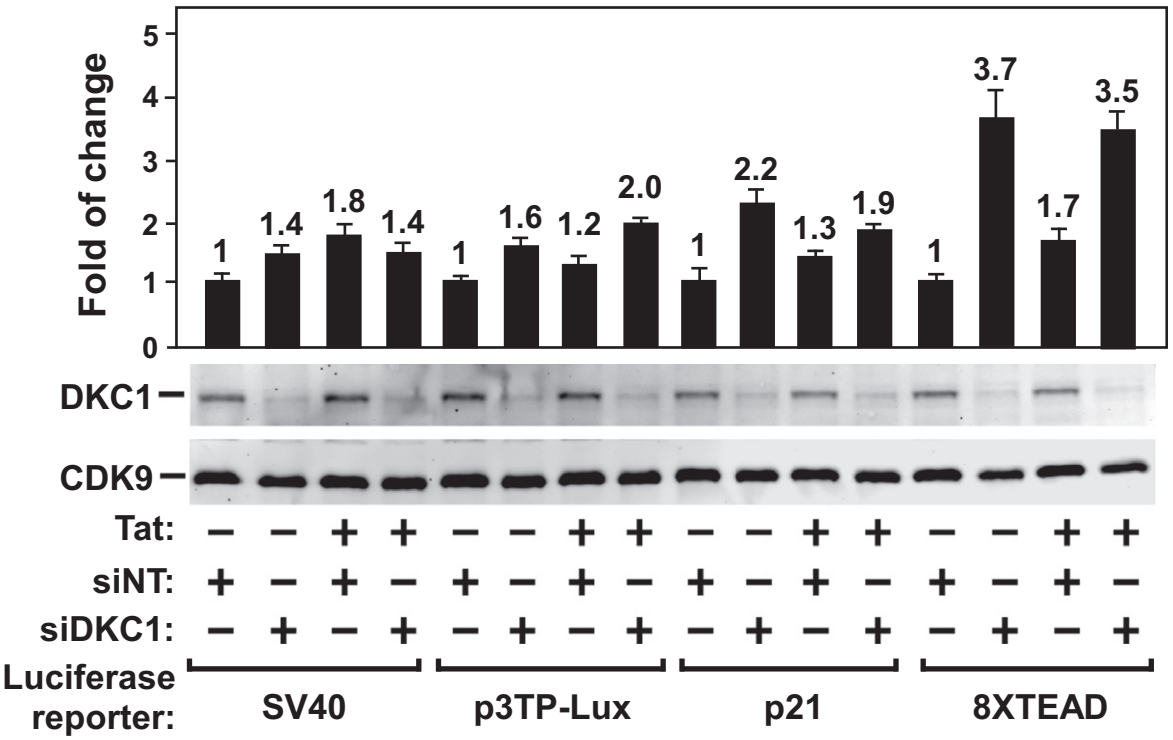

Figure EV4. Tat-transactivation is specific for HIV-1.
Luciferase activities were measured in HeLa cells transfected with the indicated reporter constructs and siRNA, and an empty vector (−) or the Tat cDNA. For each reporter, the activity in cells expressing siNT was set to 1. The bars represent mean ± SD from three independent experiments.