

Figure S1. Activation of wild-type and SUMO site mutant forms of Elk-1 by PIASxa. Reporter gene analysis of the activities of GAL-Elk fusion proteins on the GAL-driven E1b promoter-reporter construct in 293 cells. The activities of the wild-type (WT) and mutant (K2R) GAL-Elk(1-428) fusion proteins were tested in the presence or absence of PIASxa (10 ng) as indicated and presented as reporter activity relative to the activity of wild-type Elk-1 in the absence of PIASxa (taken as 1). The fold induction by inclusion of PIASxa is indicated above each set of bars. This data is an alternative depiction of a subset of Fig. 2B, and illustrates the higher basal activity of the K2R mutant. The residual activation by PIASxa that occurs in the absence of SUMO modification sites in Elk-1 is probably due to either non-specific effects or to affects on other coactivators bound to Elk-1 such as p300 (see Figs. 7 and 8).