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mutated). The latter pathway responds to UV-irradiation-induced DNA damage and leads to the activation of p53 through phosphorylation. We now show that the activated DNA-damage response protein kinase AKT2 directly phosphorylates the p53 binding protein and transactivator JunB and promotes their transcriptional activity on the p53RE-dependent reporter gene. Our results also demonstrate that the p53RE-dependent transcriptional activation by the activated AKT2 is restricted to the transactivation by JunB and not by ATF-2. In addition, UV-induced activation of the SIAH1 ubiquitin ligase promotes degradation of JunB. Thus, our data reveal a molecular mechanism involving the co-recruitment of JunB and Fra-1 by the DNA-damage response proteins AKT2 and SIAH1, respectively, for the transcriptional activation 79a2804d6b