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p53 mysteries

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No one really understands why activation of the tumor suppressor p53 sometimes leads to cell-cycle arrest and sometimes induces an apoptotic program. It has been proposed that post-translational modifications (phosphorylation and acetylation) induced by genotoxic stress affect the DNA binding affinity of p53. In the January 8 issue of Proceedings of the National Academy of Sciences, Kaeser and Iggo suggest that such models should be reassessed in light of their results using chromatin immunoprecipitation (ChIP)(*Proc Natl Acad Sci USA* 2002, **99:**95-100). To test p53 binding *in vivo* they developed a ChIP assay using several p53 mutations that affect transcription or DNA binding. They analyzed p53 binding to the promoters of genes involved in growth arrest or apoptosis in a range of human cell lines treated with different genotoxic agents. Kaeser and Iggo found no evidence of selective recruitment of p53 to these promoters in cells undergoing apoptosis. How p53 determines the choice between arrest or death remains a mystery.

References

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2. *Proceedings of the National Academy of Sciences*, [http://www.pnas.org]

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