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Oncogenic phosphatase amplification

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Post-translational regulation of p53 regulates its activity and tumour suppressor functions. In an Advanced Online Publication in Nature Genetics, Bulavin *et al.* from the National Institutes of Health describe how oncogenic Ras regulates p53 phosphorylation (*Nature Genetics* 20 May 2002, DOI:10.1038/ng894). Using antibodies specific for different modified forms of p53 they showed that oncogenic Ras induced p53, accumulation and phosphorylation of two specific serine residues that are substrates of p38 MAP kinase Bulavin *et al.* found that the p53-inducible phosphatase *PPM1D* abrogates p53 phosphorylation indirectly by inhibiting MAP kinase activity; overexpression of *PPM1D* was oncogenic in p53-expressing cells. They found evidence for amplification and overexpression of the human *PPM1D* gene in breast cancer cells expressing wild-type p53. An accompanying report by Li *et al.* provides additional evidence for *PPM1D* locus amplification and overexpression in breast cancer cells (*Nature Genetics* 20 May 2002, DOI:10.1038/ng888). These results suggest that amplification of the chromosome 17q22/24 region containing the *PPM1D*gene suppresses p53 function by regulating post-translational modification.

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