Glycogen synthase kinase3 beta phosphorylates serine 33 of p53 and activates p53's transcriptional activity

ABSTRACT
Phosphorylation of p53 serine 33 by GSK3 can regulate transcriptional activity. However, GPK3+ is not believed to be involved in the underlying mechanism of DNA damage response. Instead, it may serve as the link between a specific form of protein KOHOH and an activating non-DNA damage mechanism.

INTRODUCTION
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In the present study, we investigated the effects of glycogen synthase kinase3 beta phosphorylates serine 33 of p53 on the transcriptional activity of the promoter of the p53-deficient mouse. The p53-deficient mice were transgenic, and the mice were homozygous for the GSK3 beta-peptide. The GSK3 beta-peptide gene was upregulated in the presence of glycogen synthase kinase3 beta phosphorylates serine 33 of p53. In contrast, the GSK3 beta-peptide gene was down Different types of cell processes trigger the activation of the p53 tumor suppressor gene. These include DNA damage caused by Ionizing Radiation and genotoxic agents, expression of activated oncogenes such as rass or myc; progression of primary cells towards senescence; and various stimuli that trigger growth arrest or apoptosis are present. Moreover, this type of gene also functions in different ways in the cell as an inhibitor of transcriptional regulators. Furthermore, the majority of cellular effects of these regulated genes involved at least one phosphoryry The phosphorylation of serine 15 and 20 of the p53 protein is caused by DNA damage and oncogene expression. The initial step in p53 activation is the stabilization of the underlying protein, but there are other steps, such as increasing DNA binding activity and altering a corresponding transcriptional factor. By stimulating the p53 kinase, such as Glycogen Synthylobacter 3 (GSK3), which is involved in the regulation of cell growth and differentiation, or by inhibiting the expression of certain transcription factors like HSF1 and NFATc, GSK3+ is shown to play a role in regulation. GSK3 inhibition is triggered by growth factors activating the p110-PI 3-kinase/Protein Kinases B (PKB) pathway. When activated PKB phosphorylates GK3, inhibiting GNK3 activity. This activation of the whole ppk pathway and selective inhibition of GKK3 leads to the delivery of a potent anti-apoptotic signal to cells. Given the well-defined role of The consensus sequence Ser-X-Y-Ser(P) is phosphorylated by GSK3, which means that only GSH3 binds to proteins that have already been cleared by a distinct priming kinase. Furthermore, there are 5 potential GK3 Phosphorylation sites in the p53 complex, with 3 in each N-terminal transactivation domain and 2 within the C- terminal regulatory domain. We demonstrate that GPK3 can repress serine 33 in vitro while it is active, but G

CONCLUSION
Remarkable conclusions This study shows GSK3 (but not GSH3), which can directly phosphorylate the serine 33 of p53 when already phosphate 37 of the latter two selines of that protein are 'phosphorylated'; that GK3+ "takes the necessary form of transcriptional activation in vivo and that it may up-regulate/activate a supposedly non-DNA damage pathway such as

oncogene activator" (the name given by Richardson).