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Human RAD9B variant 2 regulates the phosphorylation of CHK2 kinase

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Human RAD9A and RAD9B protein are two isoforms of the DNA lesion response protein RAD9. RAD9A and Rad9B form the Rad9-Rad1-Hus1 checkpoint ring at sites of DNA damage. The RAD9 protein participates in the DNA repair process through regulating cell cycle checkpoints and the apoptotic pathway. The checkpoint kinases CHK1 and CHK2 are associated with single and double stranded DNA damage jointly with ATR and ATM kinase, respectively. RAD9A and RAD9B isoforms differ from each other by their amino acid sequence; RAD9B is larger than RAD9A. So far it is unknown why human cells express to related Rad9 proteins. The RAD9B gene encodes four protein splice variants and the results presented here identify splice variant 2 as a regulator or CHK2 phosphorylation. Only over-expression of variant 2 but not variant 1 in HEK293 cells results in the hyper-phosphorylation of CHK2 in undamaged cells. It is still not yet clear which kinase is involved in the aberrant modification of CHK2. This is the first evidence identifying a specific function for Rad9B and more specifically for one of its protein variants.

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