CK2 functionally interacts with AKT/PKB to promote the ?-catenin- dependent expression of survivin and enhance cell survival

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?-Catenin is crucial in the canonical Wnt signaling pathway. This pathway is up-regulated by CK2 which is associated with an enhanced expression of the antiapoptotic protein survivin, although the underlying molecular mechanism is unknown. AKT/PKB kinase phosphorylates and promotes ?-catenin transcriptional activity, whereas CK2 hyperactivates AKT by phosphorylation at Ser129; however, the role of this phosphorylation on ?-catenin transcriptional activity and cell survival is unclear. We studied in HEK-293T cells, the effect of CK2-dependent hyperactivation of AKT on cell viability, as well as analyzed ?-catenin subcellular localization and tran-scriptional activity and survivin expression. CK2? over-expression led to an augmented ?-catenin-dependent transcription and protein levels of survivin, and consequently an enhanced resistance to apoptosis. However, CK2?-enhancing effects were reversed when an AKT mutant deficient in Ser129 phosphorylation by CK2 was co-expressed. Therefore, ou