Phosphorylation of AKT/PKB by CK2 is necessary for the AKT-dependent up-regulation of ?-catenin transcriptional activity

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?-Catenin is a key protein in the canonical Wnt signaling pathway and in many cancers alterations in transcriptional activity of its components are observed. This pathway is up-regulated by the protein kinase CK2, but the underlying mechanism of this change is unknown. It has been demonstrated that CK2 hyperactivates AKT/PKB by phosphorylation at Ser129, and AKT phosphorylates ?-catenin at Ser552, which in turn, promotes its nuclear localization and transcriptional activity. However, the consequences of CK2-dependent hyperactivation of AKT on ?-catenin activity and cell viability have not been evaluated. We assessed this regulatory process by manipulating the activity of CK2 and AKT through overexpression of wild-type, constitutively active and dominant negative forms of these proteins as well as analyzing ?-catenin-dependent transcriptional activity, survivin expression and viability in HEK-293T cells. We observed that CK2? overexpression up-regulated the ?-catenin transcriptional act