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ERRATUM

Protein kinase CK2 is involved in G2 arrest and apoptosis following spindle damage in epithelial cells

Mohamed Sayed, Steven Pelech, Connie Wong, Anthony Marotta and Baljinder Salh

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Correction to: *Oncogene* (2001) **20,** 6994–7005.

Owing to a typesetting error, the last line of the figure legends for Figures 1–4 were missing in the above paper. The full figure legends are reproduced below.

Figure 1 Function of CK2 as a mitotic checkpoint protein. (**a**) HeLa cells, treated with CK2 sense, antisense or left untreated, were deprived of serum and released into the cell cycle by addition of serum. After 18 h, cells were either not treated (control) or exposed to nocodazole (400 ng/ml) for 12 h before being photographed. (**b**) Failure of HeLa cells transfected with antisense to undergo mitotic arrest. The shaded bars and error bars represent the means and standard deviations, respectively, determined from at least three independent assessments. (**c**) Depletion of the endogenous CK2α in HeLa cells transfected with CK2α-α' antisense. Equal amounts of protein extracts were subjected to protein immunoblot analysis with monoclonal anti-CK2α antibody. Lower control panel shows equivalent amounts of GAPDH. This experiment was repeated at least four times with similar results

Figure 2 (**a**) Suppression of nocodazole-induced MPF activity by either CK2 antisense or a specific inhibitor of p38. HeLa cells were synchronized at G1-S by double thymidine block and either collected (control) or released into cell cycle. After release, the cells were incubated with or without nocodazole (400 ng/ml). In some experiments SB203580 (20 μM) or DRB (20 μM) was added 2 h prior to nocodazole treatment. Cell lysates were then prepared and histone H1 phosphotransferase activities were measured. (**b**) cdc2 activity is downstream to CK2 and p38 MAPK. Proteins were immunoprecipitated with anti-cdc2 antibody and its phosphotransferase activity was assayed toward histone H1. (**c**) Activation of CK2 in nocodazole-arrested cells is dependent on p38 MAP kinase. CK2 protein in cell lysates, prepared from the above treatment, was immunoprecipitated with monoclonal anti-CK2α antibody and assayed for phosphotransferase activity towards the CK2 specific substrate RRADDSDDDDD. The lower panel shows equal amounts of CK2 protein were immunoprecipitated. (**d**) CK2 is the major kinase phosphorylating p53 at serine 392. Cell lysates from the above treatment were immunoblotted with phospho antibodies to p53 (serine 392) and p38α. (**e**) p38 protein was immunoprecipitated with anti-p38α antibody and assayed for phosphotransferase activity toward activating transcription factor 2 (ATF2). The lower panel shows equal amount of p38 protein was immunoprecipitated. All of these experiments were repeated at least three times with similar results

Figure 3 Mitotic arrest is compromised in the absence of CK2. (**a**) Synchronized HeLa cells (in G0), treated with CK2 antisense or sense, were released into cell cycle and collected for flow cytometric analysis in the presence and absence of nocodazole (200 ng/ml) at the indicated time. Lysates from the above treated cells were immunoprecipitated with anti-cdc2 antibody and assayed for cdc2 phosphotransferase activity (**b**) or either subjected to immunoblotting with anti-cyclin B (**c**) or cdc2 (**d**) protein. This experiment was repeated at least four times with similar results

Figure 4 p53 is required for CK2 to override the mitotic arrest. (**a**) Flow cytometric analysis of exponentially growing HCT116 ($p53^{+/+}$) and HCT116 ($p53^{-/-}$) cell lines transfected with CK2 antisense or left untreated (control) in the presence and absence of nocodazole (200 ng/ml) for the indicated times (cells were synchronized as for Figure 3). (**b**) Mitotic indices of HCT116 and HeLa cells are shown at the indicated times after exposure to nocodazole. (**c**) HCT116 ($p53^{-/-}$) and $p53^{+/+}$ (**d**) cell lysates were immunoblotted with anti-cyclin B. A control immunoblot using α-GAPDH antibody is shown for the $p53^{+/+}$ cells. Similar results were obtained at least on three separate occasions