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CORRIGENDUM

CHEK2 genomic and proteomic analyses reveal genetic inactivation or endogenous activation across the 60 cell lines of the US National Cancer Institute

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Figure 5b (left panel) has been corrected. The arrows have been repositioned for some of the cell lines to indicate their p53 wild-type status based on our published data (CellMiner, http://discover.nci.nih.gov/cellminer/mutationGeneLoad.do, and ongoing exome sequencing of the NCI60).

This change strengthens our conclusions that endogenously activated Chk2 (pT68-Chk2) is only observed in p53-defective cells. All the 16 cell lines with wild-type p53 were negative for activated Chk2 whereas the 8 cell lines (including HeLa) with activated Chk2 were all p53 defective ($P<0.05$, χ^2, two-sided).

The Corrected Figure 5 is shown on the next page.

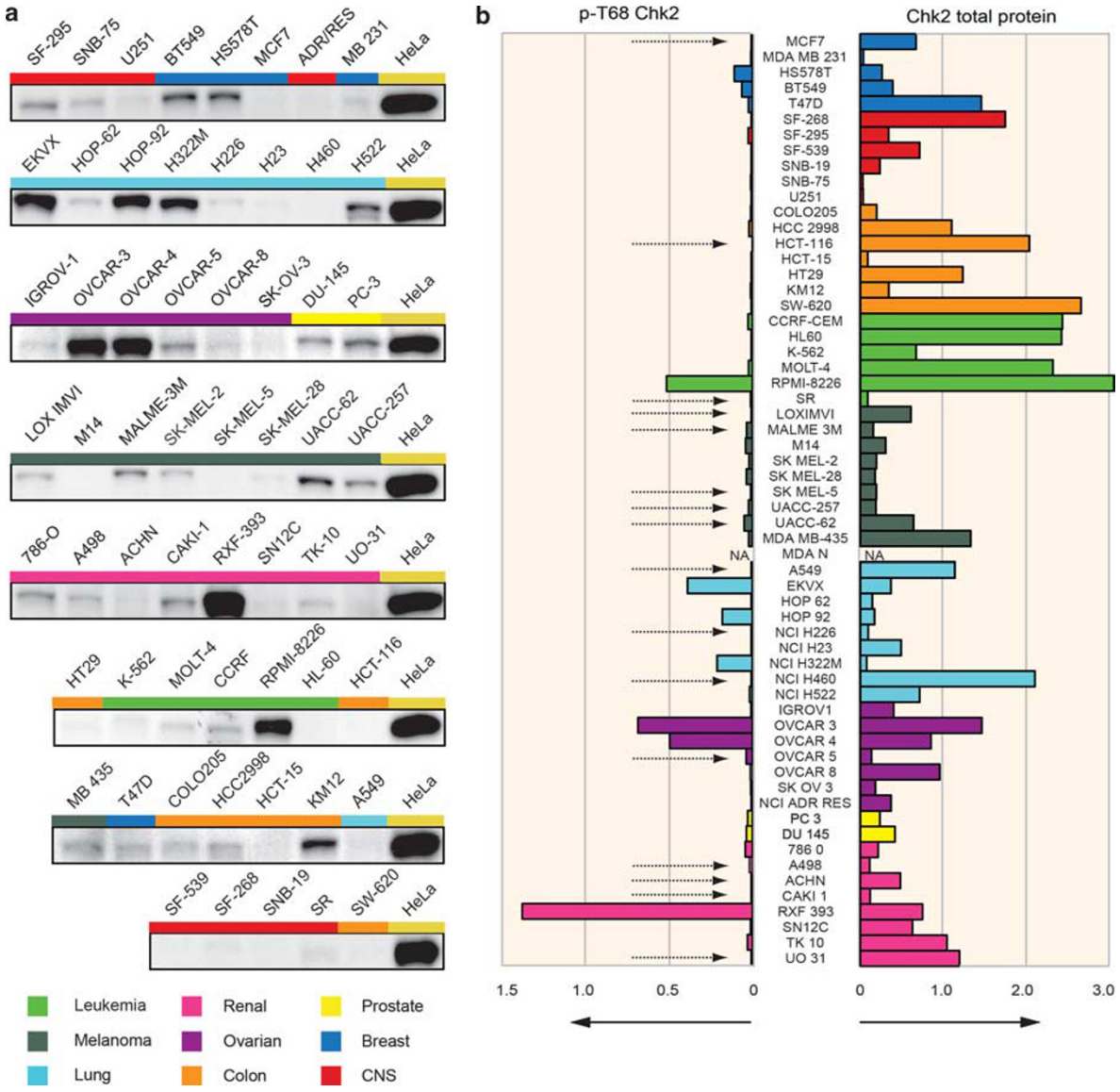

Figure 5 Proteomic analysis of total and phosphorylated (pT68) Chk2 shows high endogenous levels of Chk2 activation in several cancer cell lines across the NCI-60. (**a**) Representative western blot pictures of pT68-Chk2 in the NCI-60. The colored bars correspond to the tissues of origin (see legend at the bottom). HeLa cells were used as positive control and to calculate the Chk2 phosphorylation ratios in the NCI-60. (**b**) Quantitation of pT68-Chk2 (left) and total Chk2 (right) protein levels in the NCI-60. The colored bars represent the means of the ratios between the western blot intensities of the individual cell lines and the HeLa reference loading, after normalization by β-actin loading. The colors represent the tissues of origin (see also Figure 1). The dotted arrows indicate known p53 wild-type cancer cell lines. NA: not available.