Correction

Correction: Cyclin E/Cdk2-dependent phosphorylation of Mcl-1 determines its stability and cellular sensitivity to BH3 mimetics

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This article has been corrected: In Figure 4H, the first two panels in the β-actin row are accidental duplicate images. As a result, these changes alter the ratio of Mcl1/b-actin for this panel, which is presented on the graph (panel I). The corrected Figure 4 with new images for panel H and panel I, obtained using the original data, is shown below. The authors declare that these corrections do not change the results or conclusions of this paper.

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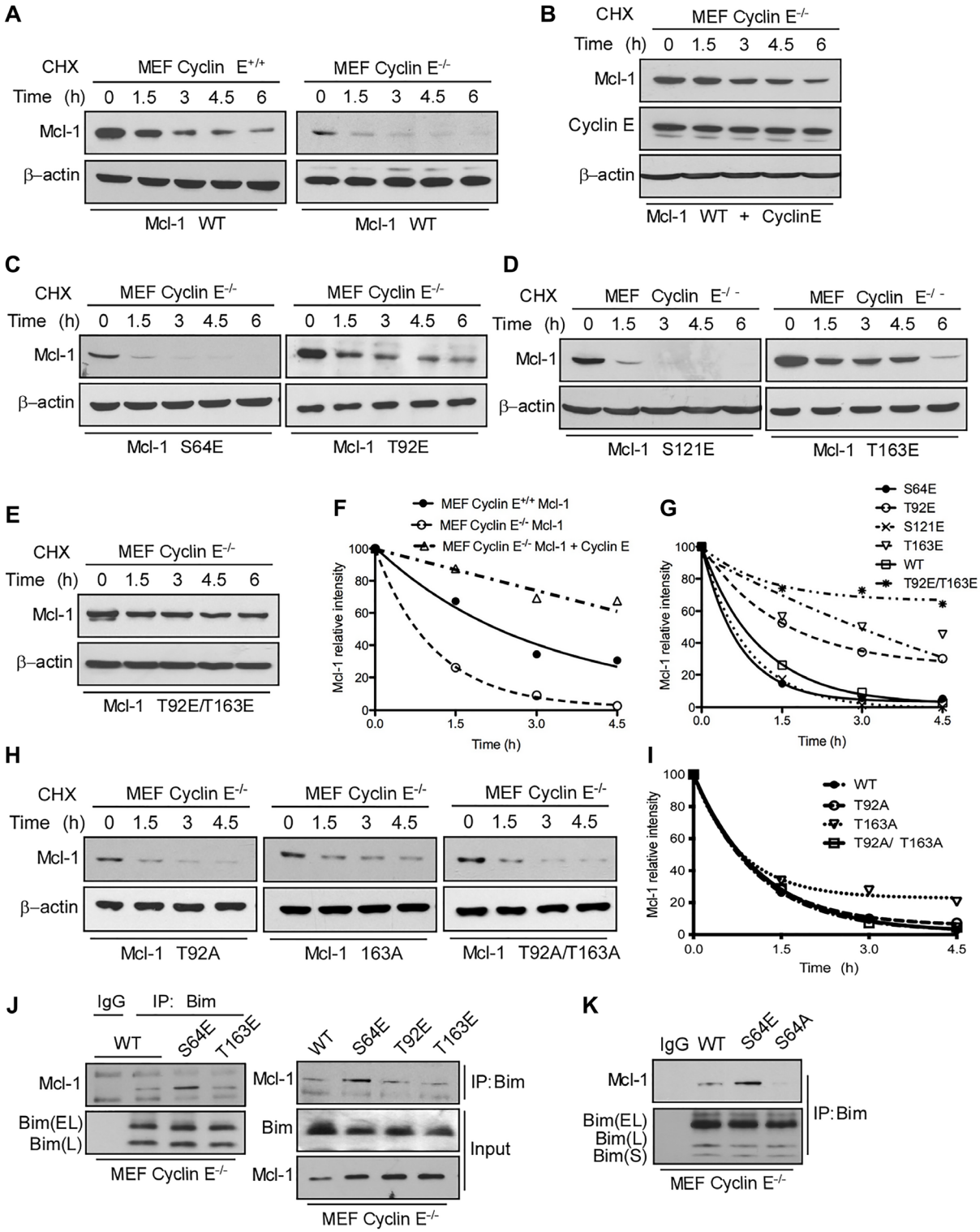

Figure 4: Mcl-1 stability and Bim sequestration is dependent on cyclin E/Cdk2. Mcl-1 protein half-life was determined by expressing WT Myc-Mcl-1 (**A**). individually in cyclin $E^{+/+}$ and cyclin $E^{-/-}$ MEFs (**B**). together with HA-cyclin E in cyclin $E^{-/-}$ MEFs and then treating with cycloheximide for the indicated time, followed by immunoblotting. Immunoblot analysis of cyclin $E^{-/-}$ MEFs transfected with (**C**). S64E, T92E, (**D**). S121E, T163E (**E**). T92E/T163E (**H**). T92A, T163A and T92A/T163A Mcl-1 mutants and treated with cycloheximide for the indicated time. Data in (**F**)., (**G**)., (**I**). were quantified by ImageJ. Cyclin $E^{-/-}$ MEFs were transfected with (**J**). Myc-Mcl-1 WT, S64E, T92E and T163E (**K**). Myc-Mcl-1 WT, S64E and S64A. After 24 h, Bim was immunoprecipitated and its association with Mcl-1 was analyzed by immunoblotting. β-actin was used as loading control. These data are representative of three independent experiments.