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Correction

Correction to: Interdependence of laforin and malin proteins for their stability and functions could underlie the molecular basis of locus heterogeneity in Lafora disease

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The image of anti-Myc blot of figure 2C (third panel; Malin-Myc [C26S]) was inadvertently used once again for the γ-tubulin loading control of figure 2B. The revised figure 2B with the correct image of the γ-tubulin loading control is given below. The interpretation and conclusion provided in the article do not change because of the correction.

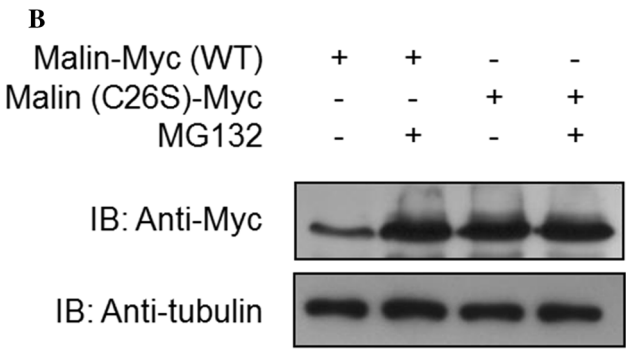

Figure 2. Malin promotes its own degradation: (**A**) The wild-type (WT) or the catalytically inactive (C26S) mutant malin was overexpressed in COS-7 cells and processed for pull-down assay using anti-ubiquitin antibody. The pull-down products (PD) and whole cell lysates (WCL) were resolved and immunoblotted (IB) with indicated antibodies. (**B**) Myc/His-tagged malin or Myc/His-tagged C26S malin was transiently expressed in COS-7 cells either in the presence or absence of MG132 as indicated. Equal amount of whole cell lysate for each combination was resolved in SDS-PAGE and immunoblotted with anti-Myc antibody or anti-γ-tubulin antibody (as loading control) to show the difference in the cellular levels of malin. (**C**) Transfections were done in COS-7 cells in 24-well plate for Myc-tagged wild-type malin or the Myc-tagged C26S mutant malin (300 ng/well) with increasing proportion of an expression construct (0, 100, 200, or 400 ng/well in lanes 1 to 4, respectively) for the HA-tagged wild-type ubiquitin (WT) or its mutant (K48R) as indicated. Equal amount of whole cell lysate from each well was resolved in SDS-PAGE and immunoblotted with anti-Myc antibody or anti-γ-tubulin antibody (as loading control) to show the difference in the cellular levels of the proteins.