Correction

Correction: HRD1 suppresses the growth and metastasis of breast cancer cells by promoting IGF-1R degradation

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This article has been corrected: In Figure 7B, the 1st and 3rd panels contain an accidental overlap. In Figure 5D, the 2nd and 4th panels also contain an accidental overlap. In Figure 4, the relative protein level of p-Akt in Figure 4A, as well as the bands of IGF-1R in Figure 4C–4E, contain mistakenly selected images. The corrected figures, produced using the original data, are shown below. The authors declare that these corrections do not change the results or conclusions of this paper.

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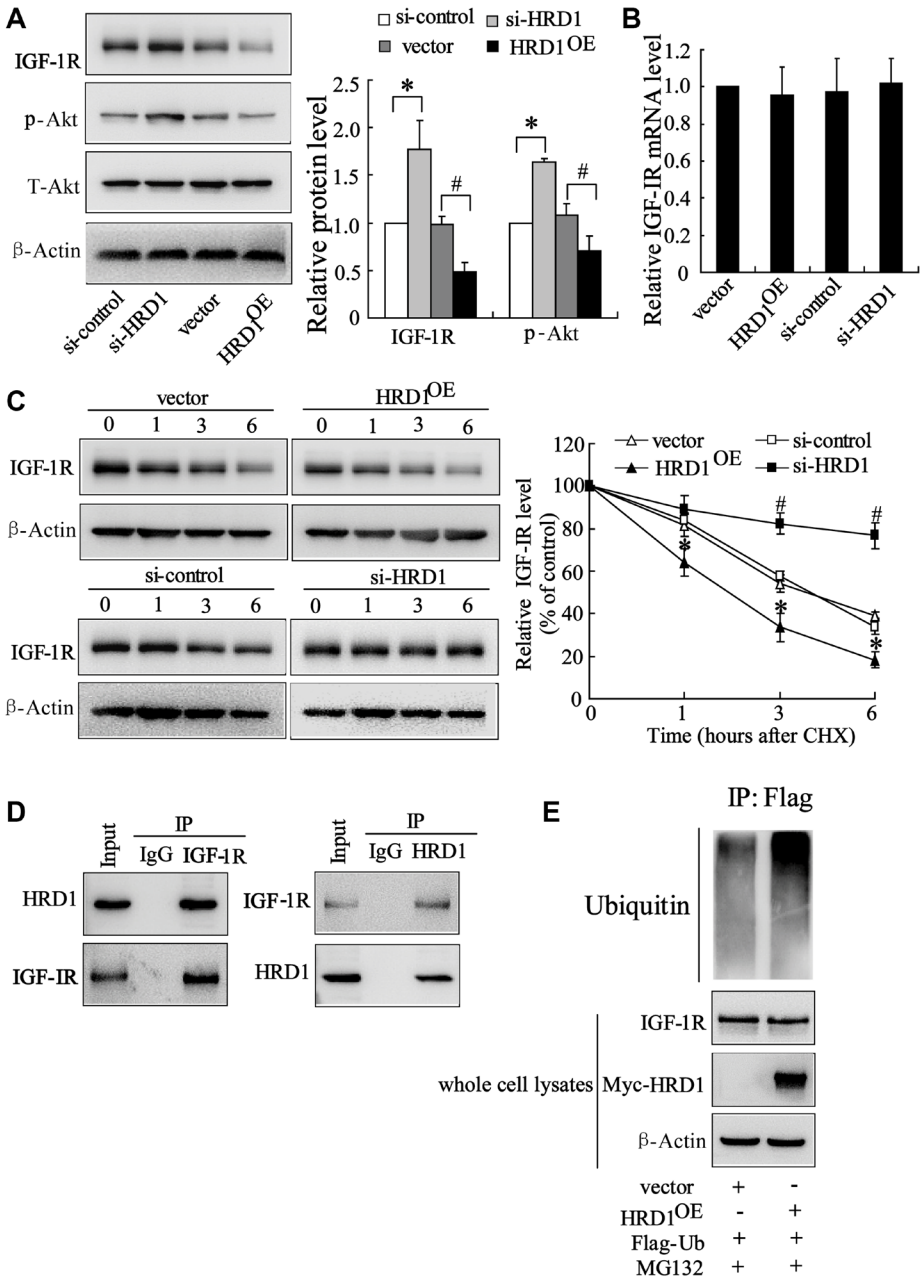

Figure 4: HRD1 promotes IGF-1R ubiquitination for degradation. (**A**) The protein levels of HRD1, IGF-1R and the downstream target P-Akt were measured by immunoblotting in HRD1 overexpressing (HRD1OE), HRD1 siRNA (si-HRD1) and their respective control transfected MCF-7 cells. (**B**) Total mRNA was prepared from HRD1OE, si-HRD1 and their respective control transfected MCF-7 cells and IGF-1R mRNA levels were quantified using real time RT-PCR. (**C**) MCF-7 cells were transfected with HRD1OE, si-HRD1 and their respective control for 48 h, followed by exposure to cycloheximide (CHX 50 mg/ml) for 0, 1, 3, or 6 h. the protein of IGF-1R and HRD1 in whole cell lysates was measured by immunoblotting. The intensity of the IGF-1R protein bands was analyzed by densitometry, after normalization to the corresponding β-Actin level. (**D**) MCF-7 cells were pretreated with MG132 (10 μM) for 6 h and endogenous protein-protein interactions between HRD1 and IGF-1R were determined by immunoprecipitation (IP) with HRD1 or IGF-1R antibodies, followed by immunoblotting. IgG was used as a negative control for IP. (**E**) Ubiquitination of IGF-1R was induced by HRD1. Flag-ubiquitin was coexpressed in MCF-7 cells with myc-HRD1 or vector control with treatment of MG132 (10 μM) for 6 h. Ubiquitinated IGF-1R protein was immunoprecipitated using Flag-Tag antibody and further detected with Anti-IGF-1R antibody. The endogenous IGF-1R and myc-HRD1 in the whole cell lysates were examined by anti-IGF-1R and anti-myc antibodies. $^{*}P < 0.05$, compared to vector. $^{\#}P < 0.05$, compared to si-control.

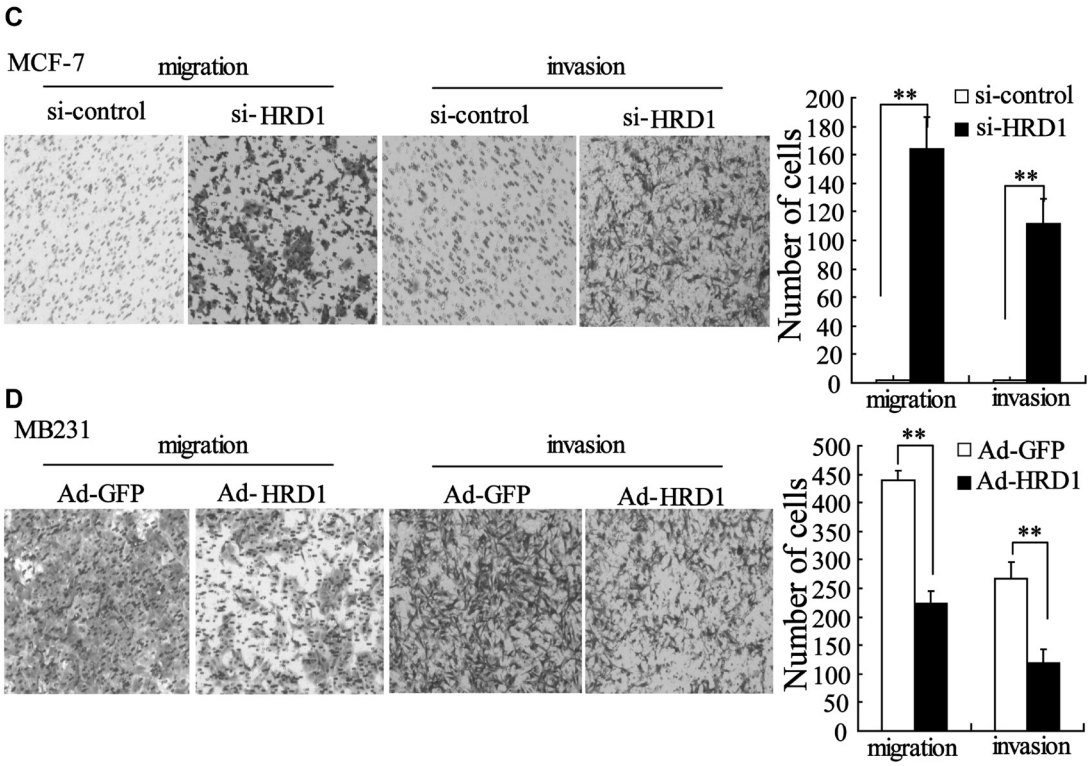

Figure 5: HRD1 inhibits growth, colony formation, migration, and invasion of breast cancer cells. (**C**) MCF-7 cells were transfected with si-control or si-HRD1 for 48 h prior to the transwell assays. $^{**}P < 0.01$, compared to si-control. (**D**) MB231 cells were infected with Ad-GFP or Ad-HRD1 for 48 h, and then transwell assays were performed. $^{**}P < 0.01$, compared to Ad-GFP.

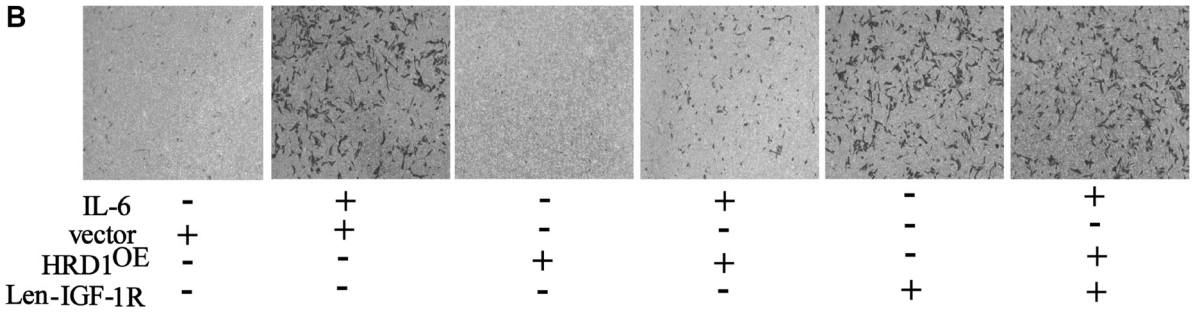

Figure 7: HRD1 is implicated in breast cancer cell EMT. (**B**) MCF10A cells stably expressing HRD1 were infected with lentivirus of IGF-1R for 48 h, and then treated with or without IL-6, followed by transwell.