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Corrigendum: Danshensu methyl ester enhances autophagy to attenuate pulmonary fibrosis by targeting lncIAPF–HuR complex

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A Corrigendum on
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In the published article, there was an error in Figure 5 as published. The quantitative grouping label of the protein bands in Figure 5D was incorrectly labeled as "TGF-β1+DME, TGF-β1+DME + si-HuR NC, TGF-β1+DME + si-HuR". The corrected Figure 5 and its caption appear below.

The authors apologize for this error and state that this does not change the scientific conclusions of the article in any way. The original article has been updated.

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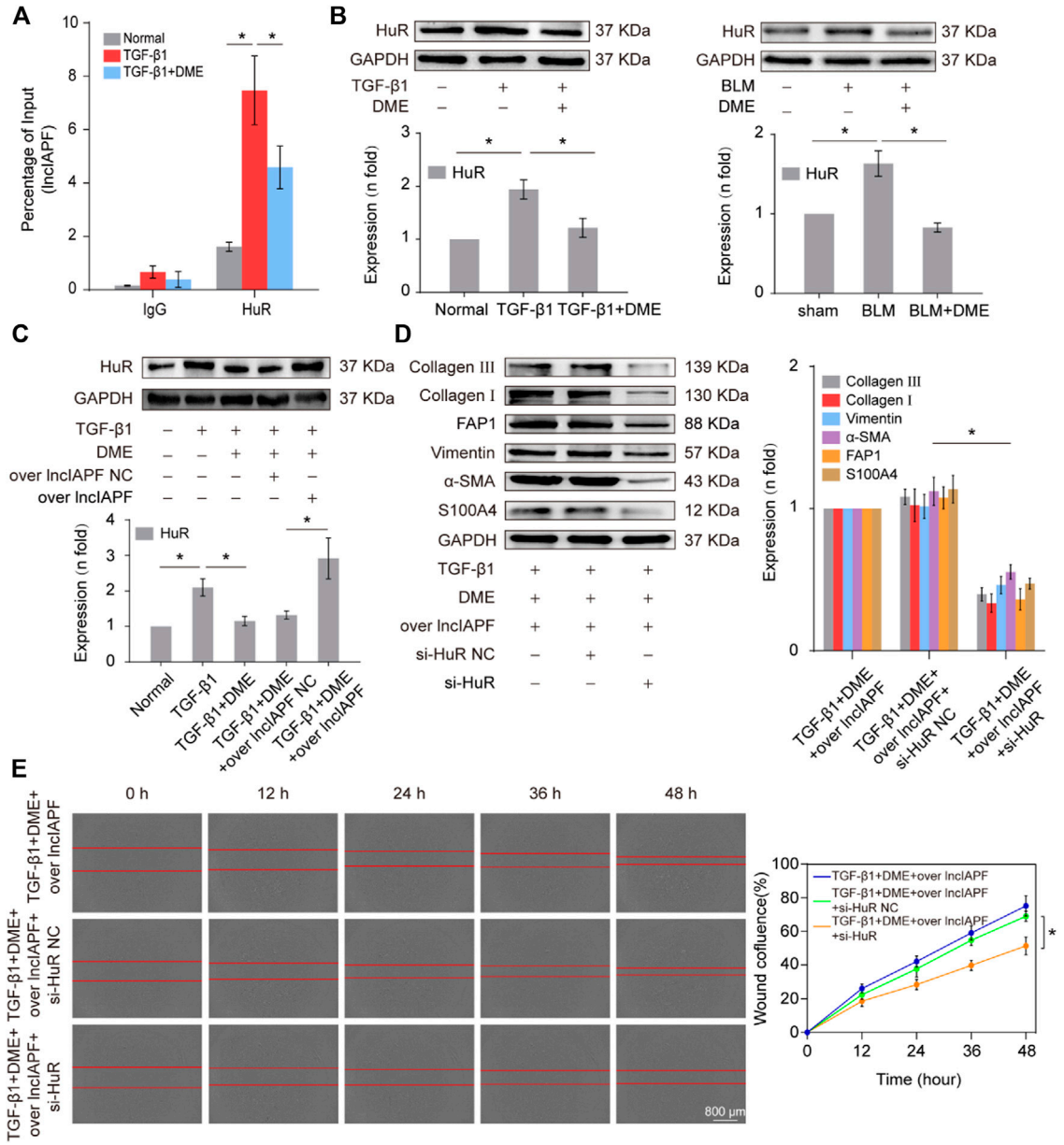

FIGURE 5
(Continued).

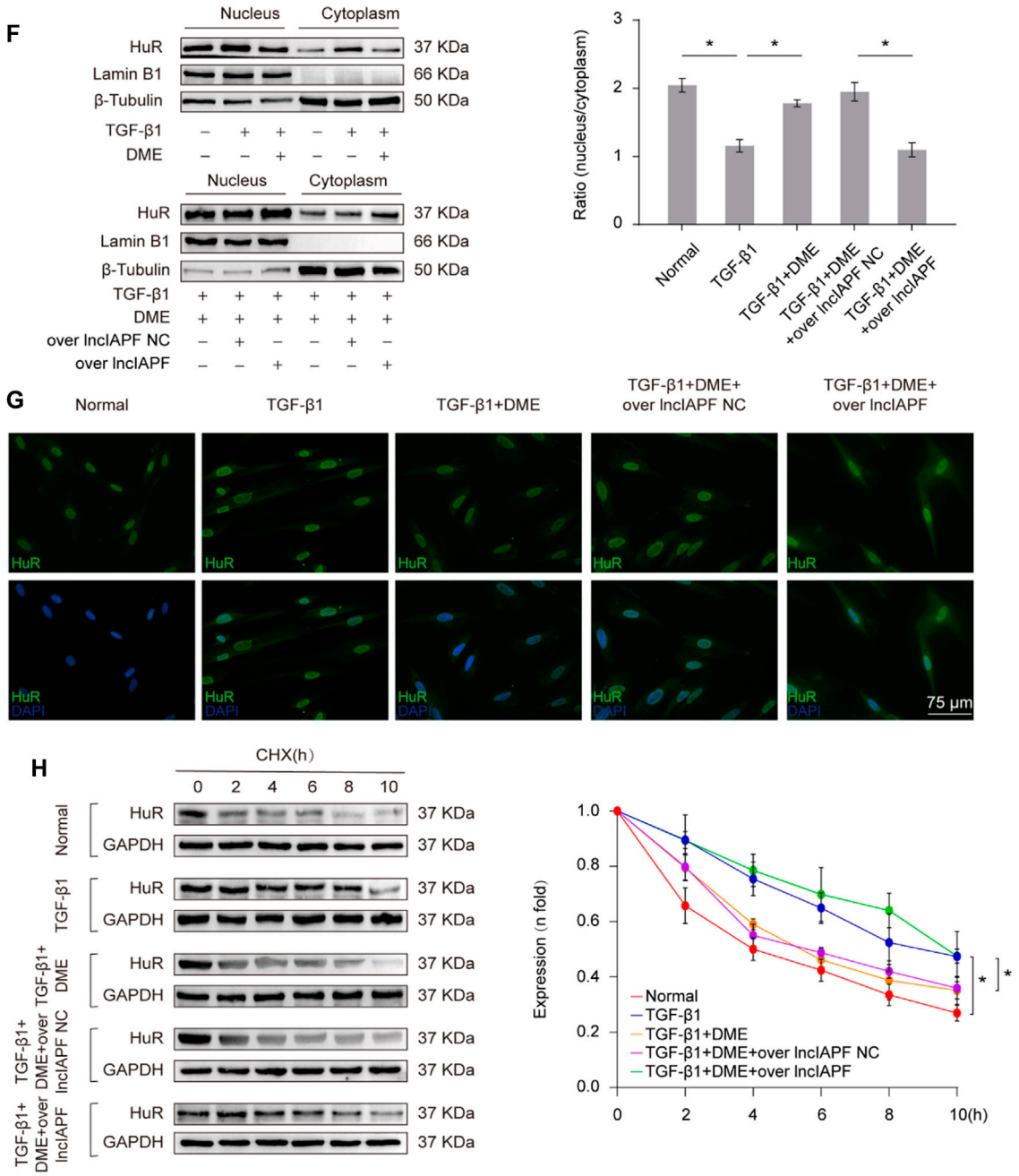

FIGURE 5
(Continued). Regulatory mechanism of DME on lncIAPF–HuR. **(A)** The RIP experiment verified the binding relationship between lncIAPF and HuR and the effect of DME on their binding. **(B)** Western blot result showed that the expression of HuR increased in the model group and decreased in the treatment group. **(C)** The rescue experiment of Western blot showed that DME reduced HuR expression, and lncIAPF overexpression increased HuR expression and reversed the downward trend caused by DME. **(D)** The rescue experiment of Western blot showed that interference with HuR decreased the expression of S100A4, FAP1, α-SMA, vimentin, collagen I and III, and reversed the upward trend caused by lncIAPF overexpression. **(E)** The rescue experiment of scratch assay showed that HuR interinterference reversed the trend of accelerated migration caused by lncIAPF overexpression. **(F)** Nucleocytoplasmic separation experiment showed that DME blocked the nucleocytoplasmic translocation of HuR, but lncIAPF overexpression reversed the effect of DME. β-Tubulin was used as the cytoplasmic reference, and Lamin B1 was used as the nucleus. The results of nucleoplasmic separation were quantitatively analyzed by Image J software as follows: Normal: nucleus/plasm = 2.0, TGF-β1: nucleus/plasm = 1.3, TGF-β1+DME: nucleus/plasm = 1.8, TGF-β1+DME + overlncIAPF NC: nucleus/plasm = 1.9, TGF-β1+DME + overlncIAPF: nucleus/plasm = 1.1. **(G)** Immunofluorescence experiment showed that HuR was primarily localized in the nucleus of normal cells, and it transferred from the nucleus to the cytoplasm under the action of TGF-β1 or lncIAPF overexpression. DME blocked the nucleocytoplasmic translocation of HuR, but lncIAPF overexpression reversed the effect of DME. **(H)** Cycloheximide experiment verified the stability of the HuR protein. DME weakened HuR stability, but lncIAPF overexpression reversed this trend. The half-life of HuR in each group was presented as follows: normal: T1/2 = 3.07 h, TGF-β1: T1/2 = 10.17 h, DME: T1/2 = 3.92 h, DME + over lncIAPF NC: T1/2 = 4.76 h, DME + lncIAPF: T1/2 = 12.33 h. The concentration of DME used was 10 μg/ml. Each bar represents the mean ± SD; n = 6; *$p < 0.05$.