Corrigendum

Extracellular heat shock protein-90 α : linking hypoxia to skin cell motility and wound healing

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The authors have identified an error in Figure 2 of the above article.

During the construction of Figure 2, the loading control (panel d) for panel c was mistakenly re-used as a loading control (panel g) for panel f.

As shown in the new Figure 2B below, an internal loading control (panel g') for HIF-1 β (panel g), as well as an internal loading control (panel f) for HIF-1 α (panel e), has been included.

Conclusions of the data, however, remain unchanged.

Also on page 1229, the second line of the left column in the text, 'sp90 α ' should be 'Hsp90 α '.

The authors apologize for any confusions these errors might have caused.

Figure 2 Induction of HIF-1 α in HDFs by hypoxia versus PDGF-BB and establishment of HIF-1 α constitutively expressing cells. The lysates of HDFs, which were subjected to (A) hypoxia (1% O₂) or normoxia (20% O₂) for the indicated time, (B) various levels of hypoxia or (C) stimulation with PDGF-BB (15 ng/ml, under 20% O₂) for the indicated times, were analyzed by Western immunoblotting analysis using antibodies specifically against HIF-1 α (panels a, c, e and h) or HIF-1 β (panel g). Anti-p38 antibody blotting of the lower portions of the same or duplicate membranes was used as sample loading control (panels b, d, f, g' and i). (D, E) HDFs were infected with lentivirus carrying vector (vec.), HIF-1 α wt, constitutively activated HIF-1 α (CA) or dominant-negative HIF-1 α (DN). After 48 h following infection, the lysates of the cells were immunoblotted with anti-HIF-1 α antibodies (D, panel j) or anti-HA tag antibody (E, panel l). Anti-p38 antibody blotting of the lower portions of the same membranes was used as a sample loading and subsequent densitometry scan control (panels k and m).