CORRIGENDUM

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Hypoxia drives the transition of human dermal fibroblasts to a myofibroblast-like phenotype via the TGF-β1/Smad3 pathway

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Following the publication of this article, the authors noticed that the published versions of Figs. 2, 7 and 8 contained incorrect western bands. In examining their raw data, the authors realized that they had used the fibroblasts of keloids [high expression of alpha-smooth muscle actin (α-SMA)] instead of adult dermal fibroblasts (low expression of α-SMA) in certain experiments. Note that no significant differences in morphology exist between myofibroblasts (from keloids) and fibroblasts (from normal dermal tissue). These errors were brought to light since the authors identified that the expression of α-SMA in Fig. 8 was higher compared with that in Fig. 4. After careful scrutiny, they established that the first author, Bin Zhao, who performed the experiments and analyzed the data shown in Figs. 2, 7 and 8, had mislabelled the myofibroblasts as fibroblasts. However, for all the other experiments in the above-mentioned article, the cells had been used correctly.

The authors regret that these errors were featured in the above-mentioned article, which may possibly have caused confusion for the readers, and the corrected versions of Figs. 2, 7 and 8 are shown opposite and on the next page. These changes did not affect either the results or the conclusions reported in this paper. The authors apologize to the Editor of *International Journal of Molecular Medicine* and to the readership for any inconvenience caused.

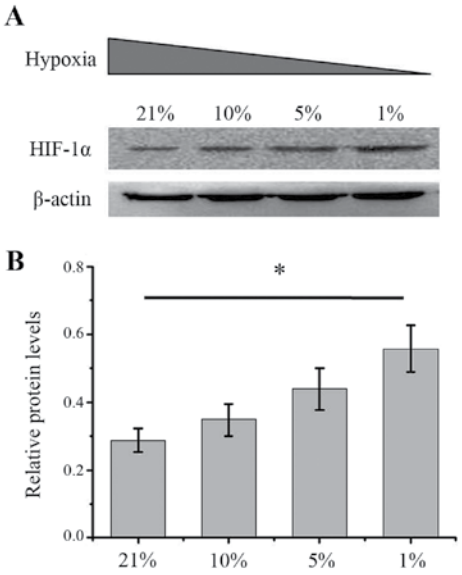

Figure 2. The expression of HIF-1α in dermal fibroblasts under different hypoxic conditions. (A) The protein expression of HIF-1α in dermal fibroblasts cultured under normoxic [21% oxygen (O_2)] and hypoxic (10%, 5% and 1% O_2) conditions for 48 h. (B) Densitometric analysis of HIF-1α expression. *P<0.05, n=3. HIF-1α, hypoxia-inducible factor.

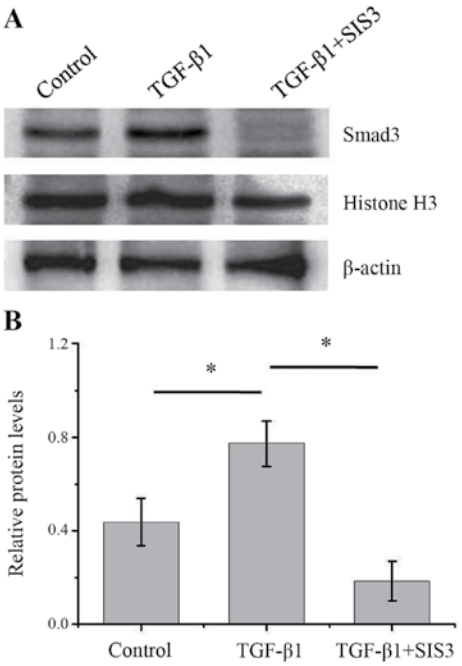

Figure 7. Changes in protein expression during the TGF-β1-induced conversion of fibroblasts into myofibroblasts, inhibited by the specific inhibitor of Smad3, SIS3. (A) Nuclear fractions were obtained from i) non-treated fibroblasts and ii) fibroblasts treated with 10 ng/ml TGF-β1 in the presence or absence of 5 μM SIS3 for 48 h, and expression of Smad3 was analyzed. Representative western blots showing the detection of Smad3 in the nucleus are shown. (B) Densitometric analysis of Smad3 in the presence or absence of 5 μM SIS3. Protein levels were normalized against histone H3 for nuclear fractions. *P<0.05, n=3. TGF-β1, transforming growth factor.

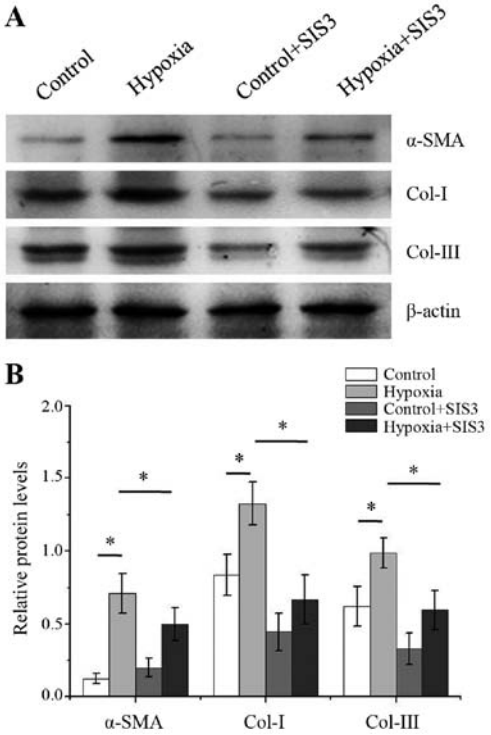

Figure 8. Changes in protein expression during the hypoxia-induced conversion of fibroblasts into myofibroblasts, inhibited by the specific inhibitor of Smad3, SIS3. (A) Fibroblasts were incubated in the absence or presence of 1% hypoxia for 48 h and treated in the absence or presence of 5 µM SIS3. The protein expression of α-SMA, Col-I and Col-III was then analyzed. (B) Densitometric analysis of α-SMA, Col-I and Col-III expression. *P<0.05, n=3. α-SMA, alpha-smooth muscle actin.