

CORRECTION

Correction: Induction of Connective Tissue Growth Factor Expression by Hypoxia in Human Lung Fibroblasts via the MEKK1/MEK1/ERK1/GLI-1/GLI-2 and AP-1 Pathways

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The α -tubulin displayed in the bottom panel of [Fig 4C](#) is incorrect. Please see the complete correct [Fig 4](#) here.



OPEN ACCESS

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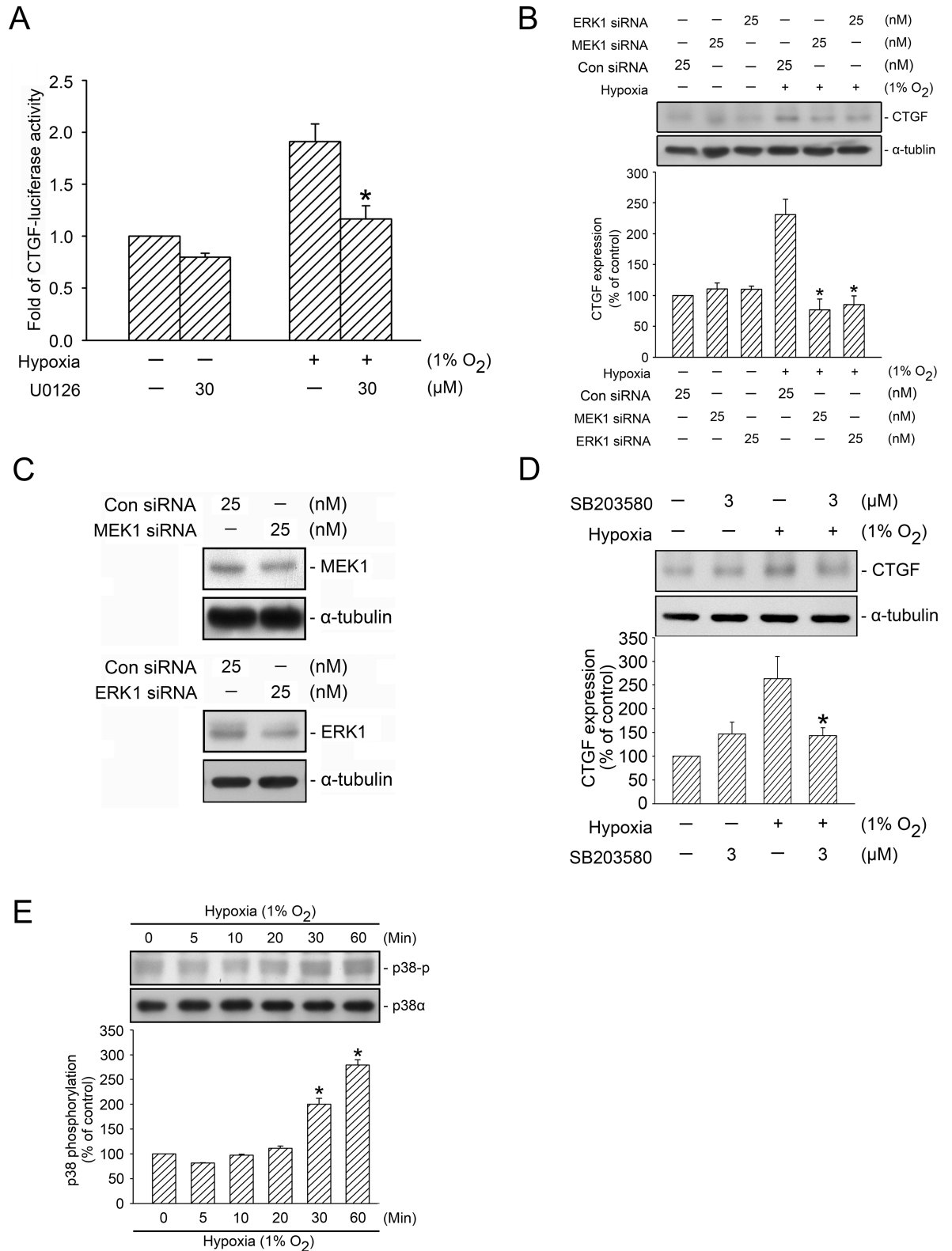


Fig 4. Involvement of ERK and p38 MAPK activation in hypoxia-induced CTGF expression. A, WI-38 cells were transfected with 0.5 μg of CTGF-Luc and 0.1 μg of pBK-CMV-Lac Z for 24 h. The cells were treated with U0126 before they were subjected to hypoxia (1% O₂) for an additional 24 h. The luciferase activity assay is described in the “Material and Methods” section. The results are expressed as the mean ± SEM of three independent experiments performed in duplicate. * *p* < 0.05,

compared with the hypoxia group without U0126 treatment. B, WI-38 cells were transfected with control siRNA (con siRNA), MEK1 siRNA, and ERK1 siRNA. After 24 h, the cells were subjected to hypoxia for an additional 24 h. CTGF and α -tubulin levels were detected using western blotting, as described previously. The results are expressed as the mean \pm SEM of three independent experiments. * $p < 0.05$, compared with hypoxia plus the control siRNA group. C, WI-38 cells were transfected with control siRNA (con siRNA), MEK1 siRNA, and ERK1 siRNA. After 24 h of transfection, MEK1, ERK1, and α -tubulin levels were detected using western blotting. Typical traces represent three independent experiments that yield similar results. D, WI-38 cells were pretreated with SB203580 for 20 min before they were subjected to hypoxia (1% O₂) for an additional 24 h. CTGF and α -tubulin levels in the cell lysates were detected using western blotting. The results are expressed as the mean \pm SEM of five independent experiments. * $p < 0.05$, compared with the hypoxia group without SB203580 treatment. E, WI-38 cells were subjected to hypoxia (1% O₂) for the indicated time intervals, after which the levels of p38 phosphorylation and p38 α were detected using western blotting. The results are expressed as the mean \pm SEM of three independent experiments. * $p < 0.05$, compared with the control group without hypoxia treatment.

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In Fig 5D, the row containing Lamin A/C incorrectly appears twice. Please see the complete correct Fig 5 here.

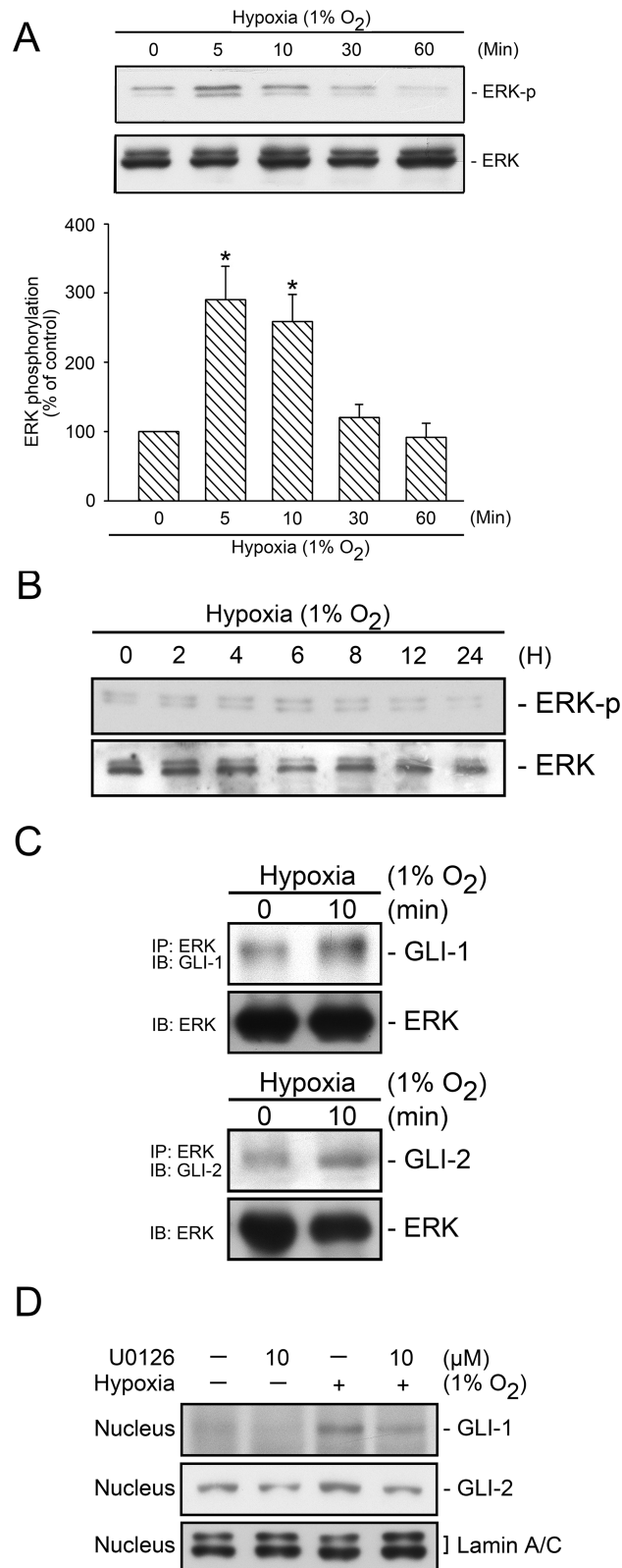


Fig 5. Involvement of ERK in hypoxia-induced GLI-1 and GLI-2 translocation. WI-38 Cells were subjected to hypoxia (1% O₂) for a time interval of 0–60 min (A) or 2–24 h (B), after which the levels of ERK phosphorylation and ERK in cell lysates were detected using western blotting. The results are expressed as the mean ± SEM of three and four independent experiments. * $p < 0.05$, compared with the control group without hypoxia treatment. C, WI-38 cells were subjected to hypoxia (1% O₂) for 10 min, before they were immunoprecipitated with a specific ERK antibody and mag sepharose magnetic beads, as described in the “Materials and methods” section. After elution from the beads, the levels of GLI-1, GLI-2, and ERK in the lysates were detected using western blotting. Typical traces represent three independent experiments that yield similar results. D, WI-38 cells were pretreated with U0126 for 20 min before they were subjected to hypoxia (1% O₂) for an additional 2 h, after which nuclear protein was collected. The levels of GLI-1, GLI-2, and lamin A/C in the nuclear extract were detected using western blotting. Typical traces represent three independent experiments that yield similar results.

<https://doi.org/10.1371/journal.pone.0188608.g002>

Reference

1. Cheng Y, Lin C-h, Chen J-Y, Li C-H, Liu Y-T, Chen B-C (2016) Induction of Connective Tissue Growth Factor Expression by Hypoxia in Human Lung Fibroblasts via the MEKK1/MEK1/ERK1/GLI-1/GLI-2 and AP-1 Pathways. PLoS ONE 11(8): e0160593. <https://doi.org/10.1371/journal.pone.0160593> PMID: 27486656