ERRATUM

PR39, a peptide regulator of angiogenesis

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Nature Medicine 6, 49-55 (2000)

On page 51, Fig. 2a,c and d should be as presented here:

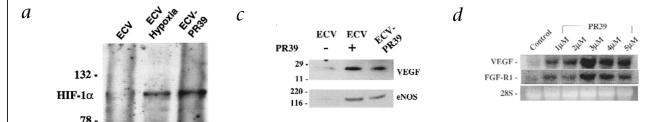


Fig. 2 PR39 induces HIF- 1α and HIF- 1α -dependent gene expression. a, HIF- 1α protein levels in ECV cells cultured in normoxic (ECV) or hypoxic conditions (ECV Hypoxia) or stably transfected with the PR39 expression construct (ECV–PR39). Proteins immunoprecipitated with an antibody against HIF- 1α were assessed by western blot analysis with an antibody against HIF- 1α . There is increased HIF- 1α expression

in hypoxic ECV and PR39-transfected cells. \emph{c} , Western blot analysis of VEGF (top) and NOS3 (eNOS; bottom) in ECV cells cultured for 24 h in the absence (–) or presence (+) of 1 μ M PR39, or in the ECV–PR39 cell line. \emph{d} , Northern blot analysis of VEGF and FGFR1 levels in PR39-treated bovine aortic endothelial cells, showing a dose-dependent response.

On page 52, Fig. 3c should be as presented here:

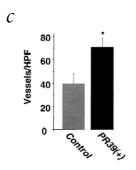


Fig. 3c, Quantitative morphometric analyses of microvessels in α MHC–PR39 mice (right) and control mice (left); *, P < 0.001, compared with control.

On page 53, Fig. 5b should be as presented here:

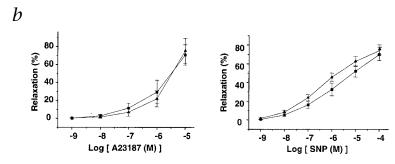


Fig. 5 Physiologic evaluation of PR39-induced angiogenesis b, Analysis of microvessels from α MHC–PR39 mice (\blacksquare) and control mice (\triangle), showing similar vessel reactivity to the endothelium-dependent vasodilator A23187 (left) and the smooth muscle relaxant sodium nitroprusside (SNP; right).

We regret these errors.