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Perlecan domain V is neuroprotective and proangiogenic following ischemic stroke in rodents

Boyeon Lee, … , Sarah A. Thomas, Gregory J. Bix

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Corrigendum

Original citation: J. Clin. Invest. 2011;121(8):3005–3023. doi:10.1172/JCI46358. Citation for this corrigendum: J. Clin. Invest. 2012;122(2):777. doi:10.1172/JCI61899. In the legends for Figures 3, 5, and 9, it was not explicit that the same data were used for multiple figures. The correct figure legends appear below. Figure 3 DV is neuroprotective. (A and B) Mean ischemic lesion volumes measured from brain sections stained with TTC (PSD 1–3) or H&E (PSD 7 and 15) in WT mice treated with different doses of DV (A) or in WT and Pln–/– mice treated as indicated (B) (*$P < 0.05$, **$P < 0.01$, $n = 15$ per treatment group per PSD). Mean ischemic lesion volumes for WT and Pln–/– on PSD 1–3 are repeated from Figure 1E, as these volumes were obtained in the same experiments as the volumes obtained with DV treatment of WT and Pln–/– mice. (C) WT or Pln–/– mouse brain TTC staining at PSD 3, or WT H&E staining on PSD 15, after animals received i.p. PBS or DV injections (1 mg/kg). Yellow asterisks and red circles indicate ischemic lesions. PBS-treated WT and Pln–/– brain images are repeated here from those shown in Figure 1F, as they were obtained from the same experiments shown in that figure. (D) Cresyl violet, cleaved caspase-3, and TUNEL staining, with propidium iodide (PI) nuclear counterstain, in […]

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The correct figure legends appear below.

Figure 3
DV is neuroprotective. (**A** and **B**) Mean ischemic lesion volumes measured from brain sections stained with TTC (PSD 1–3) or H&E (PSD 7 and 15) in WT mice treated with different doses of DV (**A**) or in WT and *Pln*$^{-/-}$ mice treated as indicated (**B**) (*$P < 0.05$, **$P < 0.01$, $n = 15$ per treatment group per PSD). Mean ischemic lesion volumes for WT and *Pln*$^{-/-}$ on PSD 1–3 are repeated from Figure 1E, as these volumes were obtained in the same experiments as the volumes obtained with DV treatment of WT and *Pln*$^{-/-}$ mice. (**C**) WT or *Pln*$^{-/-}$ mouse brain TTC staining at PSD 3, or WT H&E staining on PSD 15, after animals received i.p. PBS or DV injections (1 mg/kg). Yellow asterisks and red circles indicate ischemic lesions. PBS-treated WT and *Pln*$^{-/-}$ brain images are repeated here from those shown in Figure 1F, as they were obtained from the same experiments shown in that figure. (**D**) Cresyl violet, cleaved caspase-3, and TUNEL staining, with propidium iodide (PI) nuclear counterstain, in the peri-infarct area in WT mice treated with PBS or with DV. Scale bars: 5 μm (cresyl violet) and 10 μm (caspase-3 and TUNEL).

Figure 5
DV neuroprotection is VEGF and VEGFR mediated. (**A**) Anti-VEGF Western blot analysis of ipsilateral stroke hemispheres as labeled, with GAPDH as internal loading control. (**B**) Densitometry analysis of VEGF Western blot as shown in **A** as normalized to corresponding GAPDH bands (**$P < 0.01$, $n = 15$ per treatment group, per PSD). (**C**) Plot of VEGF ELISA ipsilateral stroke brain tissue treated as labeled ($^{\#}P < 0.01$ as compared with corresponding PBS-treated WT control or as labeled, $n = 3$ per treatment group per PSD). (**D**) Mean ischemic lesion volumes of stroke WT mice on PSD 1–3 treated as labeled (**$P < 0.01$, $n = 15$ per treatment group per PSD). (**E**) Vibrissae-elicited forelimb placement test on WT mice treated as labeled. DV had no effect in animals also treated with PTK787/ZK 222584 (P = NS). Stroke PBS and stroke DV (1 mg/kg) results are repeated from the identically labeled data in Figure 4B, as these experiments were performed in parallel using the same groups for comparison. (**F**) NeuN and VEGFR2 co-immunohistochemistry of PSD 5 peri-infarct brain tissue of mice treated as labeled. White arrows indicate cells that were positive for both NeuN and VEGFR2. Scale bar: 50 μm. (**G**) Number of NeuN- and VEGFR2-positive cells per mm^2 in the peri-infarct regions as labeled (**$P < 0.01$, $n = 10$ images per animal, 5 animals per treatment condition).

Figure 9
DV effects are mediated via the α5β1 integrin in vivo. (**A**) Anti-α5β1 Western blot analysis from PSD 3 mouse brain tissue treated as labeled, with GAPDH as internal control. (**B**) α5β1 immunohistochemistry of mouse PSD 3 peri-infarct brain tissue with or without DV treatment. Scale bar: 10 μm. (**C**) Quantification of mean ischemic lesion volumes of stroke WT mice on PSD 1–3 as labeled (*$P < 0.05$, $n = 15$ per treatment condition per PSD). (**D**) Cresyl violet staining, caspase-3 17- to 20-kDa cleavage product immunostaining, and TUNEL staining with PI of peri-infarct brain regions as labeled. Scale bars: 10 μm. (**E**) Vibrissae-elicited forelimb placement test on WT mice treated as labeled ($n = 15$ mice per condition from 3 separate experiments with 5 mice each). WT and WT + DV values are repeated here from Figure 5E (there labeled stroke PBS and stroke DV, 1 mg/kg), as these experiments were all performed in parallel using the same groups for comparison. (**F**) Von Willebrand factor immunohistochemistry (green) on PSD 5 from WT mice treated as labeled. Scale bar: 10 μm. Representative WT stroke + PBS and WT stroke + DV images are repeated here from the identically labeled images in Figure 8A, to ease their visual comparison with animals treated with α5 antibody or DV + α5 antibody. (**G**) Peri-infarct blood vessel quantification as labeled (*$P < 0.05$, **$P < 0.01$ compared with PBS + IgG on the same day, $n = 20$ images analyzed per animal, 10 animals per experimental condition). (**H**) Anti-VEGF Western blot analysis of mouse stroke hemispheres with internal GAPDH as control. (**I**) Optical density quantification of VEGF Western blot analysis as shown in **H** (**$P < 0.01$, $n = 5$ per experimental condition).

The authors regret the error.