



Corrigendum

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

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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 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 mm² 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.