Chen et al., http://www.jcb.org/cgi/content/full/jcb.201212032/DC1

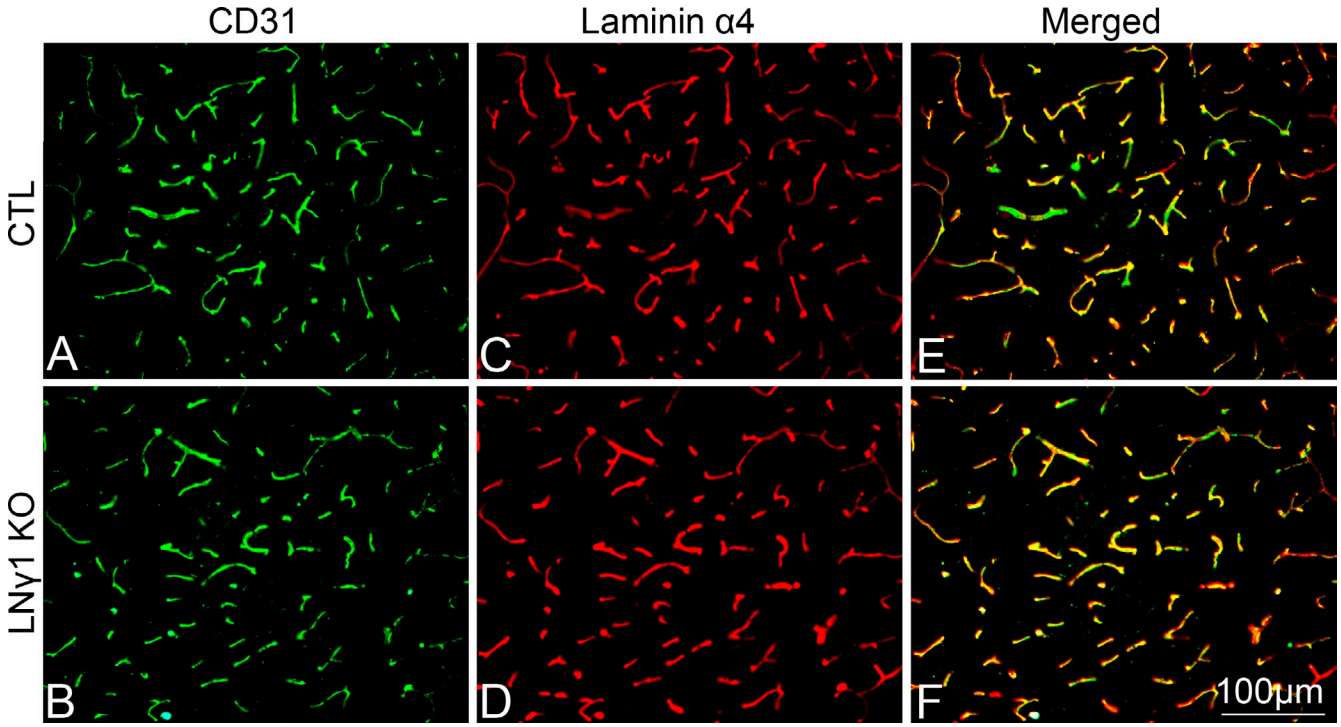

Figure S1. **All endothelial cells express laminin α4 in both control and LNγ1-KO mice.** Brain sections from control and LNγ1-KO mice were stained for CD31 (A and B) and laminin α4 (C and D), and the images were merged (E and F). All CD31staining colocalized with laminin α4 staining, indicating all endothelial cells express laminin α4 in both control and LNγ1-KO mice. Bar, 100 μm.

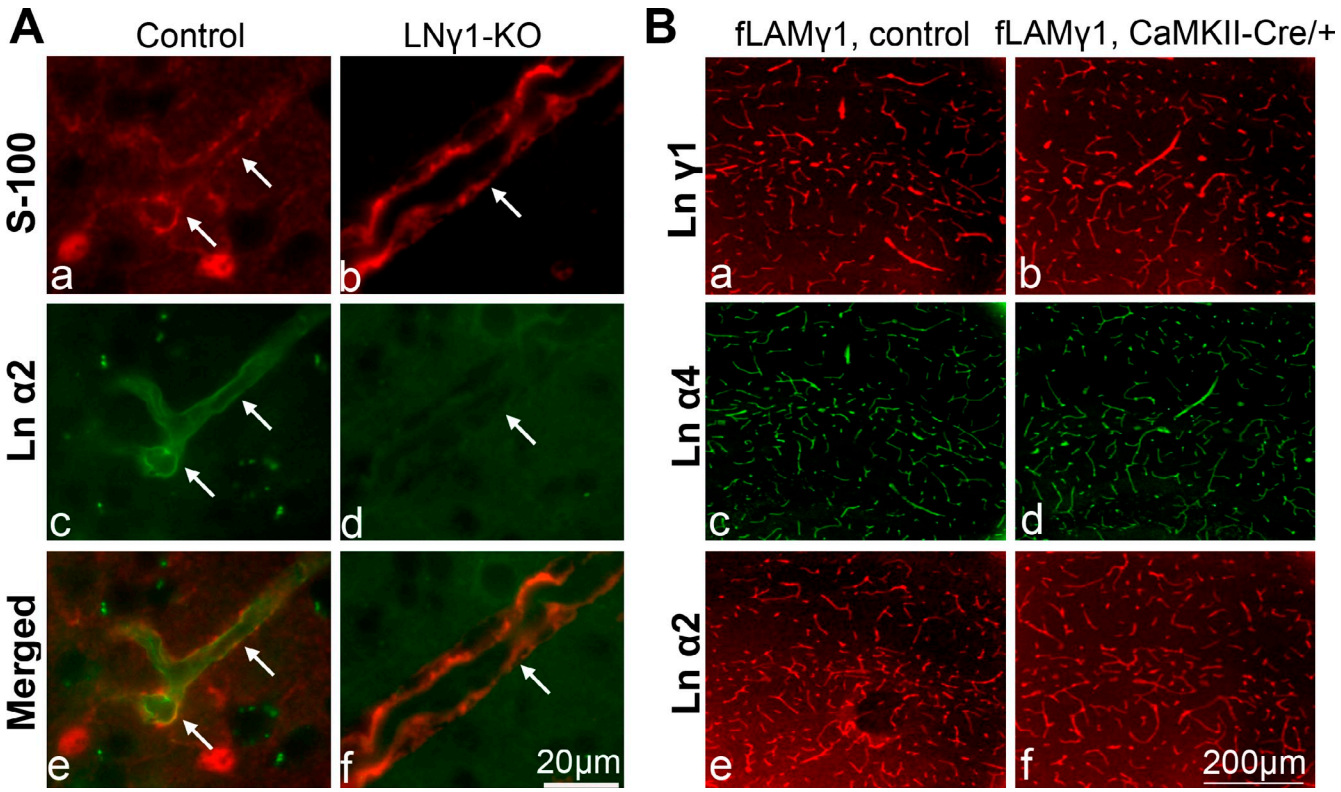

Figure S2. **Disruption of laminin α2 expression at the astrocytic endfeet of LNγ1-KO mice and neuronal laminin does not contribute to the vascular matrix.** (A) Brain sections from control and LNγ1-KO mice were stained for S-100 to identify astrocytic endfeet (Aa and Ab, arrows) or laminin α2 (Ac and Ad, arrows), and the images were merged (Ae and Af). Astrocytic endfeet expressed laminin α2 in control mice (Ae), but endfeet of LNγ1-KO mice did not (Af). Bar, 20 μm. (B) Brain sections from control mice and mice deficient in laminin by the CaMKII-Cre promoter were stained for laminin γ1 (Ba and Bb), laminin α4 (Bc and Bd), and laminin α2 (Be and Bf). All laminin chains were expressed similarly between control and knockout mice. Therefore, disruption of laminin α1 and α2 expression in LNγ1-KO mice is due to ablation of laminin γ1 in astrocytes, and not due to lack of laminin γ1 in neurons or endothelial cells. Bar, 200 μm.

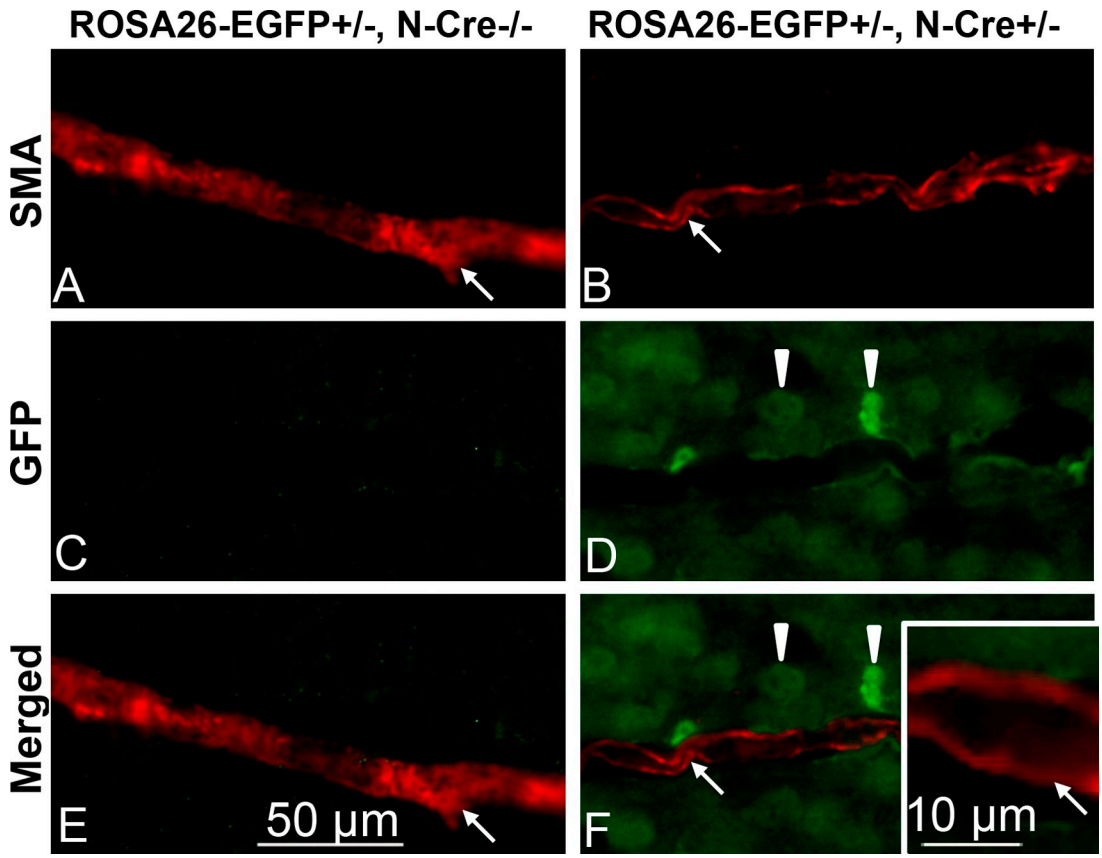

Figure S3. **Cre expression patterns in nestin-Cre mouse line.** Brain sections were stained for SMA (A and B) and EGFP (C and D), and the images were merged (E and F). EGFP was not expressed in brain tissues of ROSA26-EGFP$^{+/-}$:nestin-Cre$^{-/-}$ mice (C). It was expressed in the brain parenchyma cells (astrocytes and neurons, and arrowheads in D and F) but not VSMCs (arrows in F) of ROSA26-EGFP$^{+/-}$:nestin-Cre$^{+/-}$ mice, indicating Cre is active in astrocytes and neurons, but not VSMCs in the nestin-Cre mouse line. Inset in F shows higher magnification of the images. Bar, 50 μm

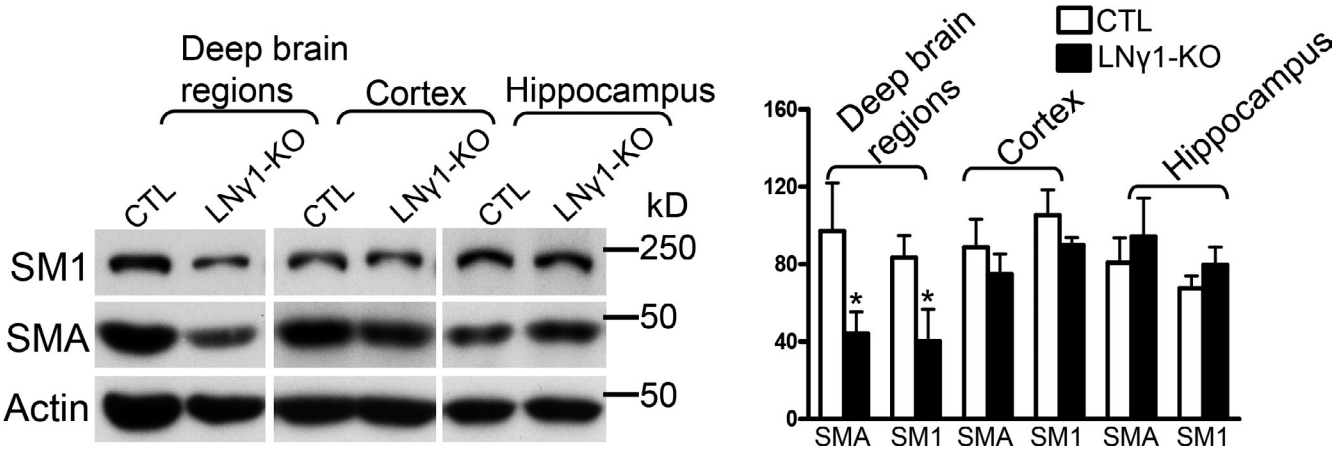

Figure S4. **Region-specific VSMC contractile protein expression changes in LNγ1-KO mice during development.** Western blot analysis showed that at postnatal day 28 expression levels of SMA and SM1 were significantly decreased in deep cerebral regions of LNγ1-KO mice compared with controls. SMA and SM1 expression in cerebral cortex and hippocampus of LNγ1-KO mice were not significantly changed compared with the same regions of the control mice.

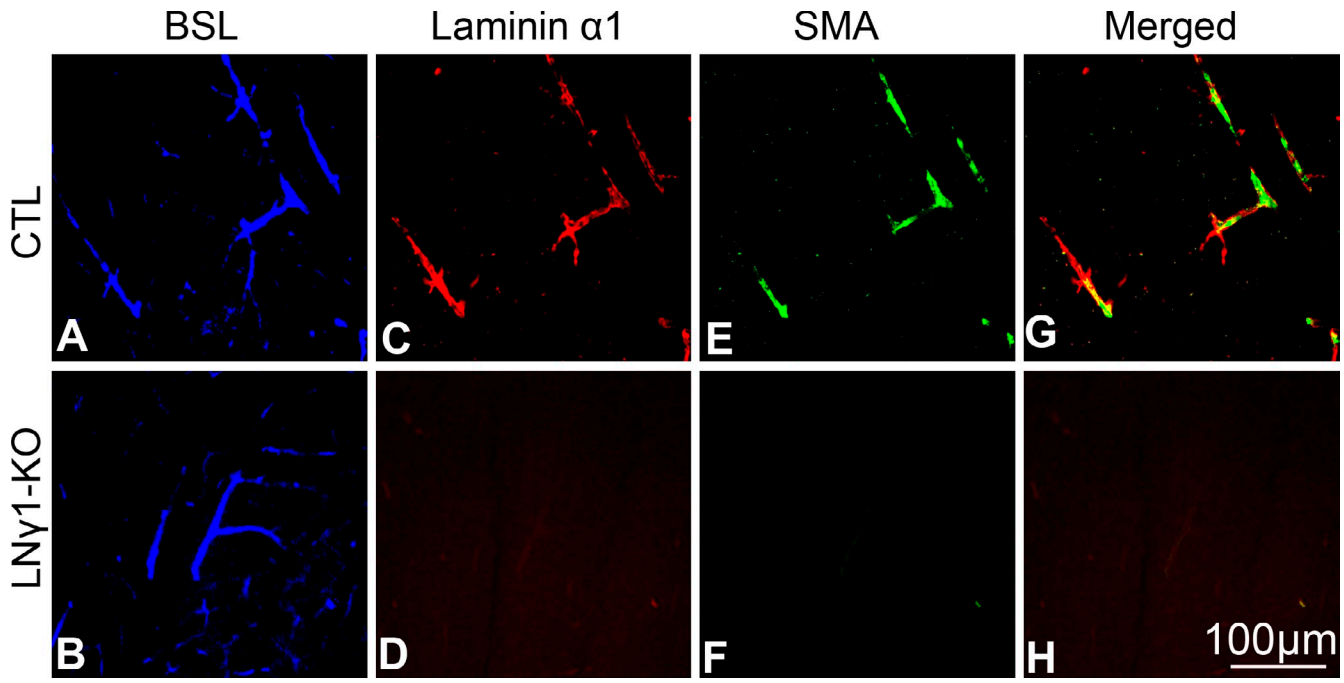

Figure S5. **Relationship between astrocytic laminin α1 and SMA expression.** Brain sections from control and LNγ1-KO mice were stained for BSL to identify large caliber blood vessels (A and B), laminin α1 (C and D), or SMA (E and F). Images of laminin α1 and SMA were merged (G and H). In control mice, laminin α1 expression was mostly associated with large blood vessels (A vs. C) and colocalized with SMA expression (C, E, and G). However, expression levels of laminin α1 (D) and SMA (F) in LNγ1-KO mouse brain were both dramatically decreased (D, F, and H). Bar, 100 μm.