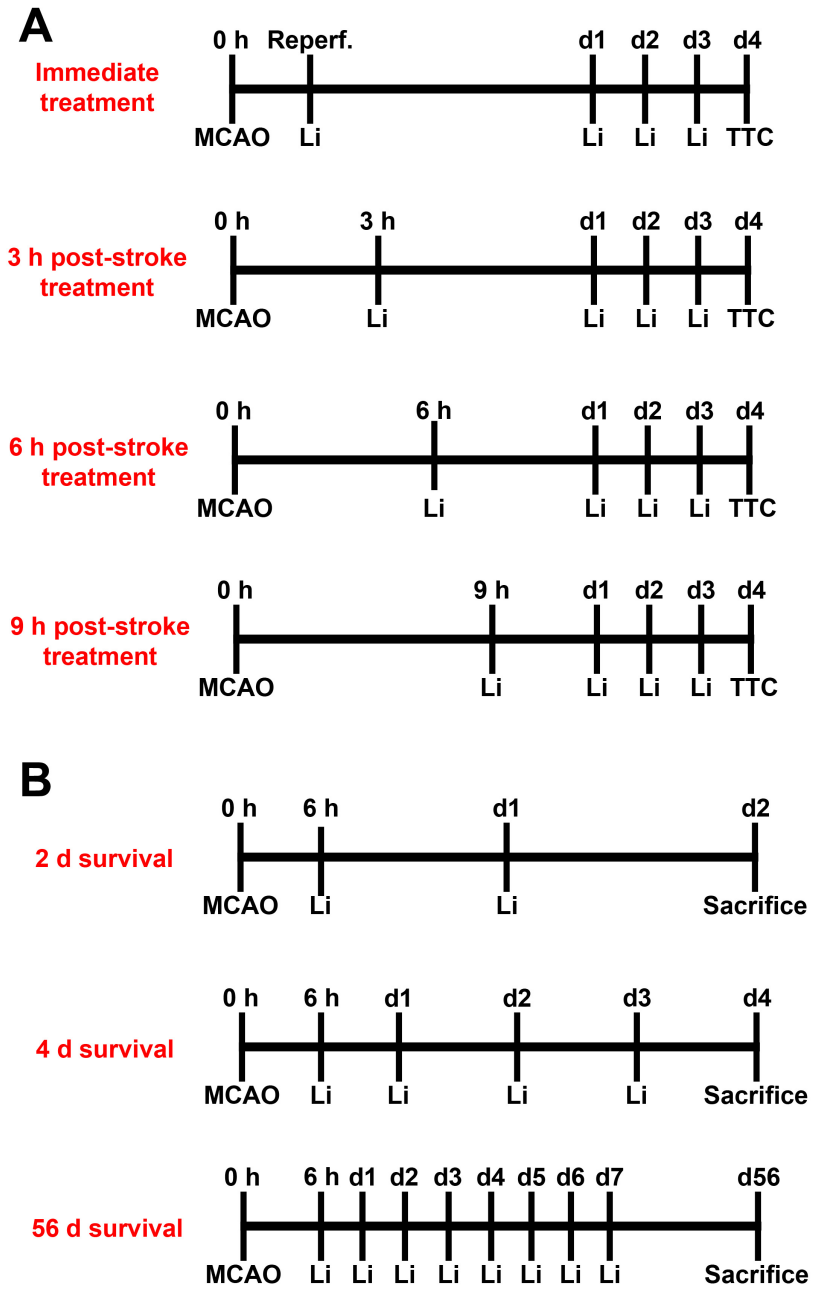
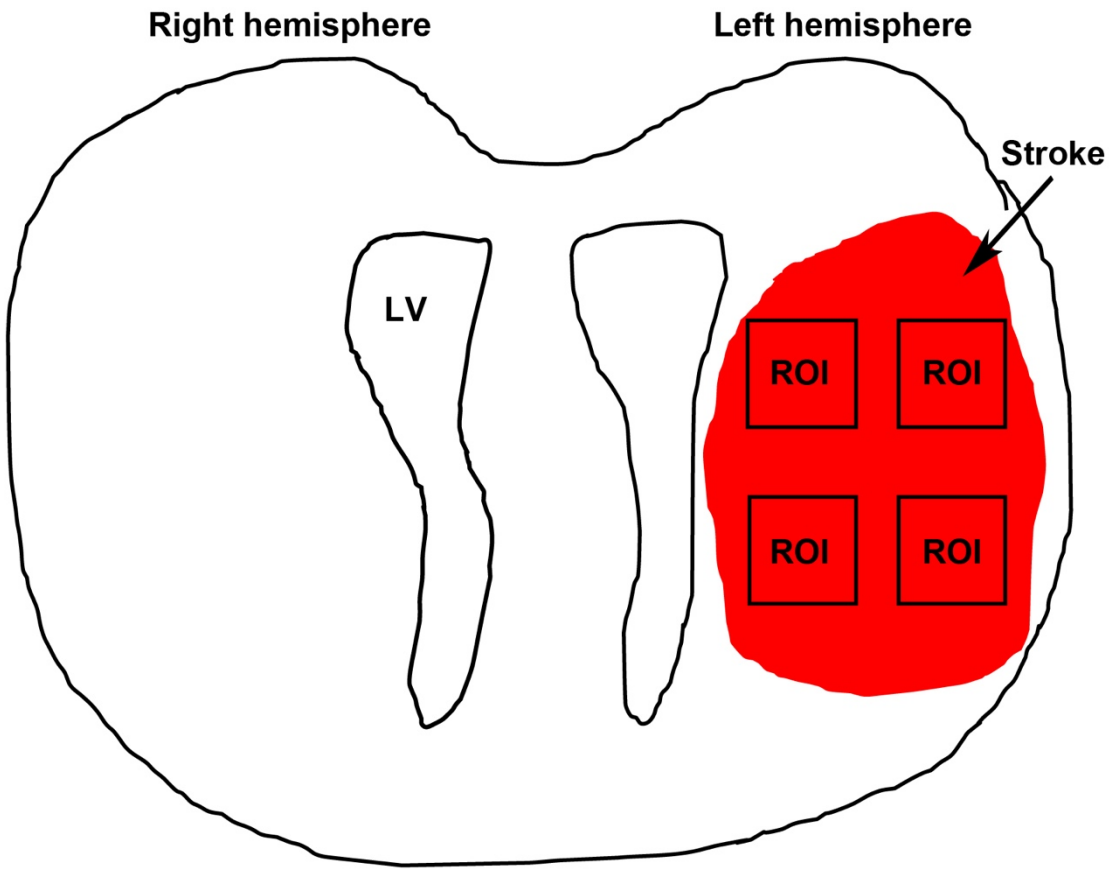
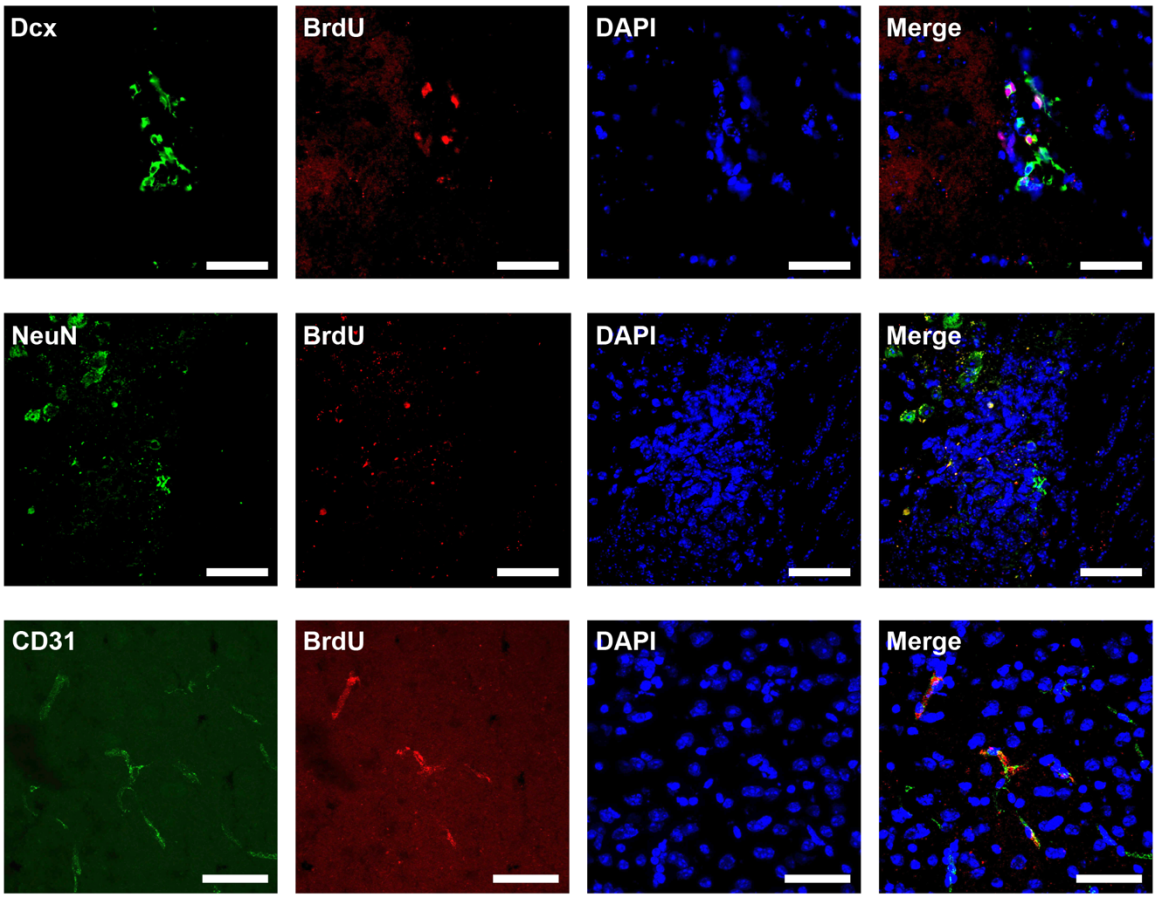


Suppl. Fig. S1. Lithium treatment paradigm. Mice were exposed to 45 min of middle cerebral artery occlusion (MCAO) followed by intraperitoneal delivery of lithium (Li). First delivery of was always at dose of 1 mmol/kg, whereas the following ones were done with dosages of 2 mmol/kg. **(A)** Assessment of therapeutic time window of lithium. Infarct volume analysis was done on day 4 using TTC staining. Only the timing of the first injection was changed with constant delivery protocols for the remaining injections. **(B)** Different injection protocols for the three survival periods. The first injection was always performed at 6 h post-stroke, whereas the other injection paradigms were changed depending on animal survival.



Suppl. Fig. S2. Definition of regions of interest (ROI). Cerebral ischemia was induced after insertion of a silicon-coated nylon monofilament into the left middle cerebral artery. The filament was removed after 45 min to allow reperfusion. Immunohistochemical analyses were performed by defining ROIs within the ischemic hemisphere at 0.14 mm anterior, 2.5-3.25 mm ventral and 1.5-2.25 mm lateral to bregma. LV: lateral ventricle. For further information, please refer to the materials and method section of the main manuscript.



Suppl. Fig. S3. Post-stroke stimulation of angioneurogenesis by lithium. Additional photographs for co-localizations between the neural markers Dcx/NeuN and the endothelial marker CD31. Photographs depicted are taken from mice that underwent the same experimental procedure as described in Fig. 5. Scale bars: 50 µm.