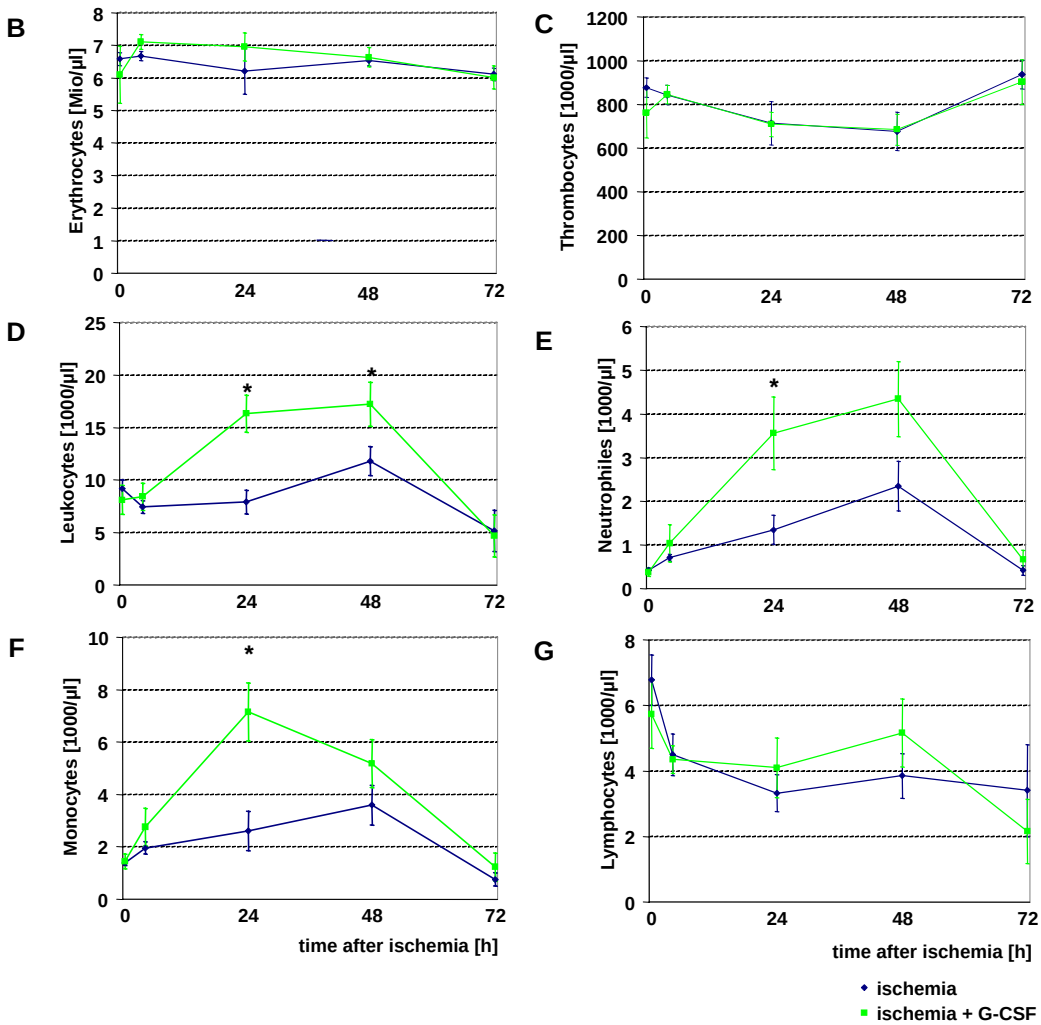
A

MCAO

time after MCAO	Hb [g/dl]	Ery [x10⁶/µl]	Hk	MCV [fl]	MCH [pg]	Thrombo [1000/µl]	Leuko [1000/µl]	Neutro [1000/µl]	Lymph [1000/µl]	Mono [1000/µl]
0h	13.23 +/- 0.41	6.58 +/- 0.19	0.40 +/- 0.01	60.67 +/- 0.76	20.17 +/- 0.32	877.33 +/- 43.32	9.18 +/- 0.83	0.41 +/- 0.06	6.78 +/- 0.78	1.39 +/- 0.09
4h	13.45 +/- 0.33	6.68 +/- 0.14	0.40 +/- 0.01	60.08 +/- 0.73	20.25 +/- 0.30	843.58 +/- 44.15	7.44 +/- 0.61	0.71 +/- 0.08	4.50 +/- 0.63	1.96 +/- 0.23
24h	13.50 +/- 0.32	6.20 +/- 0.71	0.36 +/- 0.04	52.20 +/- 5.89	17.70 +/- 1.99	714.00 +/- 98.52	7.91 +/- 1.10	1.34 +/- 0.33	3.32 +/- 0.57	2.61 +/- 0.75
48h	12.89 +/- 0.44	6.54 +/- 0.16	0.38 +/- 0.01	58.10 +/- 1.25	19.70 +/- 0.33	676.30 +/- 88.28	11.82 +/- 1.39	2.35 +/- 0.56	3.85 +/- 0.68	3.60 +/- 0.76
72h	11.97 +/- 0.43	6.11 +/- 0.19	0.37 +/- 0.01	60.83 +/- 0.63	19.67 +/- 0.28	937.75 +/- 65.16	5.15 +/- 1.97	0.41 +/- 0.11	3.40 +/- 1.40	0.76 +/- 0.25

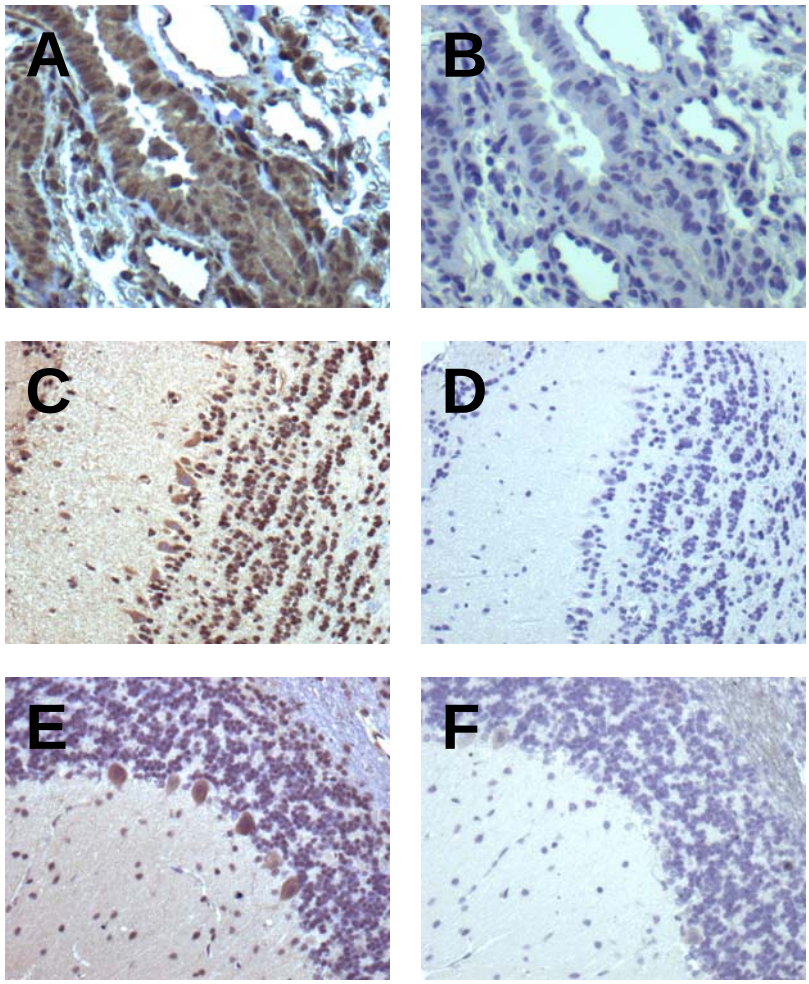
MCAO + G-CSF

time after MCAO	Hb [g/dl]	Ery [x10⁶/µl]	Hk	MCV [fl]	MCH [pg]	Thrombo [1000/µl]	Leuko [1000/µl]	Neutro [1000/µl]	Lymph [1000/µl]	Mono [1000/µl]
0h	13.79 +/- 0.22	6.10 +/- 0.88	0.36 +/- 0.05	51.63 +/- 7.40	17.38 +/- 2.50	761.75 +/- 114.72	8.12 +/- 1.39	0.38 +/- 0.09	5.73 +/- 1.03	1.44 +/- 0.28
4h	14.09 +/- 0.29	7.11 +/- 0.23	0.42 +/- 0.01	59.22 +/- 0.94	19.89 +/- 0.35	845.00 +/- 42.63	8.41 +/- 1.29	1.04 +/- 0.43	4.35 +/- 0.42	2.75 +/- 0.71
24h	13.53 +/- 0.67	6.96 +/- 0.43	0.40 +/- 0.02	58.14 +/- 1.40	19.57 +/- 0.37	709.00 +/- 55.85	16.33 +/- 1.75	3.56 +/- 0.83	4.09 +/- 0.92	7.15 +/- 1.10
48h	12.86 +/- 0.48	6.63 +/- 0.29	0.38 +/- 0.01	58.33 +/- 1.13	19.67 +/- 0.24	684.56 +/- 70.02	17.22 +/- 2.09	4.34 +/- 0.86	5.16 +/- 1.04	5.17 +/- 0.92
72h	11.61 +/- 0.62	6.01 +/- 0.35	0.36 +/- 0.02	60.33 +/- 0.73	19.56 +/- 0.29	902.33 +/- 102.49	4.69 +/- 2.01	0.66 +/- 0.22	2.16 +/- 0.98	1.23 +/- 0.53

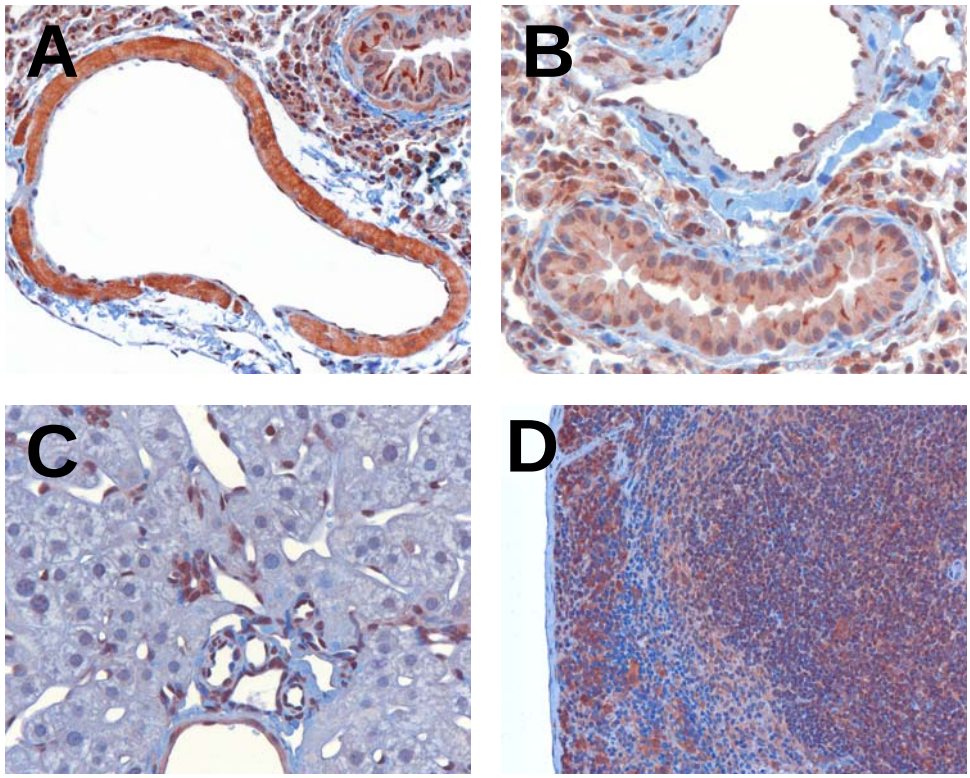


Supplementary figure 1: Blood parameters after G-CSF treatment in rats subjected to MCAO.
Rats were subjected to 90 min intraluminal filament occlusion. G-CSF was applied at 60 µg/kg bodyweight 30 min following onset of ischemia as i.v. bolus. 2 additional G-CSF injections [30 µg/kg bodyweight] were given subcutaneously at day 2 and 3. 72 h after MCAO occlusion, the last blood sample was drawn, and animals were sacrificed. A, We determined total hemoglobin (Hb), erythrocyte count (Ery), hematokrit (Hk), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), thrombocyte count (Thrombo), leukocyte count (Leuko), and differential white blood cell count (neutrophils, lypmphocytes, monocytes, eosinophils). B-G, Ischemic animals developed a rise in total leukocytes (D), neutrophils (E), and monocytes (F), that was increased by G-CSF treatment. Parameters that were significantly different in the G-CSF treated animals were total leukocytes at 24 and 48 h following onset of occlusion, neutrophils at 24 h, and monocytes at 24 h. Maximum differences evoked by G-CSF were seen at 24 h, where total leukocytes and neutrophils doubled, while monocytes almost tripled. Interestingly, a steep decline of total leukocytes, monocytes, and neutrophils was noted after 72 h, that occurred both in G-CSF-treated animals and controls, suggesting normalization of ischemia-induced leukocyte elevation. Of note, there was a temporary drop in thrombocyte count at 24 and 48 h following ischemia in both groups, that normalized on day 3.

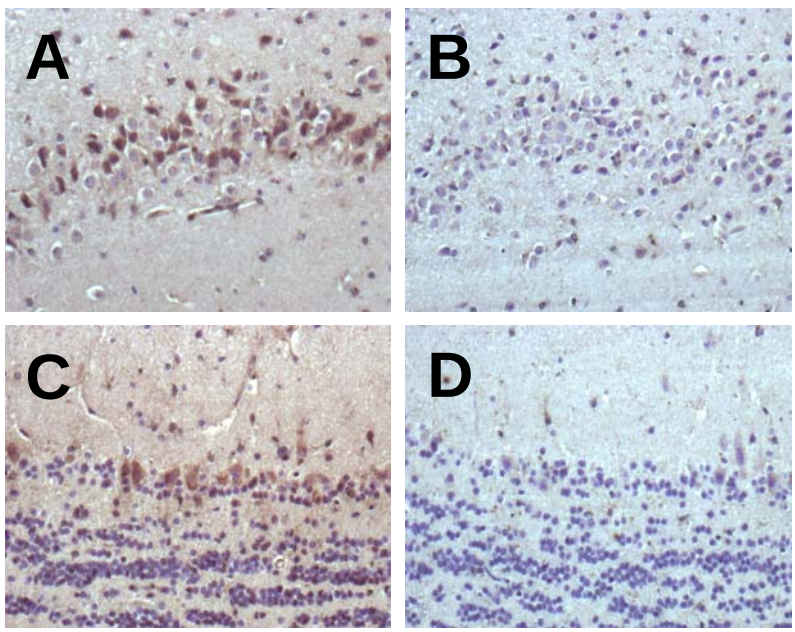
Supplementary figure 1



Supplementary figure 2: Specificity of immunohistochemical stainings with the G-CSF receptor antibody demonstrated by the use of a blocking peptide in neural and extraneural tissues of mouse and rat. 2 µm paraffin sections were stained with the G-CSF receptor antibody (**A,C,E**), and after pre-incubation with the appropriate blocking peptide (**B,D,F**). **A,B** mouse lung with positvie staining in the endothelium, smooth muscle and epithelial cells. **C,D** mouse olfactory bulb with strong staining in the mitral cells. **E,F** rat cerebellum with prominent immunopositivity of Purkinje cells.

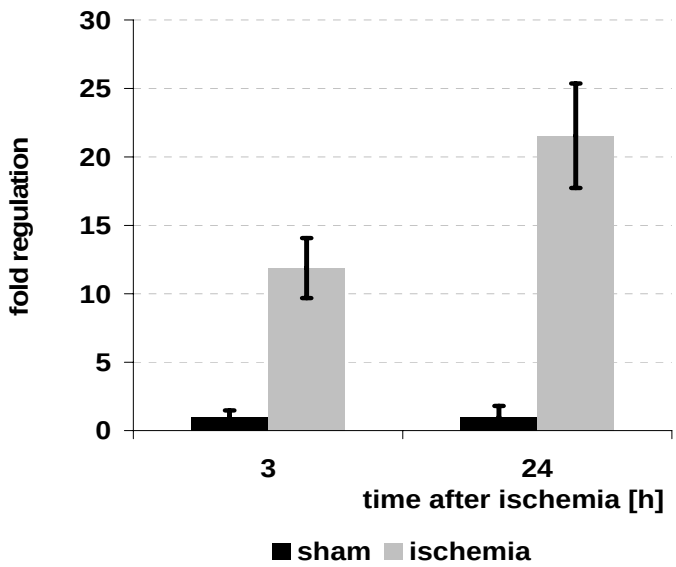


Supplementary figure 3: Positive control stainings in various mouse tissues known to express G-CSF.
A, Lung: Intrapulmonary blood vessel with a single disrupted outer elastica located in a distance to an airway indicating a pulmonary vein displays a strong immunostaining against G-CSF in the smooth muscle cells and in the endothelial cells. Moreover, immunostaining of G-CSF is found in distinct pneumocytes, in some blood derived cells, and with a spatial distribution in the surface epithelium of the bronchus. **B**, Lung: Intrapulmonary blood vessel with an internal and an external elastic membrane and accompanied by an airway indicative of an arteriole. Strong immunostaining against G-CSF is found in endothelial cells lining the luminal circumference, in distinct pneumocytes, in some blood derived cells, and occasionally in the surface epithelium of the bronchus. **C**, Liver: Tissue section of the liver displaying a portal tract with bile duct, hepatic arteriole, portale venule, and clearly defined limiting plate. Strong immunostaining against G-CSF is especially found in endothelial cells and in the tall columnar epithelial cells lining the bile duct. Immunostaining against G-CSF was occasionally found in smooth muscle cells of some blood vessels, but not in hepatocytes. **D**, Spleen: Histological section of spleen tissue comprising white and red pulp in a subcapsular position. Strong immunostainig against G-CSF is found in probably blood-derived cells in the red pulp and to a minor extent in flat endothelial cells of the sinus. There is some immunoreactivity against G-CSF in the germinal center of the B-cell follicle. In the T-cell area, some cells immunostained against G-CSF are occasionally found.



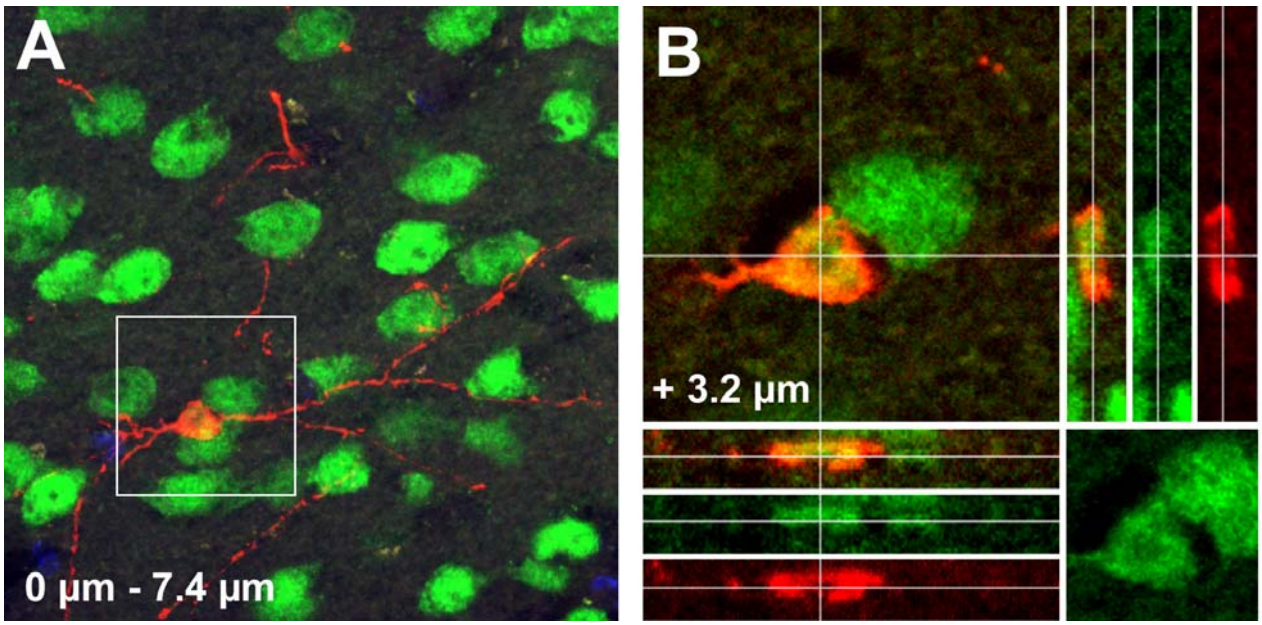
Supplementary figure 4: Specificity of immunohistochemical stainings with an unrelated antibody against G-CSF (sc-9351, Santa Cruz). Shown is the specific staining of GCSF in neurons of the CA3 region of the hippocampus (**A**) and in mitral cells of the olfactory bulb (**C**) in the rat brain. Staining was blocked by pre-incubating the antibody with the appropriate peptide (**B**, **D**, original magnification 20x).

Supplementary figure 4



Supplementary figure 5: G-CSF is induced in the cortex in a global ischemia model in rats.
Rats were subjected to a combined hypotonia and bilateral carotid occlusion. At 3 and 24 h following hypoxia, animals were perfused transcardially, and cortex dissected. 2-3 animals per group were pooled, and quantitative PCR performed as described.

Supplementary figure 5



Supplementary Figure 6: Colocalisation of the progenitor marker DCX (red) and the neuronal marker NeuN (green) indicates neuronal differentiation of newly generated cells. Confocal 3-D analysis conveys evidence for coexpression of both markers within single cells. A DCX-NeuN double-positive striatal cell is shown in (**A**) as a z-projection of 18 optical slices of 0.4µm thickness to show the complete morphology of the cell. **(B)** A single optical slice at the z coordinate +3.2µm as well as x-z and y-z projections demonstrate colocalization of immunofluorescence signals within the same cell.

Supplementary figure 6