phosphatase inhibitors (1:100; Sigma-Aldrich) were mixed with RIPA buffer (Sigma-Aldrich) to lyse the tissues. The ELx800G Universal Microplate Reader was used to determine the protein content (BioTek Instruments Inc.). Equal amounts of protein (30 μg per lane) were electrophoresed on 8% to 12% sodium dodecyl sulfate-polyacrylamide gels and transferred to polyvinylidene fluoride (PVDF) membranes. After blocking with 5% non-fat dried milk for 1.5 hours at room temperature, the membrane was incubated at 4 ℃ overnight with primary antibodies, p-eNOS (9571, 1:3,000; Cell Signaling), neurotrophin-3 (NT-3) (1:3,000; Santa Cruz), phospho-phosphoinositide-3-kinase (p-PI3K) (1:3,000; Cell Signaling), vascular endothelial growth factor (VEGF) (1:3,000; Santa Cruz), brain-derived neurotrophic factor (BDNF) (1:3,000; Santa Cruz), nerve growth factor (NGF) (1:3,000; Santa Cruz), angiopoietin-1 (ab8451, 1:3,000; Abcam), and glyceraldehyde-3-phosphate-dehydrogenase (GAPDH) (1:5,000; ABclonal), then washed five times with PBS-T. Thereafter, signals were visualized using the ECL detection system (Amersham Pharmacia Biotech, Piscataway).

Supplement Table 1. Physiologic and metabolic parameters: 2 weeks after PBM treatment

	Sham	CNI			
		Heat	RED	NIR	RED+NIR
Body weight (g)	26.03±0.51	27.10±0.94	26.59±1.79	27.62±0.96	25.82± 0.64
MSBP (cm H_2O)	79.21±3.46	82.81±2.14	76.14±2.06	80.41±2.16	75.76±2.14

Values are the mean±standard error of the mean for n=6 animals per group.
Body weight and MSBP measured two weeks post-PBM irradiation therapy with heat, RED, NIR, or RED+NIR for five consecutive days.
PBM: photobiomodulation, CNI: cavernous nerve injury, NIR: near-infrared, MSBP: mean systolic blood pressure.