Low-Level Laser Therapy Applied Transcranially to Rats After Induction of Stroke Significantly Reduces Long-Term Neurological Deficits

Amir Oron, MD; Uri Oron, PhD; Jieli Chen, MD; Anda Eilam, MD; Chunling Zhang, MD; Menachem Sadeh, MD; Yair Lampl, MD; Jackson Streeter, MD; Luis DeTaboada, MSEE Michael Chopp, PhD

From the Department of Orthopedics (A.O.), Assaf Harofeh Medical Center, Zerifin, Israel; Photothera Inc (U.O., J.S., L.D.), San Diego, Calif; the Department of Neurology (J.C., C.Z., M.C.), Henry Ford Health Sciences Center, Detroit, Mich; and the Department of Neurology (A.E., M.S., Y.L.), Wolfson Medical Center, Holon, Israel.

Correspondence to Amir Oron, MD, Department of Orthopedics, Assaf Harofeh Medical Center, Zerifin 70300, Israel. E-mail amiroronmd@gmail.com

Background and Purpose— Low-level laser therapy (LLLT) modulates various biological processes. In the present study, we assessed the hypothesis that LLLT after induction of stroke may have a beneficial effect on ischemic brain tissue.

Methods— Two sets of experiments were performed. Stroke was induced in rats by (1) permanent occlusion of the middle cerebral artery through a craniotomy or (2) insertion of a filament. After induction of stroke, a battery of neurological and functional tests (neurological score, adhesive removal) was performed. Four and 24 hours poststroke, a Ga-As diode laser was used transcranially to illuminate the hemisphere contralateral to the stroke at a power density of 7.5 mW/cm2.

Results— In both models of stroke, LLLT significantly reduced neurological deficits when applied 24 hours poststroke. Application of the laser at 4 hours poststroke did not affect the neurological outcome of the stroke-induced rats as compared with controls. There was no statistically significant difference in the stroke lesion area between control and laser-irradiated rats. The number of newly formed neuronal cells, assessed by double immunoreactivity to bromodeoxyuridine and tubulin isotype III as well as migrating cells (doublecortin immunoactivity), was significantly elevated in the subventricular zone of the hemisphere ipsilateral to the induction of stroke when treated by LLLT.

Conclusions— Our data suggest that a noninvasive intervention of LLLT issued 24 hours after acute stroke may provide a significant functional benefit with an underlying mechanism possibly being induction of neurogenesis.

Key Words: laser • neurogenesis • neuroprotection • rat • stroke