RED LIGHT HELPS PREVENT DIABETIC RETINOPATHY

One thing syntonists would love to be able to offer patients is syntonic treatment to reverse early-stage retinal disease. Now comes new research that shows promise of reversing early stage diabetic retinopathy with low-level red visible light, at least in mice. The light wavelength used in the study is 670 nm from an LED source. Our closest syntonic filter would be alpha or perhaps alpha-upsilon. One of the research authors is Janis Eells, who lectured at our 72th Annual Conference on Light and Vision in Niagara Falls (2004). She presented about her research finding that red visible light prevents retina damage from wood alcohol toxicity (also with mice). This pioneering research gained broad worldwide attention and lead to a new field of phototherapy research, one that syntonic devices are capable of rendering. Below is the abstract of the study. -RG

Low-Intensity Far-Red Light Inhibits Early Lesions That Contribute to Diabetic Retinopathy: In Vivo and In Vitro

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PURPOSE.
Treatment with light in the far-red to near-infrared region of the spectrum (photobiomodulation [PBM]) has beneficial effects in tissue injury. We investigated the therapeutic efficacy of 670-nm PBM in rodent and cultured cell models of diabetic retinopathy.

METHODS.
Studies were conducted in streptozotocin-induced diabetic rats and in cultured retinal cells. Diabetes-induced retinal abnormalities were assessed functionally, biochemically, and histologically in vivo and in vitro.

RESULTS.
We observed beneficial effects of PBM on the neural and vascular elements of retina. Daily 670-nm PBM treatment (6 J/cm2) resulted in significant inhibition in the diabetes-induced death of retinal ganglion cells, as well as a 50% improvement of the ERG amplitude (photopic b wave responses) (both $P < 0.01$). To explore the mechanism for these beneficial effects, we examined physiologic and molecular changes related to cell survival, oxidative stress, and inflammation. PBM did not alter cytochrome oxidase activity in the retina or in cultured retinal cells. PBM inhibited diabetes-induced superoxide production and preserved MnSOD expression in vivo. Diabetes significantly increased both leukostasis and expression of ICAM-1, and PBM essentially prevented both of these abnormalities. In cultured retinal cells, 30-mM glucose exposure increased superoxide production, inflammatory biomarker expression, and cell death. PBM inhibited all of these abnormalities.

CONCLUSIONS.
PBM ameliorated lesions of diabetic retinopathy in vivo and reduced oxidative stress and

cell death in vitro. PBM has been documented to have minimal risk. PBM is noninvasive, inexpensive, and easy to administer. We conclude that PBM is a simple adjunct therapy to attenuate the development of diabetic retinopathy.
Keywords: photobiomodulation, diabetic retinopathy, retinal ganglion cells