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Metanx and early stages of diabetic retinopathy.

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Abstract

PURPOSE: l-Methylfolate, pyridoxal 5'-phosphate, and **methylcobalamin**, individually have been reported to have beneficial effects on diabetes-induced defects. The possibility that combining these therapeutic approaches might have additional benefit led us to investigate the effect of Metanx against development of early stages of diabetic retinopathy in a mouse model.

METHODS: C57BL/6J mice were made diabetic with streptozotocin, and some were given Metanx (a combination food product) mixed in the food at a dose of 5 mg/kg of body weight. Mice were killed at 2 months and 10 months of study for assessment of retinal function, retinal vascular histopathology, accumulation of albumin in neural retina, and biochemical and physiological abnormalities in retina.

RESULTS: Two months of diabetes significantly increased leukostasis within retinal vessels and superoxide generation by the retina. Diabetes also significantly increased expression of intercellular adhesion molecule-1 (ICAM-1) and phosphorylation of IκB. Daily consumption of Metanx significantly inhibited all of these abnormalities. Ten months of diabetes significantly increased the degeneration of retinal capillaries and impaired visual function (spatial frequency threshold (SFT) and a parameter of contrast sensitivity) compared to nondiabetic controls. Daily consumption of Metanx for 10 months inhibited impairment of SFT but had no significant beneficial effect on capillary degeneration, pericyte loss, or the estimate of contrast sensitivity.

CONCLUSIONS: Metanx inhibited a diabetes-induced defect in retinal spatial frequency threshold and inhibited measures of oxidative stress and inflammation. It had no significant effect on contrast sensitivity or retinal capillary degeneration. Nutritional management with Metanx may help inhibit diabetes-induced defects in visual function.

KEYWORDS: Metanx; diabetic retinopathy; inflammation; oxidative stress

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