

Influence of lecithin on mitochondrial DNA and age-related hearing loss

(Otolaryngol Head Neck Surg 2002;127:138-44.)

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OBJECTIVES:

Lecithin is a polyunsaturated phosphatidylcholine (PPC), which are high energy functional and structural elements of all biologic membranes. PPC play a rate-limiting role in the activation of numerous membrane-located enzymes, including superoxide dismutase and glutathione, which are important antioxidants protecting cell membranes from damage by reactive oxygen species (ROS). ROS-induced damage to mitochondrial DNA may lead to reduced mitochondrial function in the cochlea and resultant hearing loss.

STUDY DESIGN AND SETTING:

The effects of lecithin on aging and age-associated hearing loss were studied in rats by measuring hearing sensitivities using auditory brainstem responses (ABR). In addition, mitochondrial function as a measure of aging was assessed by determining mitochondrial membrane potentials using flow cytometry and by amplifying mitochondrial DNA deletions associated with aging.

Harlan-Fischer rats aged 18 to 20 months (n = 14) were divided into 2 groups. The experimental group was supplemented orally for 6 months with lecithin, a purified extract of soybean phospholipid (Nutritional Therapeutics, Allendale, NJ).

RESULTS:

The data obtained were compared with the control group. ABRs were recorded at 2-month intervals and showed significant preservation of hearing sensitivities in the treated subjects. Flow cytometry revealed significantly higher mitochondrial membrane potentials in the treated subjects, suggesting preserved mitochondrial function. Finally, the common aging mitochondrial DNA deletion (mtDNA₄₈₃₄) were amplified from brain and cochlear tissue including stria vascularis and auditory nerve. This specific deletion was found significantly less frequent in all tissues in the treated group compared with the controls.

CONCLUSION:

These experiments support our hypothesis and provide evidence that lecithin may preserve cochlear mitochondrial function and protect hearing loss associated with aging.