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Role of Apolipoproten E in liver aging protection

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Aging is characterized by a progressive decline of cellular functions. Reactive oxygen species (ROS) are involved in the aging process and result mainly from nonenzymatic processes in the liver. Endogenous free radicals are generated within mitochondria and suspected to cause severe injury to mitochondrial DNA. This damaged DNA accumulates with aging. In addition, polyunsaturated fatty acids, highly sensitive to ROS, decrease in liver mitochondria from human centenarians, a feature acquired during evolution as a protective mechanism to favor longevity. Diet is considered the main environmental factor having effect on lifespan. It has a major impact on aging liver, the central metabolic organ of the body. Apolipoprotein E (apoE) null mice are a very popular model for studying spontaneous hypercholesterolemia, but only limited data are available for the role of apolipoprotein E in liver disease. The purpose of this study is to evaluate liver disease in apolipoprotein E deficient mice.

For this study, apoE null mice and control mice at different ages (6 weeks and 15 months) were used. Liver morphological damage and proteins involved in oxidative stress, apoptosis and aging (Bax, Sirt 1, p53) were analyzed. ApoE deficient mice have morphological alterations that are the hallmark of liver pathogenesis, which increase with the age of the animals.

In apoE null mice livers, there is also increased oxidative stress as compared to control mice at the same age and fewer antioxidant enzymes. Our findings add to the growing list of protective effects that apoE possesses.

Keywords: Liver, ApoE, Bax, Sirt 1, p53

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