OBJECTIVE: Mitochondrial abnormalities are suggested to be associated with the development of nonalcoholic fatty liver. Liver mitochondrial content and function have been shown to improve in oral feeding of acetyl-L-carnitine (ALC) to rodents. Carnitine is involved in the transport of acyl-coenzyme A across the mitochondrial membrane to be used in mitochondrial β-oxidation. We hypothesized that oral administration ALC with the antioxidant lipoic acid (ALC + LA) would benefit nonalcoholic fatty liver.

METHODS: To test our hypothesis, we fed Balb/C mice a standard diet (SF) or SF with ALC + LA or high-fat diet (HF) or HF with ALC + LA for 6 months. Acetyl-L-carnitine and LA were dissolved at 0.2:0.1% (wt/vol) in drinking water, and mice were allowed free access to food and water. Along with physical parameters, insulin resistance (blood glucose, insulin, glucose tolerance), liver function (alanine transaminase [ALT], aspartate transaminase [AST]), liver histology (hematoxylin and eosin), oxidative stress (malondialdehyde), and mitochondrial abnormalities (carbamoyl phosphate synthase 1 and electron microscopy) were done.

RESULTS: Compared with SF, HF had higher body, liver, liver-to-body weight ratio, white adipose tissue, ALT, AST, liver fat, oxidative stress, and insulin resistance. Coadministration of ALC + LA to HF animals significantly improved the mitochondrial marker carbamoyl phosphate synthase 1 and the size of the mitochondria in liver. Alanine transaminase and AST levels were decreased.

CONCLUSION: In a nonalcoholic fatty liver mice model, ALC + LA combination improved liver mitochondrial content, size, serum ALT, and AST without significant changes in oxidative stress, insulin resistance, and liver fat accumulation.

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