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Suggested function of *Tabebuia avellanedae* extract on alcoholic fatty liver disease

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Alcohol-induced liver injury is the most common liver disease in which fatty acid metabolism is altered. It is thought that altered NAD+/NADH redox potential by alcohol in the liver cause fatty liver by inhibiting fatty acid oxidation and the activity of tricarboxylic acid cycle reactions. *Tabebuia avellanedae* extract (TAE), a naturally occurring quinone, has been shown to stimulate fatty acid oxidation in an obese mouse model by activating adenosine monophosphate-activated protein kinase (AMPK). In this report, we clearly show that TAE reduced alcohol-induced hepatic steatosis and induced fatty acid oxidizing capacity in ethanol-fed rats. TAE treatment markedly decreased hepatic lipids while serum levels of lipids and lipoproteins were increased in rats fed ethanol-containing liquid diets with TAE administration. Furthermore, inhibition of lipolysis, enhancement of lipid mobilization to mitochondria and up-regulation of mitochondrial β-oxidation activity in soleus muscle were observed in ethanol/TAE-treated animals compared to the ethanol-fed rats.

Biography

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