A Method for Inhibition of de novo Lipogenesis

Tech ID: 27665 / UC Case 2017-157-0

BACKGROUND

The obesity epidemic currently afflicting the US and other developed countries has resulted in a marked increase in the incidence of the metabolic syndrome and its associated pathologies, including nonalcoholic fatty liver disease (NAFLD), estimated to affect 30% of Americans. Although NAFLD is characterized by lipid droplet buildup in hepatocytes, it is not accompanied by liver damage, inflammation, and fibrosis unless combined with other risk factors, such as endoplasmic reticulum (ER) stress or mitochondrial dysfunction. In the context of simple, nonsymptomatic liver steatosis, ER stress or mitochondrial dysfunction trigger nonalcoholic steatohepatits (NASH), a serious disease that can progress to liver cirrhosis, resulting in loss of liver function, and hepatocellular carcinoma (HCC), one of the most deadly cancers. The obesity epidemic currently afflicting the US and other developed countries has resulted in a marked increase in the incidence of the metabolic syndrome and its associated pathologies, including nonalcoholic fatty liver disease (NAFLD), estimated to affect 30% of Americans. Although NAFLD is characterized by lipid droplet buildup in hepatocytes, it is not accompanied by liver damage, inflammation, and fibrosis unless combined with other risk factors, such as endoplasmic reticulum (ER) stress or mitochondrial dysfunction. In the context of simple, nonsymptomatic liver steatosis, ER stress or mitochondrial dysfunction trigger nonalcoholic steatohepatits (NASH), a serious disease that can progress to liver cirrhosis, resulting in loss of liver function, and hepatocellular carcinoma (HCC), one of the most deadly cancers.

TECHNOLOGY DESCRIPTION

Researchers at UC San Diego have identified a biochemical pathway responsible for activation of SREBP1/2 in mice suffering from NASH and demonstrates that the same pathway is active in human NASH patients. The invention demonstrates that inhibition of a critical component of this pathway reverses all NASH related symptoms in mice and prevents fat accumulation in liver and enhances energy expenditure.

APPLICATIONS

This invention provides a method of treating nonalcoholic steatohepatits (NASH), nonalcoholic fatty liver disease (NAFLD), and/or de novo lipogenesis (DNL) in humans. This method may also be used to potentially treat hepatocellular carcinoma.

STATE OF DEVELOPMENT

A prototype model has identified a crucial component of the pathway responsible for stimulation of de novo lipogenesis during NAFLD and NASH and have demonstrated its inhibition using a non-proprietary inhibitor which blocks DNA, increases energy expenditure and reverses NASH symptoms in a mouse model.

INTELLECTUAL PROPERTY INFO

A provisional patent has been submitted.

PATENT STATUS

Patent Pending

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OTHER INFORMATION

KEYWORDS

NASH, NAFLD, liver disease, liver cirrhosis, hepatocellular carcinoma, lipogenesis

CATEGORIZED AS

- **Biotechnology**
  - Health
- **Medical**
  - Disease: Metabolic/Endocrinology

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