OFFICIAL ABSTRACT and CERTIFICATION

Identification of a Model Agnostic Disease Driver in Non-alcoholic Steatoheaptitis; Implications for Drug Development

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Currently, there is a strong prevalence of non-alcoholic steatohepatitis (NASH), affecting a third of all American adults. This asymptomatic disease is closely associated with obesity and diabetes. NASH, which begins as non-alcoholic fatty liver disease (NAFLD), can further progress to NASH with increasing levels of fibrosis, cirrhosis and even hepatocellular carcinoma (HCC). Currently, there is no approved therapy for NASH and a number of therapeutics that has met success in the laboratory has fared poorly in clinical trials. Therefore, this study suggests a model agnostic approach to identify a disease driver in mice models that also corresponds to human NASH. Adult mice were separated by three treatments: fast food diet (FFD), FFD + thioacetamide, and FDD + CCl4 + glucose water; the 3 produced models each represent a stage of the NAFLD progression. Histopathological features were semi-quantitated on the basis of NAFLD activity score (NAS) and for fibrosis severity. Transcriptomic analysis was conducted through quantitative polymerase chain reaction (qPCR), and based on mRNA data, the majority experienced increased expression in the models representing NASH with fibrosis. However, the gpat1 gene to be sustained across all three models studied. The early detection and continued expression of gpat1 and its strong correlation with NAS suggests the functional significance of this disease driver in human NASH.

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