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Inhibitory nucleic acid molecules targeting cyclin D1 mRNA

Inhibition of non-canonical functions of cyclin D1 as a potential therapy for liver diseases.

IP Status: US Patent Pending; US Patent Application No.: 18/209,039

Applications

- Liver Disease Treatment
- Research Tool

Key Benefits & Differentiators

- **Precision Targeting:** Targets cyclin D1 mRNA precisely, minimizing off-target effects.
- **Broad Applicability:** Effective across various liver diseases, including NAFLD, NASH, and HCC.
- **Synergistic Effects:** Enhances therapeutic efficacy when combined with other treatments by reducing metabolic enzyme activity.

Technology Overview

Liver diseases such as non-alcoholic fatty liver disease (NAFLD), non-alcoholic steatohepatitis (NASH), and hepatocellular carcinoma (HCC) represent significant global health challenges. These conditions are characterized by complex pathological processes involving the liver, contributing to substantial morbidity and mortality rates. Current treatment modalities primarily focus on lifestyle modifications, yet they often fall short in addressing the intricate cellular and molecular mechanisms underlying these conditions.

Researchers at the University have developed inhibitory nucleic acid molecules targeting and suppressing cyclin D1 mRNA expression, presenting a therapeutic approach to address the challenges posed by liver diseases. This innovative technology leverages a comprehensive understanding of the intricate interplay between cell cycle regulation and metabolic pathways. Cell proliferation necessitates metabolic reprogramming to accommodate the biosynthesis of new cell components, a phenomenon mirrored in cancer cells. Cyclin D1, a pivotal cell cycle regulator often overexpressed in many cancers, exerts profound effects on biosynthetic metabolism, as revealed through studies on hepatocyte proliferation. Cyclin D1 broadly promotes biosynthetic pathways, including glycolysis, the pentose phosphate pathway, and purine and pyrimidine nucleotide synthesis in hepatocytes. Furthermore, proteomic analyses indicate that cyclin D1 binds to numerous metabolic enzymes, activating key enzymes in glycolysis and de novo pyrimidine synthesis. Pharmacologic inhibition of cyclin D1/Cdk4 significantly reduces the activity of these enzymes, offering a promising therapeutic approach to target liver diseases.

Phase of Development

TRL: 3-4
Researchers have obtained significant in vivo data (mouse liver models) and cell culture data.

Technology ID
2022-145

Category
Life Sciences/Biologics
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Researchers

- **Jeffrey Albrecht , MD** Professor of Medicine, Division of Gastroenterology, Hepatology, and Nutrition

References

1. Heng Wu, Betsy T. Kren, Andrew N. Lane, Teresa A. Cassel, Richard M. Higashi, Teresa W. M. Fan, George S. Scaria, Laurie L. Shekels, Mark A. Klein, and Jeffrey H. Albrecht , https://doi.org/10.1016/j.jbc.2023.105407, Journal of Biological Chemistry