THE FLORIDA STATE UNIVERSITY
OFFICE OF IP DEVELOPMENT & COMMERCIALIZATION

technology opportunity




Method of Using RNA as an Inhibitor of HCV

The NIH estimates that four million Americans are infected with Hepatitis C Virus (HCV) and an estimated 8,000 to 10,000 Americans die annually of HCV related complications. This figure is expected to triple in the next 10 to 20 years. FSU researchers have utilized siRNA HT-161 to effectively block HCV replication and infection in cell culture. The replication is inhibited by clearing human cells of a protein essential for HCV replication. HT-161 inhibits diverse HCV strains including the genotype 1a and 1b that are prevalent and resistant to interferon therapy.

Applications

- The method has been shown to prevent new infection and eliminates replicating HCV RNA from infected cells.
- It can be utilized against a diverse selection of HCV.

Advantages

- HT-161 targets a cellular gene necessary for viral replication thereby significantly reducing the likelihood of viral escape and resistance due to mutation.
- Current therapies, such as interferon (IFN), have significant adverse side effects and HCV strains develop resistance to IFN treatment.
- Unlike other siRNAs, the treatment does not target the viral genome, which, when targeted, increases the risk of mutations conveying resistances to treatment.

Technology Opportunity

The Inventors

Dr. Hengli Tang is an Assistant Professor at Florida State University. He received his Ph.D from the University of California in San Diego in 1998. The general area of research interest in his lab is virus-host cell interactions concerning human immunodeficiency virus (HIV) and hepatitis C virus (HCV). Currently, the molecular and cell biology of HCV replication is the main focus of the lab.

Projects:
1. Characterization of cellular factors for HIV infection and HCV replication using a variety of biochemistry, cell biology and molecular biology techniques.
2. Dissection of the molecular mechanisms of HCV replication in vitro, especially of the adaptive mutations.