Efficacy of Modern Drugs against Recurrence of Hepatitis C Virus (HCV) in Co-Infected HIV Infected Patients

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Abstract
Recent advances towards the cure for hepatitis C virus (HCV) infection have largely been successful, more so with minimal or no side effects of the anti-HCV drugs and not requiring combination therapies. In patients co-infected with human immunodeficiency virus (HIV) the chances of reoccurrence of HCV co-infection is more likely clinically either due to contaminated blood transfusions, or the immuno-suppressive nature of HIV. Hence the curative action of drugs against HCV infection is unrelated to any concomitant reduction of co-infection unrelated HIV replication. Thus HIV infected patients are always at risk to re-contract HCV infection. However any progress of the even in a transiently cured HCV infected patients are much less likely to progress into the slow developing hepato cellular carcinoma (HCC) and associated liver cirrhosis leading to HCV related death. Whereas the protease inhibitors of HCV are efficacious for a cure those of HIV seem to be restricted to its containment but not elimination from the human circulatory system.

Key words: HIV; HCV; Mutations; Prevalence; Cure

Introduction and Background
A major stumbling block to culture HCV in vitro has been the difficulty of its integration into host genome (Our initial patent: In Vitro Tissue Culture Assay to Screen Potential Drugs for HCV, University of California (UC) Case No. 2001-067-1 (Hepatitis C Virus), approved February 2007. Chronic HCV infection leads to HCC in 50% of the affected patients with 2-3% of the world population currently remain infected. Globally 150-170 million people are infected with HCV (Figure 1). Though the most current statistical distribution for HIV is not quoted herein, HIV prevalence globally (Figure 2) has always significantly been lower than that of HCV.

Uncontrolled HCV infection can lead to HCC and death, so also high proviral loads of chronic HIV replication. Whereas HCV is an RNA virus, HIV despite being a lentivirus (retrovirus derivative) its provirus for integration into the human host genome is DNA, post-reverse transcription from RNA into DNA [1]. From the rapidity with which a cure for HCV has been developed it seems that the mutations in the NS3 region of HCV genome [2] could be tackled therapeutically considerably easier [3] than the yet incurable HIV infection with its extremely high mutation rate of V3 loop region of HIV [1-4].

Discussion
Interferon-α (IFN-α) has long been known to carry anti-viral efficacy including against HCV but with some confined virus species specific effectiveness particularly with reference to inaction against HIV. IFN-α is known for its side effects and hence initial anti-HCV treatments using IFN-α were not successful. In recent times following development of HCV specific drugs that have been developed, the need for using IFN-α in a combination therapy is significantly, if not dramatically reduced. Such single use drugs are developed in 2014 and include ledipasvir and sofosbuvir, the latter comes in an easy once-a-day pill. It takes as
few as 12 weeks to work, and it cures up to 90% of HCV infected patients.

**Conclusion**

It is heartening to note that cure for HCV is well on its way through advancement into further generations, but for HIV it is still elusive though to-date the longevity of the infected patients is greatly increased and with fewer side effects.

**References**