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Commentary

Phylogenetic Analysis of Acute and Chronic Sequences and Mathematical Models of Early HCV Diversification

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DESCRIPTION

As many as 170 million people, or around 3% of the world's population, are infected with the Hepatitis C Virus (HCV).The virus results in a wide range of pathologic effects, the most notable of which are chronic liver disease, cirrhosis, and the almost always fatal hepatocellular carcinoma. The most common reason for liver transplantation in the US is HCV infection. HCV is an RNA virus with a positive strand, non-segmented envelope, and a length of about 9.6 kb. The virus belongs to the wider family of Flavivirus, which also contains the human illnesses West Nile virus, yellow fever virus, and dengue fever virus, among others. It is classified under the genus Hepacivirus. The Flaviviridae share the ability to replicate only when an RNA-dependent RNA polymerase (RdRp) that is expressed by the virus is present. RdRp is prone to errors, and HCV is famous for having a wide range of variations both within and across individuals. The nucleotide sequences of the seven main HCV genotypes varied by about 30% globally.

The extreme diversity of HCV makes it difficult to study the virus' biology, pathology, and susceptibility to cutting-edge treatments. In terms of the clinical setting, the various HCV genotypes have varying natural histories and responses to interferon, ribavirin, and the more recent Direct Acting Antiviral (DAA) drugs. The understanding of viral immunopathogenesis and the development of effective vaccinations against HCV are both hampered by HCV variation. It is intriguing that the extreme variety of HCV is comparable to that of HIV-1 and that a cutting-edge experimental method to detect Transmitted/Founder (T/F) HIV-1 genomes has revealed new information on the spread and persistence of the virus. Conventionally, the first six months of HCV infection are considered to be the period of acute infection. During this time, virus-host interactions are set into motion, greatly influencing the course of the disease. A portion of newly infected people naturally control or eliminate virus, depending on viral genotype and host immunogenetic variables, most importantly IL28B alleles.

If the infection is treated with interferon and ribavirin separately or in combination with DAA medications, a higher percentage of patients will be cured. Mechanistically, it is not fully understood how this happens. The acute infection period is crucial from the standpoint of a vaccine because transmitted viruses are the obvious targets of a vaccine and because the virus may be most susceptible to being eliminated by vaccine-elicited immune responses during the early stages of infection, when viral diversity is lowest. For all of these reasons, the molecular characteristics of the initial virus population "bottleneck" connected to virus transmission and the subsequent processes of virus evolution that result in persistence are of great interest. In earlier papers, various experimental methods for studying the HCV transmission bottleneck were presented. Among them are studies that used the DNA heteroduplex gel shift method to calculate viral diversity, traditional Polymerase Chain Reaction (PCR) techniques to mass amplify, sequence, or clone and sequence HCV genome fragments, and 454 pyrosequencing to more thoroughly but narrowly probe early viral sequences. Despite the use of increasingly sensitive techniques, a precise quantitative, molecular description of HCV transmission and early diversification has remained elusive. These results showed a constraint in viral variety linked with virus transmission.

In the current investigation, we expected that Single Genome Amplification (SGA), followed by direct amplicon sequencing, also known as single genome sequencing, would enable unequivocal identification of T/F HCV genomes and detailed mapping of their early pathways of diversification. By giving gene-wide or genome-wide viral sequences proportional to their representation in human plasma and free from template resampling, Taqpolymerase mistakes of nucleotide misincorporation, or recombination, this approach differs from earlier approaches used to study HCV. To account for variations in the biology of replication between HIV-1 and HCV, we amplified and sequenced the HCV core, E1, E2, P7, NS2 and NS3 genes. We next evaluated these genes using a model of random HIV-1 evolution. New estimates of the HCV mutation rate in people were obtained using an agent-based stochastic

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model of acute HCV replication dynamics that Ribeiro and colleagues developed using these sequences and plasma viral load kinetic data.