

Hepatitis c virus – 26 years on!

Editorial

We have come a long way in understanding the hepatitis C virus (HCV) and the disease it causes. Originally described as hepatitis non-A non-B, via a process of elimination while the screening the blood product supply, the virus was discovered in 1989 by molecule cloning methods, which at that time was a first. Back then when I first started virology we were trying to find the reservoirs for the virus in the body and also grow the virus in cell culture, the classical way to characterize virus. We now know HCV is primarily in blood the key reservoirs the liver. I had grown many other viruses and was using liver cell lines like HepG2 and primary cell culture methods to see if the virus would grow. In 1991 there was a report by a Japanese group that the virus had been grown in culture, but that was never reproduced. Until the full-length clone was developed much of the protein expression and pathophysiology of the RNA Hepacivirus was unknown. To this day the exact mechanism of viral entry into the liver cell is not known, but is associated with several viral and cellular factors such as human scavenger receptor SR-BI, tetraspanin CD81, and tight junction molecules occludin and Claudin-1. During HCV assembly and release from infected cells, virus particles associate with lipids and very-low density lipoproteins, and circulate in the blood form of triglyceride-rich particles.¹

Now there is a range of treatments for HCV aiming to permanently eliminate the virus, or to induced a sustained virologic response (SVR), 6 months after antiviral treatment. Those with genotypes 2 or 3 are more likely to achieved SVR than those with genotypes 1, 4, 5 or 6. Treatment originally was with interferon and ribavirin, but as recently as 2011 approval of the first direct-acting antiviral (DAA) agents occurred. The NS3/4A protease inhibitors boceprevir and telaprevir produced improved outcomes for genotype 1, have since been replaced by two new medications in 2013, simeprevir another NS3/4A protease inhibitor and sofosbuvir, the first-in-class NS5B polymerase inhibitor. We now need to wait and see if HCV can truly

Volume 1 Issue 1 - 2014

Rachel Cameron

Department of State Development, Program Trade and Industry Development, Business and Innovation, Medical Communications Manager, Amgen Australia Pty Ltd, and Consultant to Skilled and Business Migration, Australia

Correspondence: Rachel Cameron, Medical Communications Manager, Amgen Australia Pty Ltd, and Consultant to Skilled and Business Migration Program Trade and Industry Development, Department of State Development, Business and Innovation, Melbourne, Victoria, Australia,
Email rcameron2@iprimus.com.au

Received: May 18, 2014 | **Published:** May 19, 2014

be cured with permanent SVR, which would be significant progress in less than 26 years, despite still some fundamental virological questions about HCV being unanswered!

Acknowledgments

None.

Conflict of interest

Author declares that there is no conflict of interest.

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