CPQ Microbiology (2018) 1:3
Editorial

# Management of Hepatitis B Virus Infection Using CRISPR/Cas Technology

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Received: 15 June 2018

Published: 19 June 2018

**Keywords:** *Hepatitis B Virus; Bacteriophages; Effector Protein; Viral Genome*

HBV (hepatitis B virus) infection is a major cause of mortality and morbidity, particularly in Asia and Africa. One of the main issues in present drugs for management of hepatitis B virus infection is that, although inhibitors can inhibit the formation of viral DNA from pre-RNA transcripts but the translation and transcription of viral mRNAs from the cccDNA template do not stop by these inhibitors which are present in the nucleus of infected cells. In addition, because cccDNAs are very stable, prolonged hepatitis B virus infections are least treated with the use of inhibitors and cannot completely stop the expansion of hepatitis B virus-related infections like hepatocellular carcinoma and cirrhosis. Consequently, there is a lot of attention in the possibilities of emerging the methods of treatment which will be targeted to cccDNA directly to eliminate.

The capacity of the bacterial CRISPR/Cas machinery which is DNA editing machinery as a specific instrument for the destruction and cleavage of hepatitis B virus cccDNAs in the nucleus of infected cells. This machinery will be regulated and consider which measures CRISPR/Cas targeting of hepatitis B virus DNA will be necessary, and a clinically viable approach to treat permanent infections in human being HBV cccDNAs.

CRISPR/Cas loci are present in broad range of bacteria, is ready to target specific RNAs of various DNA bacteriophages. Bacterial CRISPR/Cas type ii system is transcribed with respect to a close permanent area to form a crRNA (CRISPR RNA), crRNA intricate with the tracrRNA (trans-activating RNA). Then this heterodimer bound by Cas9 (effector protein). Cas9 recognized PAM (protospacer adjacent motif) which is a small DNA sequence. Then a DNA target genome scans by Cas9 protein for the protospacer adjacent motifs sequence, then binds the DNA to the crRNA. If larger complementarity is recognized, then Cas9 protein chops target phage DNA by using two different domains of protein: Cas9 RuvC domain and Cas9 HNH (nuclease domain), one chops non-complementary strand of DNA, while other chops complementary strand of DNA. After this dsDNA disruption brings the degradation of the viral genome and stops infection.

Once combination of both Cas9/sgRNA expressed in hepatocyte cells infected with HBV, cleavage of cccDNA occurs. Due to this mechanism a lots of cccDNA molecules are eliminate. Alternatively, NHEJ repaired cleaved cccDNA of HBV. Again cleaved those molecules which are restored in the wild-type, resulting in the gathering of mutated DNA molecules of HBV, which are usually contain small fragments that are rejected for further cleavage due to un-complementarity with sgRNA.

Cleavage of HBV genomes is promised by CRISPR/Cas9 to eliminate cccDNAs of persistent HBV. Therefore, it seems that by combined CRISPR/Cas9 and sgRNA, it is a possible strategy to cure long-lasting hepatitis B. However, Vitro is stable and valuable cultural system that can produce authentic cccDNAs of HBV, is presently missing and need to find the utility of CRISPR/Cas.