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Computational analysis to detect resistance mutations to direct acting antivirals in hepatitis C virus

Karina Salvatierra
National University of Misiones, Argentina

Hepatitis C virus (HCV) infection is considered as a major public health problem with an estimate of 200 million people infected worldwide. HCV infection is the major cause of chronic liver disease, with severe outcomes including cirrhosis and hepatocellular carcinoma and it is the main cause of liver transplantation. The treatment for HCV chronic infection with pegylated interferon alpha plus ribavirin inhibitors is unspecific; consequently, the treatment is effective in only 50% of patients infected. This has prompted the development of direct-acting antivirals agents (DAA's) that target virus proteins. Unfortunately, since the virus has a high replication rate and its RNA polymerase lacks proofreading activity, genetic variations might produce resistance against the DAA's. These DAA's have demonstrated a potent effect *in vitro* and *in vivo*; however, virus mutations associated with the development of resistance have been described. The objective of this work is to detect mutations in known aminoacids to be implicated in resistance to DAA's in sequences obtained of conventional Sanger and cloning sequencing. We have designed and developed an online information system named Biomedical Mutation Analysis (BMA), which allows users to calculate changes in nucleotide and amino acid sequences for each selected sequence from conventional Sanger and cloning sequencing. BMA allows the computational analysis quickly, easily and effectively. Furthermore, the development of different visualization techniques allows a proper interpretation and understanding of the results. The data obtained from BMA will be useful for HCV resistance surveillance for the design of broad-range inhibitors and rationale therapeutic regimen.

kariales@gmail.com