The Influence Of NS5A-HCV In The Response To Treatment Of Hepatitis C And InThe Stablishment Of Virus-Host Interactions.

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INTRODUCTION AND OBJECTIVE Hepatitis C virus causes the hepatitis C disease which affects over 170 million people worldwide. This disease is known to chronificate in up to 80 percent of cases and relies on the viral manipulation of the cellular machinery to persist and evolve, causing fibrosis, cirrhosis and cellular hepatocarcinoma. Identifying viral factors associated with viral drug resistance and host factors that interact with viral proteins is crucial to a better comprehension of disease progression and possible drug targets. The viral non structural protein 5a (NS5A) is an important protein in the biological cycle of the virus because it has been associated with resistance therapy. In addition, it has been described to interact with several host factors and to manipulate different cellular pathways, such as the cellular cycle. However, its complete role is yet to be elucidated. Therefore, this work aims to study viral genetic diversity in the NS5A region and its association with response to treatment and to determine novel partners of this protein inside the host cell and how it affects the disease progression. **MATERIALS AND METHODS** Patients were selected at HUCFF to assess the genetic diversity in NS5A by sequencing with Ion Torrent. The data generated will be analyzed by bioinformatics software. In order to determinate the possible interactors of NS5A, this region was amplified from the HCV 1b subgenomic replicon and cloned into the tandem affinity purification vector (pNTAP). **RESULTS AND DISCUSSION** Patients were selected. It was carried out extraction of viral RNA ant the reactions of RT-PCR were standardized. The construction NS5A-pNTAP was performed and confirmed by PCR, digestion and sequencing. **CONCLUSIONS:** The reactions for analysis of the genetic diversity of the NS5A are standardized and the interactions assays are yet to be performed therefore we can only conclude that our molecular tools are functional.

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