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*EDITORIAL*

# Recent advances in hepatitis C virus research and understanding the biology of the virus

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Since the identification of hepatitis C virus (HCV) genome in 1989[1], a lot of progresses have been done about the understanding of HCV biology, natural history and therapeutic options. HCV is a member of the Flaviviridae viral family. Its genome is a positive simple strand RNA molecule which shows significant genetic variability. The HCV nomenclature has been recently re-examined by an international group of scientific experts in the field of HCV genetic variability or involved in HCV data base[2]. HCV sequence variability and HCV classification has considerably evolved since the consensus paper in 1994[3] proposing the classification of HCV by phylogenetic methods into 6 genotypes. Each genetic group contains a variable number of closely related but distinct subtypes at the nucleotide level. Genotypes differ from each other by 31% to 33%, and subtypes by 20% to 25%. Since this time, several molecular epidemiology studies have revealed a much higher diversity especially in certain region of the world demonstrating that HCV was present a long time ago in human populations, and that recent routes of spread, like transfusions, nosocomial transmission, or Intra Venous Drug use have allowed a rapid spread of HCV subtypes. Recombination between genotypes[4,5] were also described making the classification of certain strains more difficult. Because genotype identification is clinically important in terms of response to current HCV treatments (Pegylated Interferon and Ribavirin), experts have examined the must reliable methods for HCV classification especially for phylogenetic analysis of the core, E1, NS5B genes and complete genome sequences[2]. These combined methods support the primary division of HCV into the 6 genetic groups termed genotypes. Variants of HCV above 6 are being renamed according to the genotype group. The new proposal of the scientists and experts now serve as a framework for access to the 3 Databases (which follows the current revised nomenclature and the revised criteria for HCV classification). Particularly due to the variability of HCV strains, HCV infection is characterized by an extremely high rate (60%-80%) of chronic carrier state development associated with viral multiplication. The understanding and modelisation of natural history is still debated but the chronic carrier state is associated with the development of liver fibrosis and the risk of hepatocellular carcinoma (HCC). Histopathological and clinical studies have individualized by assimilation with HIV infection low and rapid progressors. However, risk factors for liver fibrosis progression seem to be linked to many individual or environmental factors (age, sex, age at infection, route of transmission, alcohol consumption, *etc.*). One of the most recent progress in the assessment of liver fibrosis is the development of blood scores (Hepascore®, Fibrometer®, Fibrotest®, ...) or physical methods like elastometry (Fibroscan®) which are modifying the management of patients with HCV infection and may probably facilitate the follow up of fibrosis progression[6-10]. The understanding of the HCV biology has also considerably benefit from new *in vitro* and *in vivo* systems of viral replication like the replicon system and transgenic mice models. These new culture and animal models together with the growing knowledge of molecular biology of HCV have reinforced the importance of HCV variability and its potential role in the natural history of infection but also in the mechanisms of resistance to antiviral agents. In this issue of World Journal of Gastroenterology, experts have examined the main aspects of HCV infection trying to focus on these new findings or understanding about biological, clinical and therapeutic progresses.

## REFERENCES

1 **Choo QL**, Kuo G, Weiner AJ, Overby LR, Bradley DW, Houghton M. Isolation of a cDNA clone derived from a blood-borne non-A, non-B viral hepatitis genome. *Science* 1989; **244**: 359-362

2 **Simmonds P**, Bukh J, Combet C, Deléage G, Enomoto N, Feinstone S, Halfon P, Inchauspé G, Kuiken C, Maertens G, Mizokami M, Murphy DG, Okamoto H, Pawlotsky JM, Penin F, Sablon E, Shin-I T, Stuyver LJ, Thiel HJ, Viazov S, Weiner AJ, Widell A. Consensus proposals for a unified system of nomenclature of hepatitis C virus genotypes. *Hepatology* 2005; **42**: 962-973

3 **Simmonds P**, Smith DB, McOmish F, Yap PL, Kolberg J, Urdea MS, Holmes EC. Identification of genotypes of hepatitis C virus by sequence comparisons in the core, E1 and NS-5 regions. *J Gen Virol* 1994; **75** (Pt 5): 1053-1061

4 **Kalinina O**, Norder H, Mukomolov S, Magnius LO. A natural intergenotypic recombinant of hepatitis C virus identified in St. Petersburg. *J Virol* 2002; **76**: 4034-4043

5 **Noppornpanth S**, Lien TX, Poovorawan Y, Smits SL, Osterhaus AD, Haagmans BL. Identification of a naturally occurring recombinant genotype 2/6 hepatitis C virus. *J Virol* 2006; **80**: 7569-7577

6 **Oberti F**, Valsesia E, Pilette C, Rousselet MC, Bedossa P, Aubé C, Gallois Y, Rifflet H, Maïga MY, Penneau-Fontbonne D, Calès P. Noninvasive diagnosis of hepatic fibrosis or cirrhosis. *Gastroenterology* 1997; **113**: 1609-1616

7 **Castéra L**, Vergniol J, Foucher J, Le Bail B, Chanteloup E, Haaser M, Darriet M, Couzigou P, De Lédinghen V. Prospective comparison of transient elastography, Fibrotest, APRI, and liver biopsy for the assessment of fibrosis in chronic hepatitis C. *Gastroenterology* 2005; **128**: 343-350

8 **Imbert-Bismut F**, Ratziu V, Pieroni L, Charlotte F, Benhamou Y, Poynard T. Biochemical markers of liver fibrosis in patients with hepatitis C virus infection: a prospective study. *Lancet* 2001; **357**: 1069-1075

9 **Calès P**, Oberti F, Michalak S, Hubert-Fouchard I, Rousselet MC, Konaté A, Gallois Y, Ternisien C, Chevailler A, Lunel F. A novel panel of blood markers to assess the degree of liver fibrosis. *Hepatology* 2005; **42**: 1373-1381

10 **Halfon P**, Bacq Y, De Muret A, Penaranda G, Bourliere M, Ouzan D, Tran A, Botta D, Renou C, Bréchot MC, Degott C, Paradis V. Comparison of test performance profile for blood tests of liver fibrosis in chronic hepatitis C. *J Hepatol* 2007; **46**: 395-402

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