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NOVEL VIEW ON PATHOGENESIS OF ACUTE AND CHRONIC HCV INFECTION

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Słowa kluczowe: HCV, patogenezą

Key words: HCV, pathogenesis

Hepatitis C virus (HCV) is estimated to affect nearly 170 million people worldwide. The burden of liver and other organs diseases consequent to HCV has been well described, and is expected to increase dramatically over the next decades. The natural host of HCV is only man. HCV belongs to the *Flaviviridae* family, genus *Hepacivirus*, and so far 6 HCV genotypes (1-6), large number subtypes (1a, 1b) and huge number of *quasispecies* have been identified. Current availability of cell culture model systems and new insights into molecular and cellular mechanisms underlying HCV entry, replication, assembly and regress have been gained. The viral, host genetic diversity and environmental behavioral factors are believed to contribute to the spectrum of clinical outcomes of patients infected with HCV. Cellular and humoral responses are generated during HCV infection, however they are insufficient to effect viral clearance in the majority of individuals, with 80% of new infections becoming chronic. Effective clearance of an acute viral infection requires the coordinated function of many arms of the immune system: acquired or adaptive immune response specific to HCV (CD4+, CD8+ T cells), and innate immune system (natural killer (NK), natural killer T (NKT) cells and interferons). In chronically infected patients T cell responses are almost absent, but they can be easily detected in patients who are able to clear HCV after acute hepatitis or the successful IFN-alfa/RBV therapy.

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NOWE SPOJRZENIE NA PATOGENEZĘ OSTREGO I PRZEWLEKŁEGO ZAKAŻENIA HCV

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