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Impact of host gene polymorphisms on susceptibility to chronic hepatitis B virus infection

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Hepatitis B virus (HBV) infection can result in a number of different clinical conditions. Variations in cytokine genes have been discussed to affect the natural history of HBV infection. Various studies reveal that SNPs play an important role in pathogenesis of HBV. On the other hand, various outcomes of infection cannot be completely shown by genetic factors because these studies have inconsistent results with regard to the possible impacts of host genetic polymorphisms on susceptibility to infection. Host immunity and genetic factors play important roles during infections. Current review will discuss significant genetic variations in cytokine genes that may affect the susceptibility to the chronic HBV infection. Some cytokine genotyping such as IL-10 and IL-28B are suggested to predict the response to treatments because of its function in reducing the hepatic inflammation. Therefore, it can be a gene therapy target for infection. Various MHC alleles such as *DRB1*1302*, *A*0301*, *DR2*, *DR6* and *DR13* have been identified that are associated with favorable outcomes in HBV infection. The variants in the HLA class II region such as *HLA-DP*, *HLA-DQ* and *HLA-DR* are also considered to be important in determining the risk of HBV infection. There are inconsistent results in understanding the clinical roles of these factors in the field of understanding the host and disease interactions. Therefore, answers to the dichotomous findings about susceptibility to the infection will be provided by further analysis of other host genetic and environment factors. The application of novel technologies will be answered to these complicated problems.

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