

## **Severity of hepatitis C and HIV co-infection in mothers contribute to HCV transmission to child**

EurekAlert

New research shows that high maternal viral load and co-infection with human immunodeficiency virus (HIV) are the only risk factors associated with vertical transmission of the hepatitis C virus (HCV-VT). A variation in the infant's IL28B gene (CC) is associated independently with the spontaneous clearance of HCV genotype-1 among infected children. The status of IL28B in the mother or children did not increase risk of HCV-VT in this study. Findings are published in the May issue of *Hepatology*, a peer-reviewed journal of the American Association for the Study of Liver Diseases.

Chronic HCV affects 170 million individuals worldwide, with 10% to 15% of cases leading to cirrhosis and liver cancer. A major route of infection in children is vertical transmission of HCV (HCV-VT)—known also as mother-to-child transmission—and may occur in utero or following birth (breast feeding). While medical evidence has described risk factors involved in HCV-VT, underlying transmission mechanisms and timing of disease transmission is not fully understood. Prior studies have investigated the relationship between HCV-VT and maternal HCV genotype, birth mode (vaginal or caesarean) and type of feeding (breast feeding or replacement), but results have been conflicting.

"Our study analyzed the role of IL28B in HCV-VT and the spontaneous clearance of HCV among infected infants," said Ángeles Ruiz Extremera, M.D., of San Cecilio University Hospital in Spain. The team recruited 145 mothers who were infected with HCV and gave birth between 1991 and 2009. All women were Caucasian—112 were HCV RNA positive and HIV negative; 33 were HCV RNA negative and HCV antibody positive. A total of 142 children were birthed by HCV-RNA positive mothers and 43 children to HCV-RNA negative mothers, all of whom were followed for six years or more. HCV-VT was defined as children who presented with HCV-RNA positive results from two blood samples.

Analysis showed that 61% of the 31 mothers with CC polymorphism, and 82% of the 68 mothers with non-CC polymorphism were HCV-RNA positive. There were 128 infants born to HCV-RNA positive mothers who were not co-infected with HIV and 20% of the children acquired HCV infection, with 7% of these being chronic cases. In mothers who were coinfecting with HCV and HIV, the HCV-VT rate climbed to 43%. Researchers also noted that the rate of HCV-VT was higher among mothers who had elevated HCV viremia levels. Researchers did not detect HCV-VT in HCV-RNA negative mothers.

An increased risk of HCV-VT was not associated with the mothers' or children's IL28B status. However, researchers found that genotype non-1 and CC of the IL28B gene were involved with viral clearance among children infected with HCV. In

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regression analysis the child CC polymorphism was the only predictor of spontaneous HCV clearance in HCV genotype-1. "Our data are the first to account for HCV virus clearance and may provide important information about protective immunity to HCV," concluded Dr. Ruiz Extremera. "Further investigation is needed to understand the mechanisms involved with this genetic variation and the clinical impact of the IL28B variant on HCV infection."

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