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Farquhar C, Rowland-Jones S, Mbori-Ngacha D, Redman M, Lohman B, Slyker J, Otieno P, Obimbo E, Rostron T, Ochieng J, Oyugi J, Bosire R, John-Stewart G.

Abstract

Human leukocyte antigen (HLA) molecules regulate the cellular immune system and may be determinants of infant susceptibility to human immunodeficiency virus type 1 (HIV-1) infection. Molecular HLA typing for class I alleles was performed on infants followed in a Kenyan perinatal cohort. Early HIV-1 infection status was defined as infection occurring at birth or month 1, while late infection via breast milk was defined as first detection of HIV-1 after 1 month of age. Likelihood ratio tests based on a proportional hazards model adjusting for maternal CD4 T cell count and HIV-1 viral load at 32 weeks of gestation were used to test associations between infant allelic variation and incident HIV-1 infection. Among 433 infants, 76 (18%) were HIV-1 infected during 12 months of follow-up. HLA B*18 was associated with a significantly lower risk of early HIV-1 transmission [relative risk (RR) = 0.26; 95% confidence interval (CI) 0.04-0.82], and none of the 24 breastfeeding infants expressing HLA B*18 who were uninfected at month 1 acquired HIV-1 late via breast milk. We observed a trend toward increased early HIV-1 acquisition for infants presenting HLA A*29 (RR = 2.0; 95% CI 1.0-3.8) and increased late HIV-1 acquisition via breast milk for both Cw*07 and Cw*08 (RR = 4.0; 95% CI 1.0-17.8 and RR = 7.2; 95% CI 1.2-37.3, respectively). HLA B*18 may protect breast-feeding infants against both early and late HIV-1 acquisition, a finding that could have implications for the design and monitoring of HIV-1 vaccines targeting cellular immune responses against HIV-1.