A common genetic variant in FCGR3A-V158F and risk of Kaposi sarcoma herpesvirus infection and classic Kaposi sarcoma.

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Abstract
Associations of FCGR3A among men with HIV/acquired immunodeficiency syndrome suggest that host responses affect the pathogenesis of Kaposi sarcoma herpesvirus (KSHV) infection and risk of acquired immunodeficiency syndrome-associated Kaposi sarcoma. Using DNA from two HIV seronegative case-control populations in Italy, we examined whether the functional FCGR3A-V158F variant was associated with risk of KSHV infection or classic Kaposi sarcoma (CKS). In population I, we examined FCGR3A variants and risk of KSHV infection in 34 KSHV latent nuclear antigen (LANA)-seropositive and 120 LANA-seronegative adults from Sardinia (52% male; median age, 45 years; range, 31-60), whereas in population II, we examined risk of CKS from 133 CKS cases and 172 KSHV LANA-seropositive controls from Sicily, Rome, and Naples (70% males; median age, 74 years; range, 29-91). FCGR3A variants were determined by direct sequence analysis of a nested PCR of genomic DNA assay using allele-specific primers. KSHV LANA was determined by immunofluorescence assay. Overall, compared with the 158F allele, 158V was overrepresented among controls from both Mediterranean populations (frequency = 0.52 and 0.51, respectively). After controlling for age, 158V homozygous women were at increased risk of KSHV infection and CKS compared with 158F homozygous women [odds ratio (OR), 8.7; 95%
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confidence interval (95% CI), 0.8-98 and OR, 3.8; 95% CI, 1.0-14, respectively], whereas homozygous men were at decreased risk (OR, 0.4; 95% CI, 0.1-2.3 and OR, 0.4; 95% CI, 0.2-0.8, respectively). Significant gene-dose effects were observed among men and women at risk for CKS (P(trend) < or = 0.05). Our findings suggest that gender differences could possibly modify the effect of FCGR3A on risk of KSHV infection and CKS. Additional studies are required to confirm these relationships and determine their etiologic significance.

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