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Novel varicella-zoster virus (VZV) glycoprotein E (gE) gene mutations associated with genotypes A and D

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VZV is the etiologic agent of two distinct clinical syndromes in humans: chicken pox (varicella), occurring during the primary infection, and shingles (zoster), appearing after reactivation from latency. Previously, two VZV gE mutant strains were identified in the United States and Canada. These mutants contain a nonsynonymous mutation in codon 150 of the gE gene, resulting in an aspartic acid-to-asparagine exchange at position 150. This amino acid exchange causes the loss of a B-cell epitope and a phenotype of faster cell-to-cell spread.

Recently, we described a practical and simple VZV genotyping scheme based on analysis of a region of 1,990 bp of open reading frames (ORFs) 51 to 58 that allows the typing of VZV wild-type strains by high-throughput procedures directly from clinical samples without intermediate virus propagation (Schmidt-Chanasit et al., 2007, JCM).

As associations between defined VZV genotypes, gE gene mutations, and immunocompromising factors have not been considerably investigated, the objective of this study was to reveal these possible associations.

Within 45 analyzed VZV wild-type strains of genotypes A and D, five novel gE mutation were discovered. A statistically significant ($P < 0.0001$) association of certain gE mutations with VZV genotype D was found. No statistically significant association of gE mutations with age, sex, or immunocompromising factors was observed.

This unique association of genotype D VZV wild-type strains with a distinct SNP profile of the gE gene and the gE sequence variations will be studied by our group for a potential role in VZV pathogenesis.

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