Host and Pathogen Genetics Modulate HSV-1 Severity

Meena Ramchandani1, Ronnie Russell2, Lichen Jing3, Amalia Magaret4, Stacy Selke3, Meei-Li Huang3, Eric Strachan3, Anna Wald1,3,5,6, David M. Koelle1,3,6

1. Department of Medicine, 2. Department of Statistics, 3. Department of Laboratory Medicine, 4. Department of Psychiatry, 5. Department of Epidemiology, University of Washington, Seattle, 6. Fred Hutchinson Cancer Research Center, Vaccine and Infectious Disease Division.

Abstract

HSV-1 infection has a wide severity spectrum in the human population. The current understanding of how genetic variation in host and virus contribute to disease phenotype is based on limited data. We evaluated HSV-1 genotypes and immunity in mono- and dizygotic twins. The mean percent agreement in ORF response among MZ twins was 80% (p=0.002) but not among unrelated individuals (88% for negative control antigens). These data and the higher correlation in shedding rates among twins with the same HSV serostatus support a higher correlation in HSV disease severity and host genotype is supported by our observation of a higher correlation in shedding rates in twins with the same versus different virus. A relationship between HSV-1 disease severity and host genotype is supported by our observation of a higher correlation in shedding rates in twins with the same versus different virus. The higher correlation in shedding rates among twins with the same virus versus different virus suggests that viral and host genetic contributions to disease severity.

Methods

A relationship between HSV-1 disease severity and host genotype is supported by our observation of a higher correlation in shedding rates in twins with the same versus different virus. The higher correlation in shedding rates among twins with the same virus versus different virus suggests that viral and host genetic contributions to disease severity.

Results

A relationship between HSV-1 disease severity and host genotype is supported by our observation of a higher correlation in shedding rates in twins with the same versus different virus. The higher correlation in shedding rates among twins with the same virus versus different virus suggests that viral and host genetic contributions to disease severity.

Discussion

A relationship between HSV-1 disease severity and host genotype is supported by our observation of a higher correlation in shedding rates in twins with the same versus different virus. The higher correlation in shedding rates among twins with the same virus versus different virus suggests that viral and host genetic contributions to disease severity.

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References


Figure 1: Diagram illustrating the study design.

Figure 2: Representative isolation of CD4+ T cells reactive to HSV-1.

Figure 3: HSV-1 ORFscreen and responses.

Figure 4: HSV-1 ORFscreen responses between MZ twins.

Figure 5: Analysis of twin pairs to unrelated individuals.

Figure 6: HSV-1 strains and shedding rates in twin pairs.