

Measles virus spreads in the brain by relocating its proteins to neurites and calibrating cell-to-cell fusion

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Abstract

The brains of individuals affected by subacute sclerosing panencephalitis (SSPE) bear measles virus (MeV) genomes harboring adaptive mutations that functionally change their cell-to-cell transmission. However, the mechanisms supporting MeV lethal brain adaptation remain unclear. We show here that a matrix (M) protein amino acid change acts as a regulatory node controlling the location of the viral membrane fusion apparatus proteins in neurons. Variants of M protein amino acid Phe50, selected in about half of 38 SSPE cases previously examined, either enhance or restrain receptor-independent cell-to-cell fusion. Variants of the cytoplasmic tails of both viral glycoproteins, almost invariably co-selected with M-Phe50 variants in human brains, also calibrate cell fusion function. Calibration is facilitated by the distribution of mutant alleles across co-replicating genome populations. The two principles of lethal MeV brain adaptation, viral protein relocation and fusion efficiency calibration, may govern neuropathogenesis of other RNA viruses.

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