

to cultured mouse cells by virtue of interaction with both types of oligosaccharides on the cell surface. It is therefore somewhat paradoxical that the small plaque strains, with broader oligosaccharide binding capacity, are effectively inhibited from spreading in the intact host⁸. A possible explanation for the lower pathogenicity of small plaque viruses would be that sialoglycoconjugates rich in branched chains are present in tissues of at least some mouse strains, and that such structures act effectively as 'pseudoreceptors' or inhibitors specifically of these viruses. The relative densities of branched and straight-chain sialoglycoconjugates present on surfaces to which the virus can bind may also play a role. The distribution of VPI on the virus surface in principle allows simultaneous binding of many receptors without significant membrane distortion (Fig. 4), but the receptors would need to cluster within a region of 200–300 Å in diameter. The large plaque viruses may thus escape from inhibition by virtue of 'ignoring' surfaces with a high density of branched sialoglycoconjugates. □

Received 14 February; accepted 31 March 1994.

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ACKNOWLEDGEMENTS. We thank S. Gamblin for discussions and CHESS for time on the F1 beamline and the staff of CHESS and MacCHESS for their help. This work has been supported by grants from the National Cancer Institute of the NIH (to T.L.B. and S.C.H.) and by an EMBO postdoctoral fellowship (to T.S.).

ERRATUM

A relict refractory inclusion in a ferromagnesian chondrule from the Allende meteorite

Keiji Misawa & Takashi Fujita

Nature **368**, 723–726 (1994)

IN this letter, part of the legend to Fig. 2 was omitted. The penultimate sentence should read:

“The spinel exhibits Fe–Mg zoning: the core and the Ca, Al-rich silicate inclusions are iron-free, whereas in the rim (~20–40 µm width) and along the cracks the FeO concentration rises sharply (MgO ≈ 4 wt %). The silicone inclusions are composed of two phases: a Ca, Al-rich phase (low MgO content) and Ti, Al-pyroxene, fassaite.” □

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