**Supplemental methods**

**Flow cytometry**

For detection of NCAM and HS, cells were stained with anti-NCAM antibody (304601, BioLegend) or anti-HS antibody (370255, AMS Biotechnology). Mouse IgG2a (BioLegend) and mouse IgM (BioLegend) represented controls. For quantification of RABV and recombinant G protein (rG) bound on cells, RABV or rG were pretreated in PBS with/without heparin, and incubated with dissociated cells at 4°C for 1 h. After washing with PBS, RABV and rG on cells were stained by anti-RABV G antibody (3R7-4F1, HyTest). Bound antibodies were labeled by Alexa Fluor 488-conjugated anti-mouse IgG (Invitrogen) or R-phycoerythrin-conjugated anti-mouse IgM (Beckman Coulter). Data were collected with the FACS Canto (BD Biosciences) and analyzed with the Flowjo software (Tree Star).

**Expression and purification of soluble CVS G protein**

The ectodomain of CVS G was cloned into pFastBac 1 (Invitrogen) with a honeybee melittin secretion signal sequence at the N-terminus of the protein and a HRV 3C protease cleavage site, a foldon trimerization domain and a hexahistidine tag at the C-terminus of the protein. The recombinant baculovirus was generated using the Bac-to-Bac baculovirus expression system (Invitrogen). The culture supernatant was collected from the baculovirus-infected Sf9 cells maintained in Sf-900 III SFM (Gibco). The hexahistidine tagged-recombinant RABV G was purified by affinity chromatography with a HisTrap column and additional gel filtration with HiLoad 16/60 Superdex 200 column (GE Healthcare).

**Surface plasmon resonance (SPR) analysis**

SPR measurements were performed at 25°C using the Biacore 3000 instrument and BIAevaluation software (GE Healthcare) according to the methods of Mathie et al. with slight modifications [1]. Briefly, around 40 response units of heparin-biotin (Sigma-Aldrich) was immobilized on the Sensor chip SA (GE Healthcare) with flow cell 1 as a reference cell. Recombinant RABV G protein was serially diluted in HBS-P buffer [10 mM HEPES (pH 7.4), 150 mM NaCl, 0.005% (v/v) Tween 20] and used as the analytes to flow over the chip. The flow cells were exposed to the analytes at a flow rate of 20 μl/min for measurement of response units. At the end of each cycle, the chip surface was regenerated with 0.05% SDS for 1 min and then 2 M NaCl for 2 min. For binding competition assays, recombinant RABV G diluted at 25 μg/ml were treated with heparin, de-N-sulfated heparin, 2-O-desulfated heparin or 6-O-desulfated heparin at 1 to 100 μg/ml for 15 min and then used as the analytes.

**Gene quantitation by real-time PCR**

Total RNA was extracted from SK-N-SH cells and reverse transcribed into cDNA with SuperScript VILO Master Mix (Invitrogen). The expression of sulfotransferases was quantified by real-time PCR with SYBR Premix Ex Taq II (Takara Bio). Oligonucleotide primers for *NDST1*, *NDST2*, *NDST3* and *NDST4* [2] and *2OST*, *6OST1*, *6OST2* and *6OST3* [3] were previously described. The expression of *β-actin* was quantified with Brilliant III Ultra-Fast QPCR Master Mix (Agilent) using TaqMan Gene Expression Assay (ACTB, Hs01060665\_g1).

**References**

1. Mathieu C, Dhondt KP, Châlons M, et al. Heparan sulfate-dependent enhancement of henipavirus infection. MBio **2015**; 6:e02427.

2. Krenn EC, Wille I, Gesslbauer B, Poteser M, van Kuppevelt TH, Kungl AJ. Glycanogenomics: a qPCR-approach to investigate biological glycan function. Biochem Biophys Res Commun **2008**; 375:297-302.

3. Götte M, Spillmann D, Yip GW, et al. Changes in heparan sulfate are associated with delayed wound repair, altered cell migration, adhesion and contractility in the galactosyltransferase I (beta4GalT-7) deficient form of Ehlers-Danlos syndrome. Hum Mol Genet **2008**; 17:996-1009.