N-Glycosylation is a Potent Regulator of Prion Protein Neurotoxicity

Kevin M. Schilling^a, Pooja Jorwal^b, Natalia C. Ubilla-Rodriguez^a, Tufa E. Assafa^a, Jean R. P. Gatdula^b, Janelle Vultaggio^b, David A. Harris^{*b}, and Glenn L. Millhauser^{*a}

 ^a 1156 High Street. Department of Chemistry and Biochemistry, University of California, Santa Cruz, CA, 95064, United States
^b 72 E. Concord St. Department of Biochemistry, Boston University, Chobanian & Avedisian School of Medicine, Boston, MA, 02118, United States

*Correspondence: DA Harris: daharris@bu.edu, or GL Millhauser: glennm@ucsc.edu +1 617-358-4280 (phone) +1 831 459 2176 (phone) +1 617-358-4353 (fax) +1 831 459 2935 (fax)

Supporting Information

Figure S1: Representative traces from whole cell patch clamp recordings.

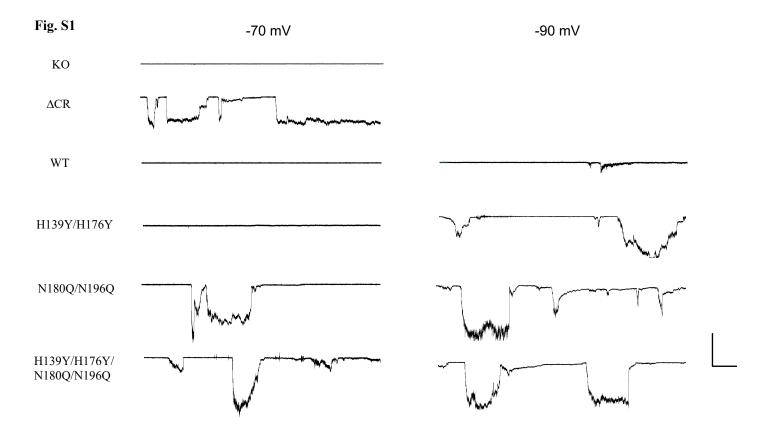


Figure S1: Representative traces of currents recorded from either untransfected N2a cells (KO) or N2a cells expressing following forms of PrP : Δ CR, WT, H139Y/H176Y, N180Q/N196Q and H139Y/H176Y/N180Q/N196Q. Quantification of the currents is shown in figure 7. Holding potential was either -70 mV (left) or -90 mV (right). Scale bars: 1 nA, 30 s.