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Supplementary Materials for

Gain-of-function mutations in protein kinase Cα (PKCα) may promote synaptic defects in Alzheimer's disease

Stephanie I. Alfonso, Julia A. Callender, Basavaraj Hooli, Corina E. Antal, Kristina Mullin, Mathew A. Sherman, Sylvain E. Lesné, Michael Leitges, Alexandra C. Newton,* Rudolph E. Tanzi,* Roberto Malinow*

*Corresponding author. Email: anewton@ucsd.edu (A.C.N.); tanzi@helix.mgh.harvard.edu (R.E.T.); rmalinow@ucsd.edu (R.M.)

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The PDF file includes:

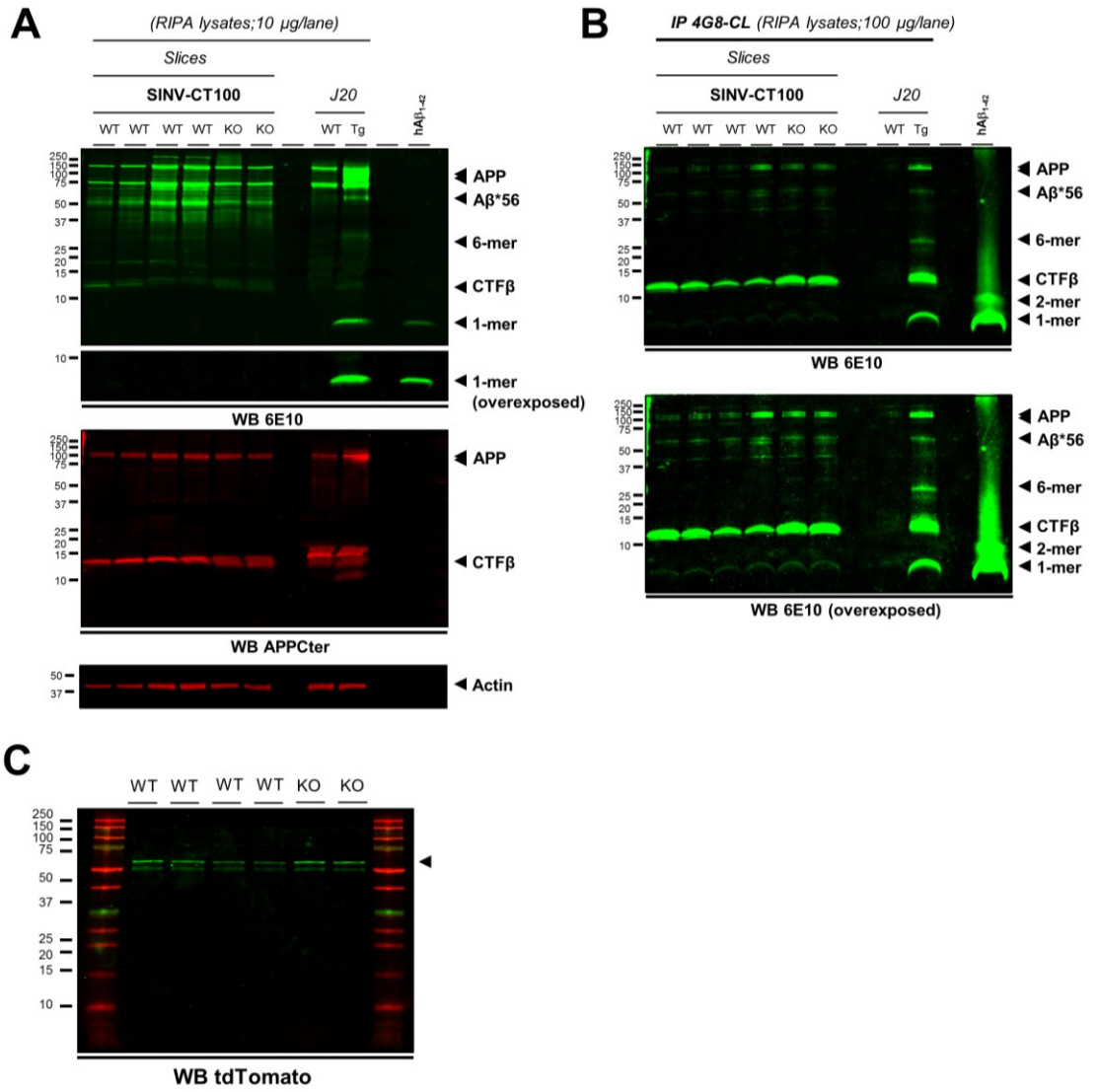
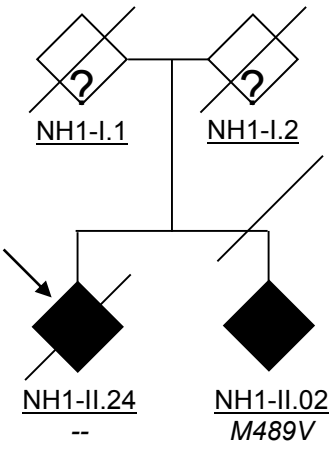
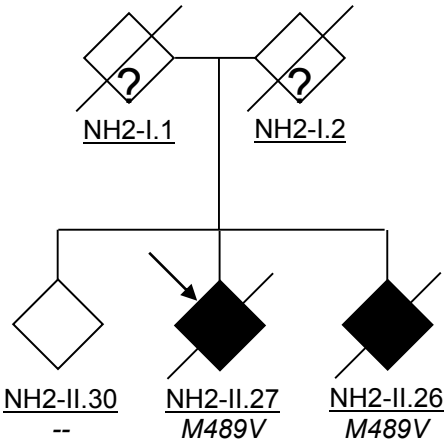


Figure S1. Aβ abundance in brain slices infected with virus producing CT100 is similar in wild-type and *PRKCA*$^{-/-}$ slices. (**A**) Representative western blot images of Aβ levels detected in brain slices infected with CT100 using 6E10 (green, top) and APPCter (red, middle) antibodies. Actin was used as internal loading control (red, bottom). RIPA lysates of forebrains from 17-month-old WT and J20 mice served as negative and positive controls as well as recombinant human Aβ1-42 peptides (3 ng). (**B**) Due to the low abundance of Aβ by direct SDS-PAGE analysis, Aβ levels were further determined by immunoprecipitation (IP) using cross-linked 4G8 beads (5 µg/IP). The lower insert corresponds to an overexposed image scan. (**C**) Western blot image illustrating the expression of tdTomato following infection of WT and KO slices. Note: CT100 = CTFβ.

NH01



NH02



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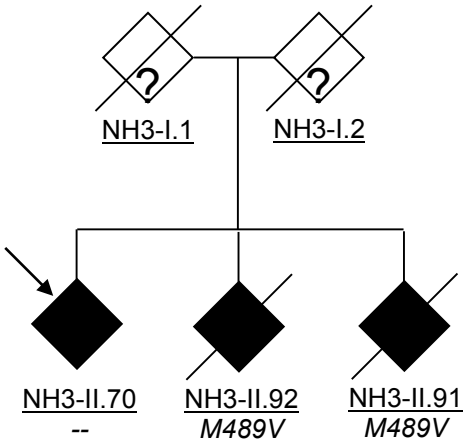

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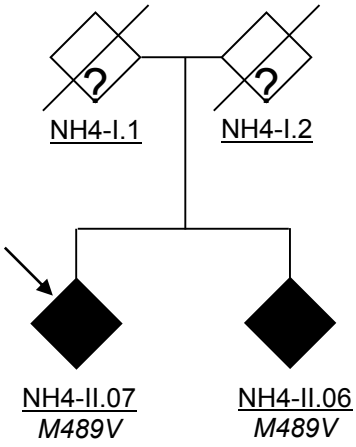

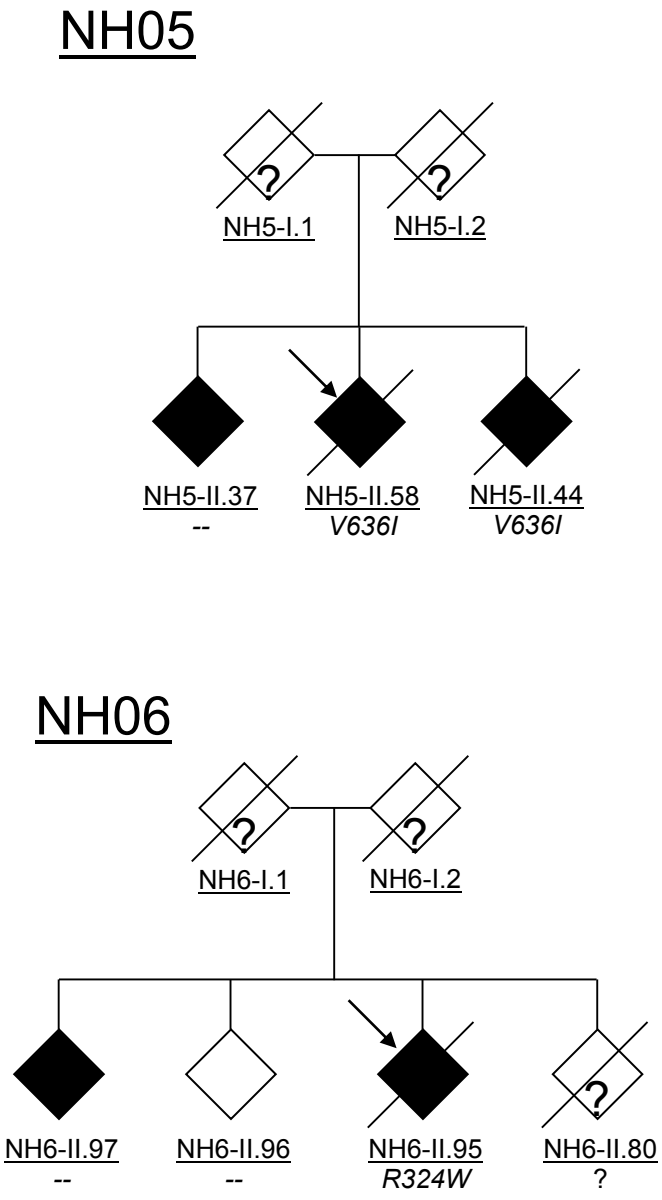

Figure S2: Pedigree charts of the NIMH families found to carry rare PKCα variants. Information for each individual is (from top to bottom): Individual ID, genotype of rare variant in PKCα if present, "--" for wild-type. Probands are indicated by arrows. No DNA or clinical information was available from the founders ("?") and in NH6-II.80.