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Supporting information for article:

ER stress sensor PERK luminal domain functions as a molecular chaperone to interact with misfolded proteins

Peng Wang, Jingzhi Li and Bingdong Sha

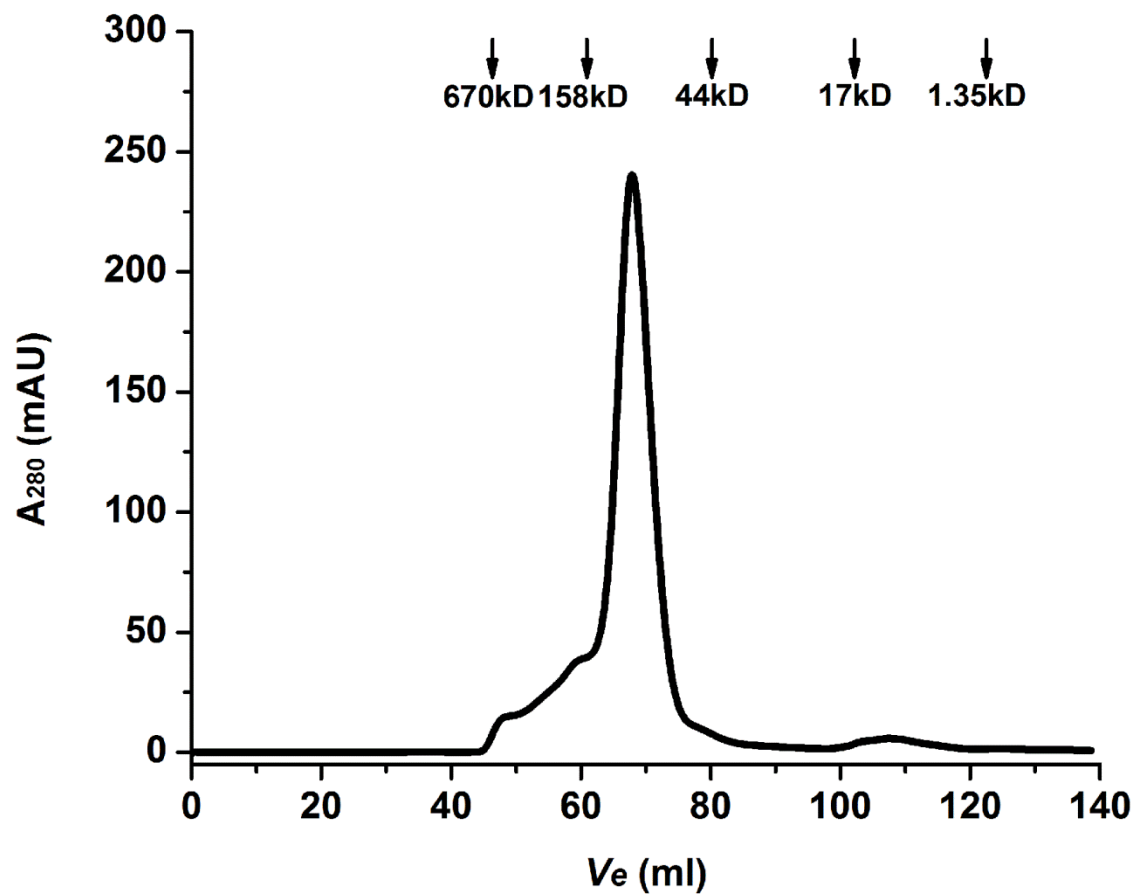
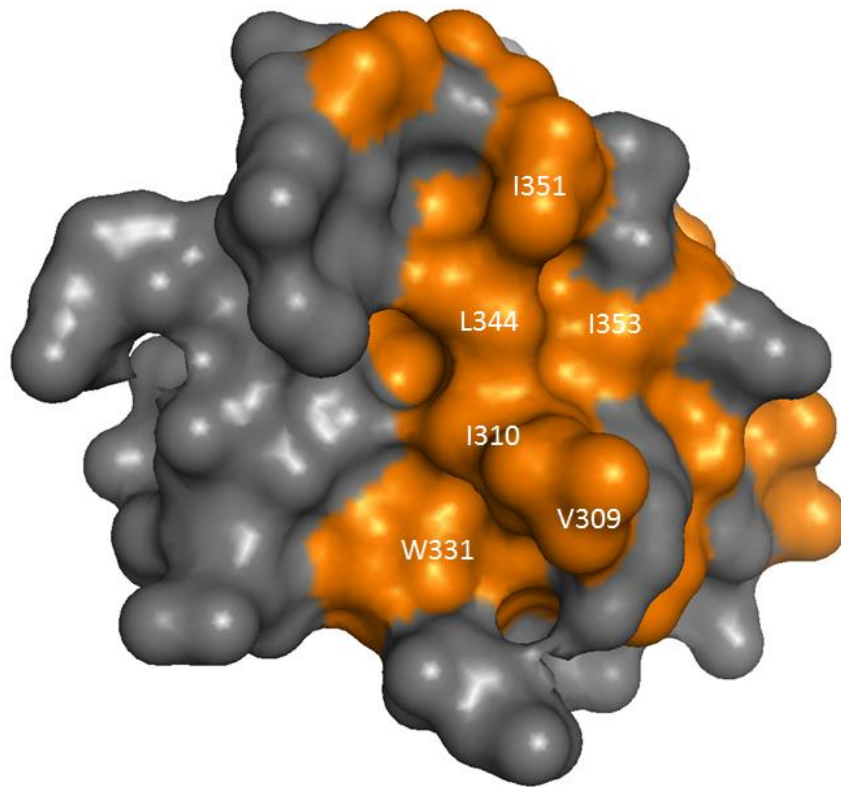
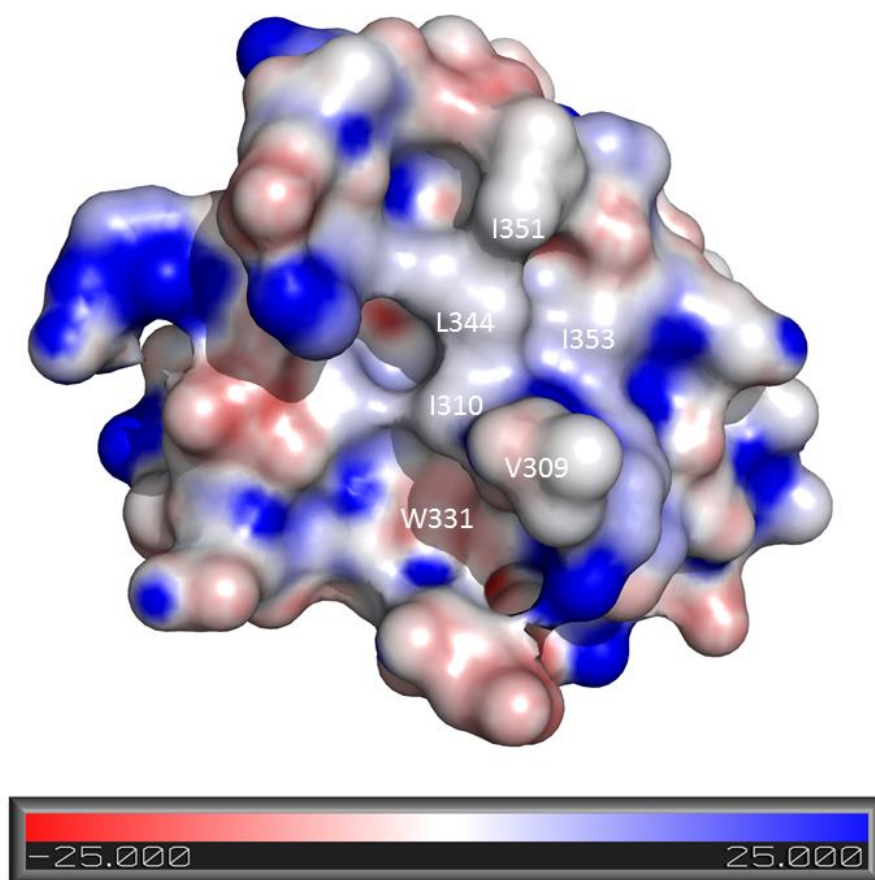


Figure S1 The gel filtration profile of recombinant human PERK luminal domain. 5ml PERK samples were loaded on Superdex 200 (GE Healthcare) mounted on the AKTA FPLC system (GE Healthcare). The flow rate was set at 1ml/min. The peak positions for the molecular standards are labeled.



(a)



(b)

Figure S2 The molecular surface drawing of PERK β -sandwich domain by Pymol. The orientation of the molecule in the figure is similar to that in the top panel of Fig. 3b. a) The hydrophobic surface drawing of PERK β -sandwich domain. The exposed hydrophobic regions are indicated in orange. The residues that constitute the large hydrophobic patch are labeled. b) The surface potential drawing of PERK β -sandwich domain.

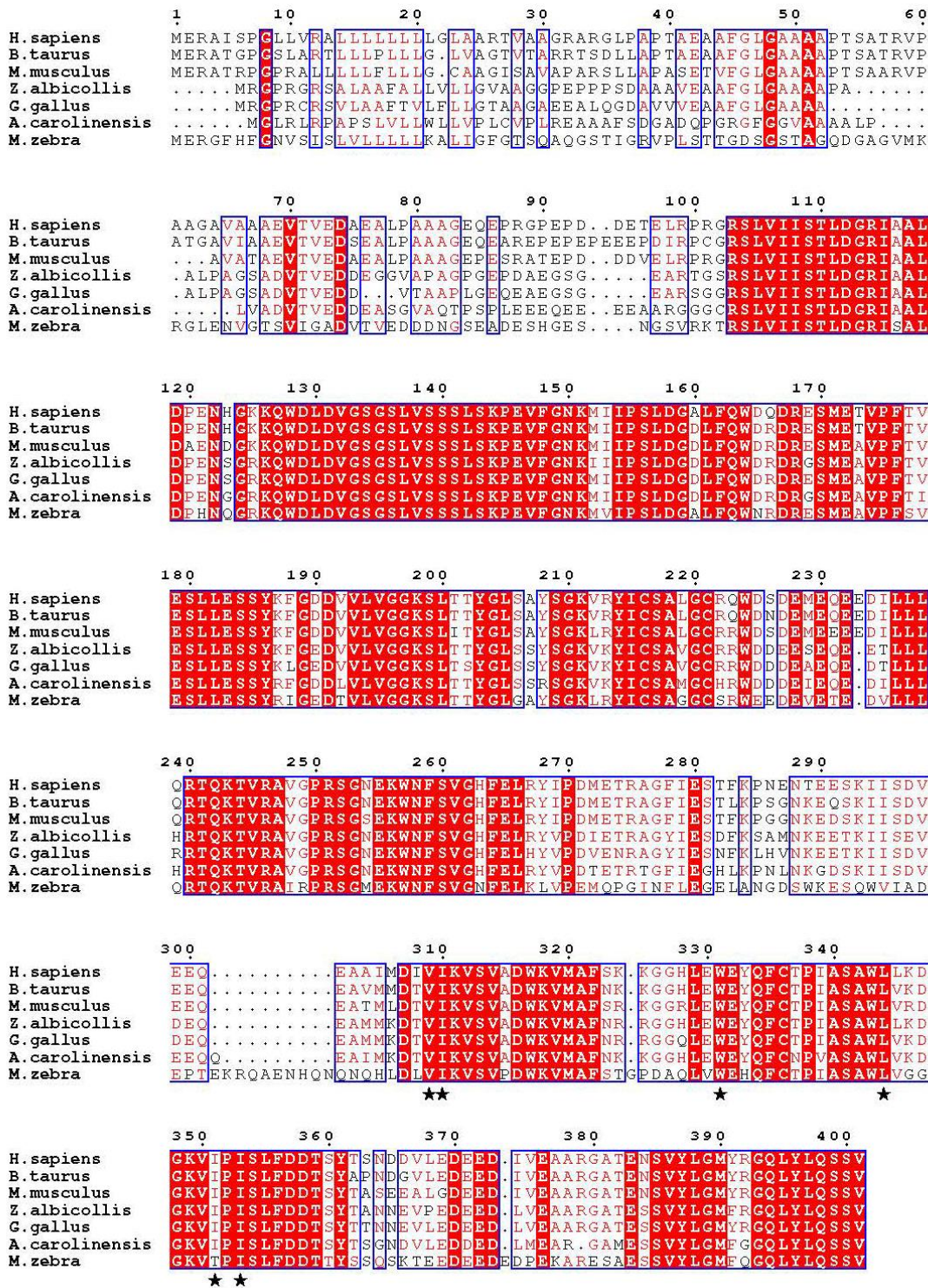


Figure S3 The sequence alignment of PERK luminal domain from various species. The residue numbering in the figure is based on human PERK sequence. The residues that may be involved in forming the putative binding site for the misfolded proteins are marked by stars.

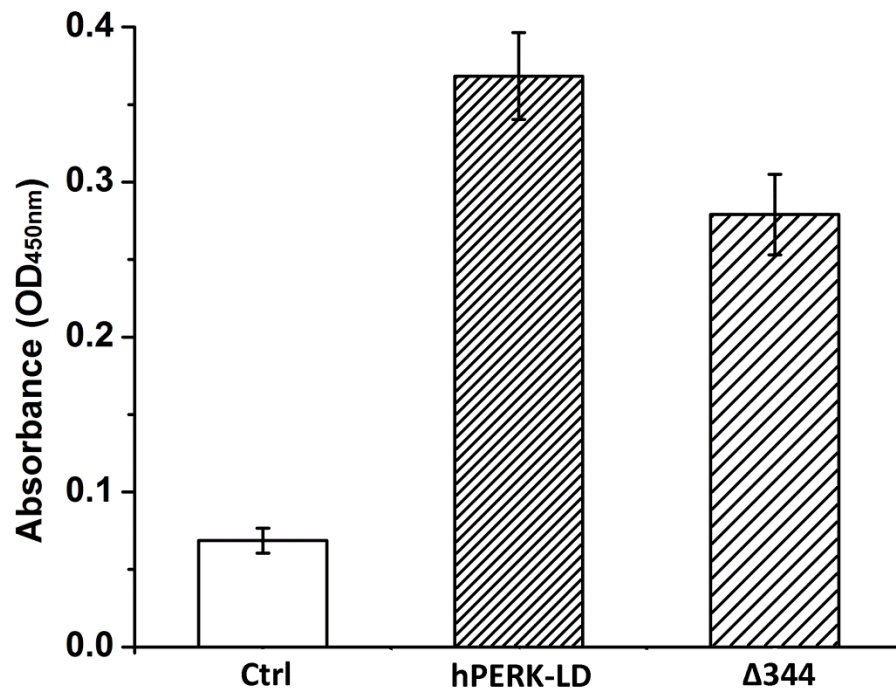


Figure S4 The ELISA assay showing that the PERK $\Delta 344$ mutant exhibits reduced binding ability to denatured model protein. In this experiment, PERK luminal domain (PERK LD) or the $\Delta 344$ mutant at 50ug/ml (0 as control) was coated on the wells, 100ul chemically denatured rhodanese at 5ug/ml were added. The bound rhodanese can be detected by use of the anti-rhodanese antibody. The OD450 readings are shown in open bars. The standard derivations of three independent experiments are indicated in the bars.