Accepted Manuscript

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PII:	\$0969-9961(16)30045-6
DOI:	doi: 10.1016/j.nbd.2016.03.001
Reference:	YNBDI 3714

To appear in: Neurobiology of Disease

Received date:2 November 2015Revised date:25 January 2016Accepted date:2 March 2016

Please cite this article as: Scott-McKean, Jonah J., Surewicz, Krystyna, Choi, Jin-Kyu, Ruffin, Vernon A., Salameh, Ahlam I., Nieznanski, Krzysztof, Costa, Alberto C.S., Surewicz, Witold K., Soluble prion protein and its N-terminal fragment prevent impairment of synaptic plasticity by $A\beta$ oligomers: Implications for novel therapeutic strategy in Alzheimer's disease, *Neurobiology of Disease* (2016), doi: 10.1016/j.nbd.2016.03.001

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ACCEPTED MANUSCRIPT

Soluble Prion Protein and Its N-terminal Fragment Prevent Impairment of Synaptic

Plasticity by Aβ Oligomers: Implications for Novel Therapeutic Strategy in

Alzheimer's Disease

Jonah J. Scott-McKean^{a1}, Krystyna Surewicz^{b1}, Jin-Kyu Choi^b, Vernon A. Ruffin^{b2}, Ahlam I. Salameh^b, Krzysztof Nieznanski^d, Alberto C.S. Costa^{a,c}, and Witold K. Surewicz^{b*}

^aDivision of Pediatric Neurology, Department of Pediatrics, ^bDepartment of Physiology and Biophysics, ^cDepartment of Psychiatry, Case Western Reserve University, Cleveland, OH, USA 44116, and ^dDepartment of Biochemistry, Nencki Institute of Experimental Biology, 02-093Warsaw, Poland

¹These authors contributed equally to this work.

²Present address: Department of Natural Resources, Virginia Union University, Richmond, VA 232250.

*To whom correspondence should be addressed: Witold K. Surewicz, Department of Physiology and Biophysics, Case Western Reserve University, School of Medicine E605, 10900 Euclid Ave., Cleveland, OH 44116-4970; Tel.: (216) 368-0139; Email: witold.surewicz@case.edu Download English Version:

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