Supporting Information

A cellular model to monitor proteasome dysfunction by alpha-synuclein Takashi Nonaka and Masato Hasegawa

Department of Molecular Neurobiology, Tokyo Institute of Psychiatry, Tokyo Metropolitan Organization for Medical Research, 2-1-8 Kamikitazawa, Setagaya-ku, Tokyo 156-8585.

Supplementary figure legends

Fig. S1 Biochemical fractionation of cells expressing both of GFP-CL1 and alpha-synuclein. SH-SY5Y cells were transfected with GFP-CL1 and alpha-synuclein wild-type (WT), A53T or Δ73-83 deletion mutant. Cells were harvested, and cell lysate was fractionated as described in Experimental procedure and analysed by immunoblotting using anti-GFP and anti-Syn102 antibodies. TS, Tris-soluble fraction; TX, Triton X-100-soluble fraction; ppt, SDS-soluble fraction.

Fig. S1 Nonaka et al

