Autophagy–lysosome pathway alterations and alpha-synuclein up-regulation in the subtype of neuronal ceroid lipofuscinosis, CLN5 disease

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Supplementary information

**Figure S1. CLN5 disease patient cells are capable of degrading P62 via lysosomes.**

WT and Stable CLN5 KD HeLa cells were incubated with HBSS for 0, 2, 4, 8 h in the presence of cycloheximide and bortezomib. Samples were analyzed by immunoblotting. β-actin was blotted as a loading control. For degradation quantification (N=3), P62 was normalized with β-actin signal in each lane. 0 h in each cell line was set as 1. Error bar represents SEM.
Figure S2. α-syn is not up-regulated by transient knockdown of CLN5. (A) Human fibroblasts were transfected with siRNA against CLN5 or control for 72 or 90 h as indicated. C: control fibroblasts; P: Patient fibroblasts. (B) SH-SY5Y cells were transfected with siRNA against CLN5 or control for 72 h. Samples were analyzed by immunoblotting. GAPDH was blotted as a loading control.