**Results**

NGly-1 knockout (KO) in K562 cells was verified by Western blot and stable cell line analysis. A full functional deglycosylation reporter for RTA was created. We exposed our K562 cell line to increasing concentrations of bortezomib (a proteasome inhibitor) and observed a ~2-fold higher sensitivity of NGLY1 KO K562 cells to treatment compared to WT K562 cells.

Having linked expression of an aggregation-prone protein to NGLY1, we then tested for protein aggregation in the NGLY1-deficient K562 cells using the Prion Protein Aggregation Assay Kit (Echelon Biosciences). We found that there was a trend toward increased staining of protein aggregation in NGLY1 KO K562 cells. However, it remains to be determined if aggregates contain α-synuclein.

**Conclusion**

1. NGLY1 deficiency results in the accumulation of aggregates.
2. NGLY1-dependent protein accumulation can be decreased through the activation of autophagy.
3. It is possible that modulation of autophagy is a therapeutic option for NGLY1-deficient patients.

**Next Steps**

1. Determine the subcellular localization of the aggregates.
2. Determine if a specific sub type of autophagy is effected by NGLY1.
3. Test other autophagy modifying compounds to determine their influence on NGLY1 deficient systems.

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**References**

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