

Molecules from marine invertebrate venoms recover cathepsin D activity, impaired by oligomerized A β 42, in a neuronal cell model of Alzheimer's disease

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INTRODUCTION & AIM

- Alzheimer's disease (AD) is characterized by the accumulation of amyloid- β (A β).
- As a result, it disrupts autophagy-lysosomal pathway in neurons, specially cathepsin D.

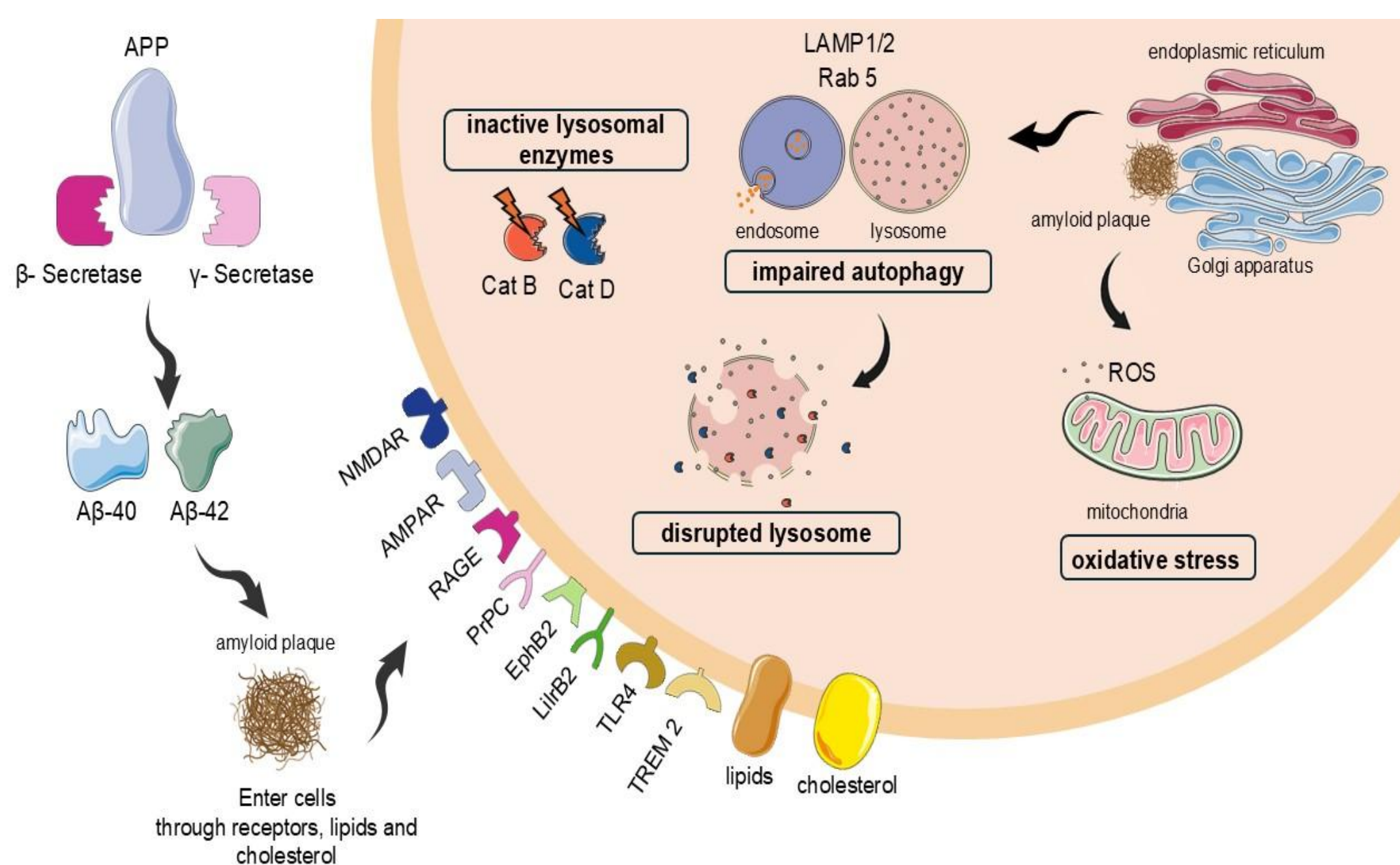


Figure 1: Alzheimer's disease pathology correlate with cathepsin D. Mançano et al., 2024.

METHOD

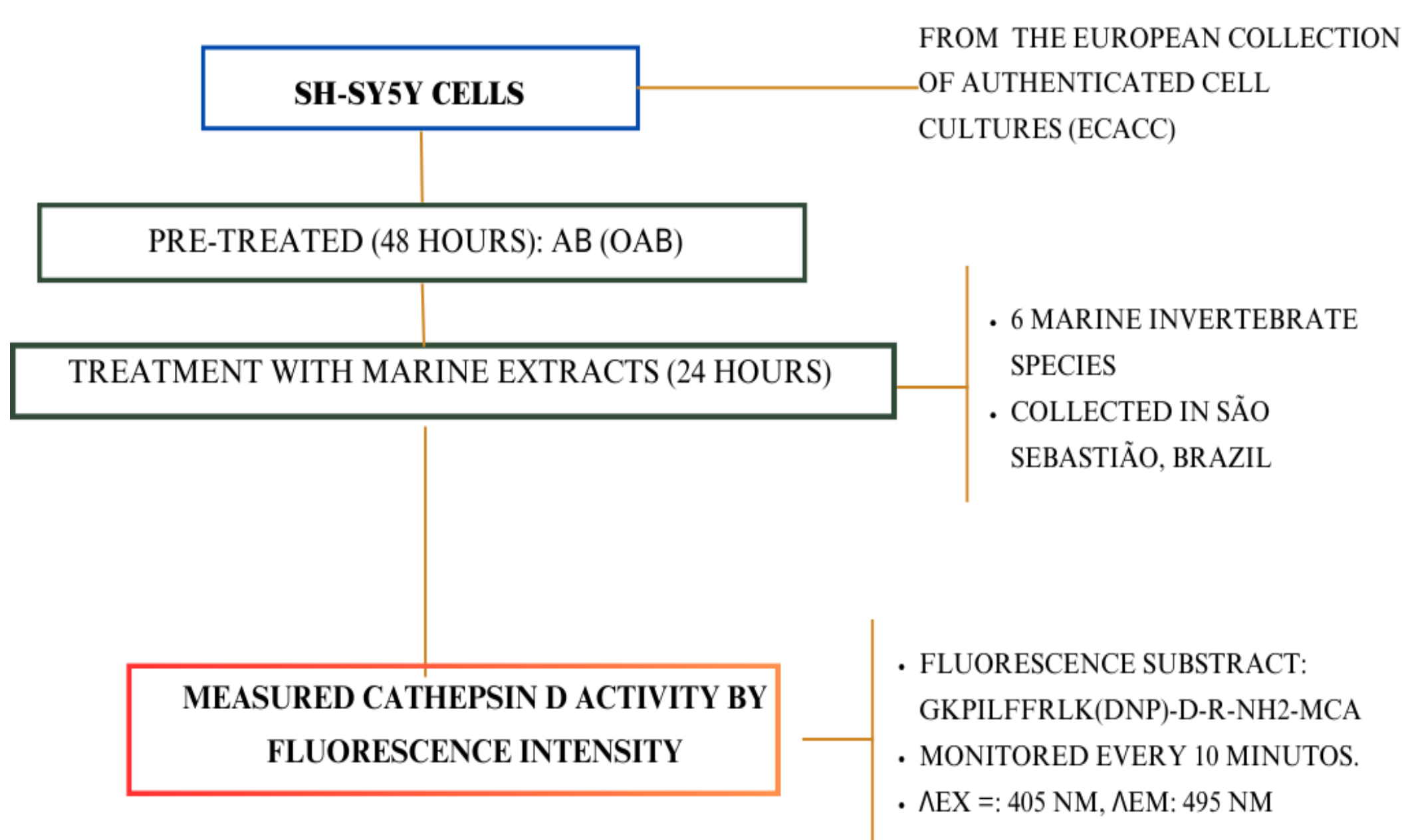


Figure 2: Methodology for cathepsin D enzymatic activity evaluation.

RESULTS & DISCUSSION

- oA β treatment decreased cathepsin D activity, measured by its velocity in AUF/min, by 40% compared to control (cells without treatment).

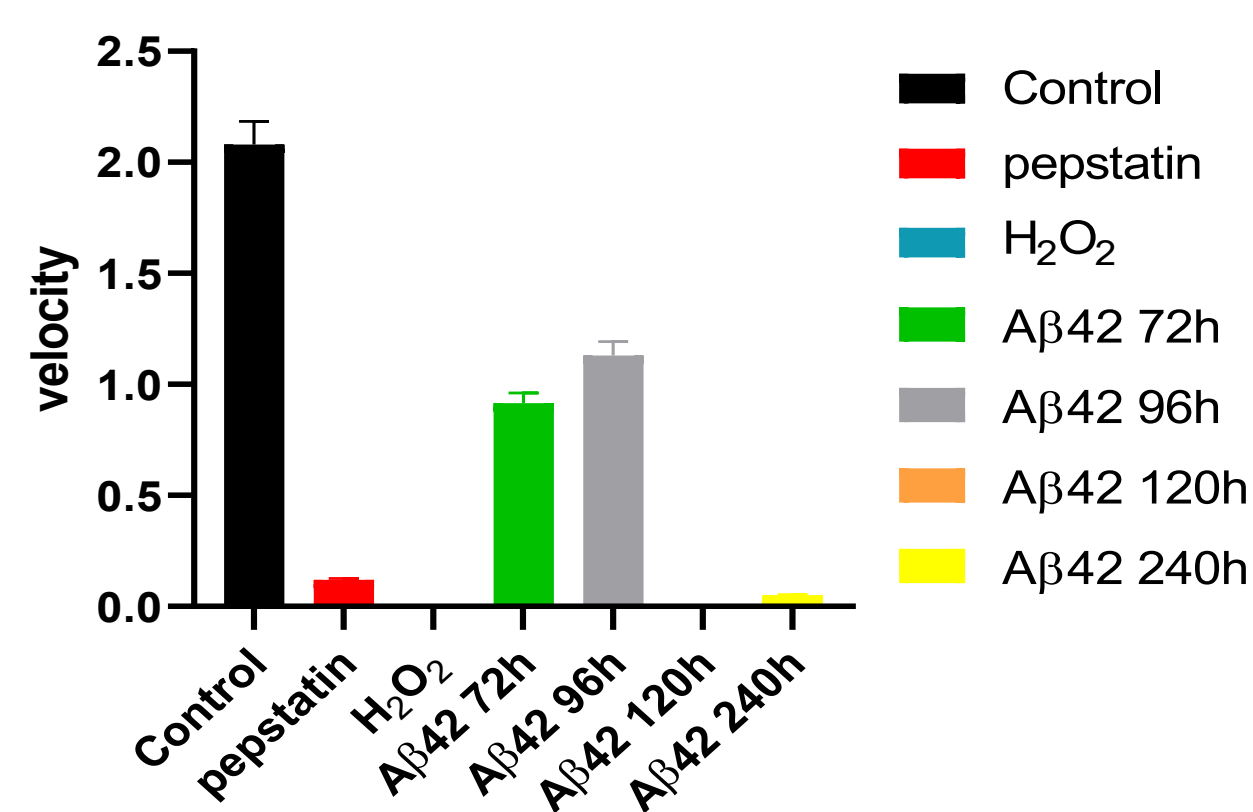


Figure 3: Velocity of reaction of cathepsin D from SH-SY5Y cells over synthetic substrates. Control= cells without treatment.

- Venoms from the corals *Renilla reniformis* and *Tubastraea tagusensis*, sea anemone *Anthopleura cascaia* and sea urchin *Echinometra lucunter* are recovered the cathepsin D activity.

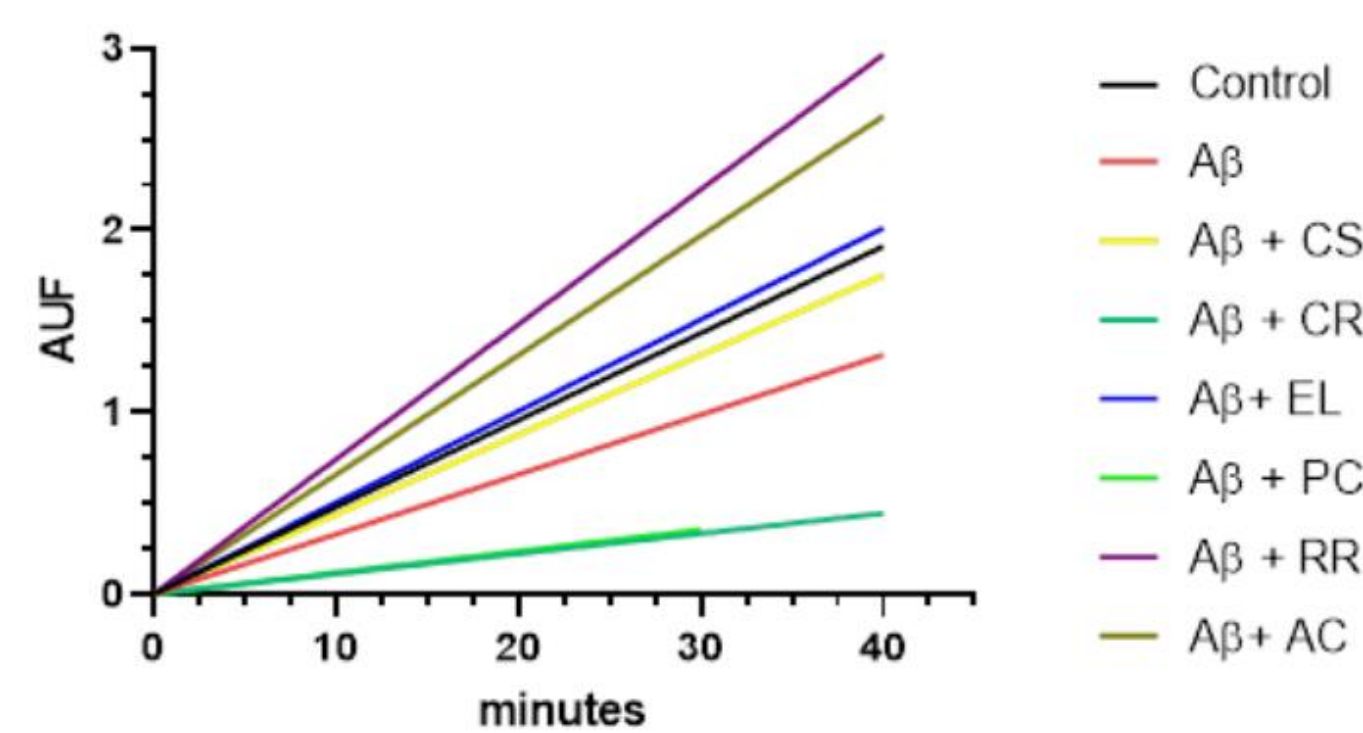


Figure 4: Cathepsin D enzymatic activity of six marine extracts, shown as arbitrary units of fluorescence (AUF) over time (minutes). Control= cells without treatment.

CONCLUSION

- The findings demonstrated a reduction or inhibition of cathepsin D enzymatic activity in neurons exposed to the oligomerized A β peptide.
- Marine-derived molecules may hold a therapeutic candidates for restoring lysosomal function.

FUTURE WORK / REFERENCES

- It's interesting to analyze the *A. cascaia* fractions, which exhibited the most intense activity, with the objective to identify the fraction of the molecule that presents the best answer.