Supplementary Figures

<u>Supplementary Fig. 1.</u> Neuronally differentiated SHSY5Y cells have elevated CGB levels but expression of CGB and its fragments did not alter resting calcium levels. (A) Western blot showing increased CGB levels after treatment with retinoic acid and recombinant human nerve growth factor to induce differentiation (compare left two lanes). NIH3T3 cells do not express CGB (third lane). PC12 cells have an abundance of CGB (far right lane), but note that the apparent molecular weight is different in rat cells (PC12) when compared to CGB from human cells (SHSY5Y). (B) Expression of CGB fragments in SHSY5Y cells did not change cytosolic calcium levels (nM) (C) Expression of CGB fragments in SHSY5Y cells did not alter resting levels of ER calcium (F/Fo).

<u>Supplementary Fig. 2.</u> Levels of cytosolic calcium and ER resting calcium levels in NIH3T3 cells were unchanged by expression of CGB fragments. (A) Expression of CGB fragments in NIH3T3 cells did not change cytosolic calcium levels (nM) (B) Expression of CGB fragments in NIH3T3 cells did not alter resting levels of thapsigargin releasable ER calcium (F/Fo).

<u>Supplementary Fig. 3.</u> **InsP₃R type I was ubiquitously expressed in SHSY5Y cells.** (**A**) The InsP₃R type I, which is the predominant isoform in neurons, was found to be equally distributed in the soma and growth cones of SHSY5Y cells. (**B**) InsP₃R type I levels are similar when CGB constructs are transfected into NIH3T3 cells.

<u>Supplementary Fig. 4.</u> Expression of CGB and C-def-CGB induced de novo secretory granule biogenesis. CGB full length, C-def-CGB, and N-def-CGB were expressed in NIH3T3 cells, and images were taken at 100x with a light microscope. C-def-CGB and CGB were able to induce *de novo* secretory vesicle biogenesis, whereas expression of N-def-CGB failed to do so.











InsP3R Type I



beta-actin

