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Data Article

Comparison of the amyloid pore forming properties of rat and human Alzheimer's beta-amyloid peptide 1-42: Calcium imaging data

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ABSTRACT

The data here consists of calcium imaging of human neuroblastoma SH-SY5Y cells treated with the calcium-sensitive dye Fluo-4AM and then incubated with nanomolar concentrations of either human or rat Alzheimer's β -amyloid peptide A β 1-42. These data are both of a qualitative (fluorescence micrographs) and semi-quantitative nature (estimation of intracellular calcium concentrations of cells probed by A β 1-42 peptides vs. control untreated cells). Since rat A β 1-42 differs from its human counterpart at only three amino acid positions, this comparative study is a good assessment of the specificity of the amyloid pore forming assay. The interpretation of this dataset is presented in the accompanying study "Broad neutralization of calcium-permeable amyloid pore channels with a chimeric Alzheimer/Parkinson peptide targeting brain gangliosides" [1].

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Specifications Table

Subject area	<i>Neurosciences</i>
More specific subject area	<i>Molecular basis of neurodegenerative diseases</i>
Type of data	<i>Text file and figures</i>
How data was acquired	<i>Fluorescent microscope</i>
Data format	<i>Analyzed</i>
Experimental factors	<i>Cells treated with the calcium-sensitive dye Fluo-4AM and then incubated with either human or rat Aβ1-42 peptide.</i>
Experimental features	<i>Control cells are preloaded with Fluo-4AM and then treated with buffer alone to determine background intracellular calcium levels. This background is subtracted from the values induced by either human or rat Aβ1-42.</i>
Data source location	<i>Not applicable</i>
Data accessibility	<i>All data are presented in this article</i>

Value of the data

- The data provides a comparative study of Ca²⁺ fluxes induced by human and rat forms of A β 1-42 peptide.
- The comparison of the Ca²⁺ fluxes induced by human and rat A β 1-42 peptides may be used as an internal reference to validate this assay for studying amyloid pore formation induced by any amyloid protein in living neural cells.
- Researchers interested in testing amyloid pore formation and inhibitors of amyloid pores might carefully choose positive and negative controls with calibrated compounds.

1. Data

Amyloid pores [1–4] are responsible for a dramatic increase of intracellular Ca²⁺ levels in brain cells that can be measured by fluorescence microscopic imaging [5–12]. The dataset presented here contains the values of intracellular Ca²⁺ concentrations induced by human and rat A β 1-42 peptides in neural SH-SY5Y cells. Amino acid sequence alignments of these peptides are presented in Fig. 1. Quantitative data (histograms) are given in Fig. 1, and fluorescence micrographs are shown in Fig. 2.

2. Experimental design, materials and methods

2.1. Materials

SH-SY5Y cells were obtained from ATCC. Dulbecco's Modified Eagle Medium: Nutrient Mixture F12 (DMEM/F12), HBSS, glutamine and penicillin/streptomycin were furnished by Gibco. Fluo-4AM was from Invitrogen. Human and rat A β 1-42 peptides were purchased from rPeptide. The purity of these peptides was > 95% as assessed by HPLC.

2.2. Cell culture

SH-SY5Y cells were cultured in DMEM/F12 supplemented with 10% fetal calf serum, glutamine (2 mM) and penicillin (50 U/mL)/streptomycin (50 μ g/mL) and maintained at 37 °C with 5% CO₂. Cells were passaged twice a week and not used beyond passage 25.

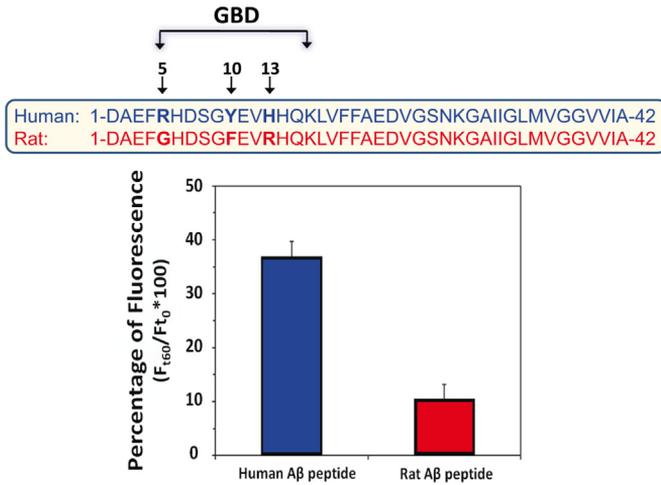


Fig. 1. Ca^{2+} fluxes induced by human and rat A β 1–42 peptides in SH-SY5Y cells: a comparative study. Amino acid sequence alignments (upper panel) show that human and rat A β 1–42 peptides differ at only three positions, all located in the ganglioside-binding domain (GBD) [1,12]. SH-SY5Y cells treated with the calcium dye Fluo-4AM were incubated with human A β 1–42 (blue histogram, $n=87$) or rat A β 1–42 (red histogram, $n=120$) and calcium-dependent fluorescence was recorded after 60 min of incubation with the indicated peptides (lower panel). Results are expressed as mean \pm SEM. Statistical significance between human and rat A β 1–42 ($p=2.75 \times 10^{-11}$) was evaluated by using Student's t -test.

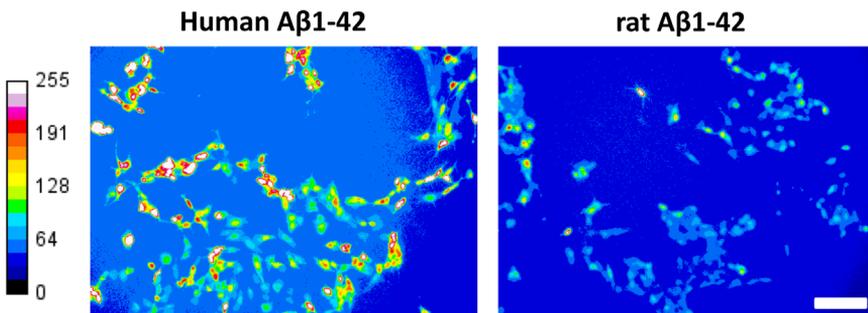


Fig. 2. Cell imaging of Ca^{2+} fluxes induced by human and rat A β 1–42 peptides in SH-SY5Y cells: a comparative study. The images show pseudocolor representations of cells (scale bar: 100 μm), warmer colors corresponding to higher fluorescence. The photographs are taken after 60 min of incubation with each peptide (same experimental conditions as in Fig. 1).

2.3. Calcium measurements

SH-SY5Y cells were plated (45,000 cells/dish) in 35 mm culture dishes and grown during 72 h at 37 $^{\circ}\text{C}$. They were loaded with 5 μM Fluo-4AM for 30 min in the dark as previously described [1,5]. The calcium fluxes were estimated by measuring the variation of cell fluorescence intensity after the injection of either rat or human A β 1–42 (220 nM) into the recording chamber directly above an upright microscope objective (BX51W Olympus) equipped with an illuminator system MT20 module. Fluorescence emission at 525 nm was imaged by a digital camera CDD (Hamamatsu ORCA-ER) after fluorescence excitation at 490 nm. Time-lapse images (1 frame/10 s) were collected using the CellIR Software (Olympus). Fluorescence intensity were measured from region of interest (ROI) centered on individual cells. Signals were expressed as fluorescence after treatment (F_{t60}) divided by the fluorescence before treatment (F_0) and multiplied by 100. The results were averaged and the fluorescence of control is subtracted of each value. The experiments were performed at 30 $^{\circ}\text{C}$ during 60 min. In the

pseudocolor representations of cells, warmer colors correspond to higher fluorescence and thus to higher Ca^{2+} levels.

3. Statistical analysis

Quantitative data are expressed as mean \pm S.E.M. and the statistical significance was assessed with the Student's *t*-test.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.dib.2016.01.019>.

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