Synaptamide is a potent mediator for neurogenic differentiation of NSCs acting through PKA/CREB activation.

**Target:**
in vitro: Synaptamide inhibits forskolin-mediated cAMP production (IC50 =6 μM) in CHO-HCR cells. Synaptamide decreases the viability of the LNCaP and PC3 prostate cancer cell lines (IC50=120-130 μM) grown in media containing 10% fetal bovine serum. [1]

Synaptamide is an endogenous DHA metabolite with endocannabinoid-like structure, promotes neurite growth, synaptogenesis and synaptic function. Synaptamide potently induces neuronal differentiation of NSCs. Treatment of NSCs with Synaptamide at low nanomolar concentrations significantly increased the number of MAP2 and Tuj-1 positive neurons with concomitant induction of PKA/CREB phosphorylation. [2]

**PROTOCOL (Extracted from published papers and Only for reference)**

Cell assay [1] To evaluate cytotoxicity, lactate dehydrogenase (LDH) released from NSCs was assayed using CytoTox 96 Non-Radioactive Cytotoxicity Assay kit. NSCs (2.5 × 105 NSCs in 0.5 mL media) were treated with different concentrations of Synaptamide or DHA for 6 days. Fifty μL supernatant was collected from the culture, transferred to 96-well plates, and 50 μL of substrate solution was added. The enzymatic reaction was allowed to proceed for 30 min at room temperature, protected from light. After stopping the reaction by adding 50 μL/well of the stop solution, the absorbance was measured at 490 nm using a plate reader. The released LDH activity was normalized to the total LDH activity determined from the cell lysate and supernatant. The final data were expressed as % of untreated control.

**References:**
