

# Lead Optimization of CRAC Channel Inhibitors for the Treatment of Alzheimers Disease

<https://neurodegenerationresearch.eu/survey/lead-optimization-of-crac-channel-inhibitors-for-the-treatment-of-alzheimers-disease/>

## Principal Investigators

RUDNIC, EDWARD

## Institution

VIVREON BIOSCIENCES, LLC

## Contact information of lead PI Country

USA

## Title of project or programme

Lead Optimization of CRAC Channel Inhibitors for the Treatment of Alzheimers Disease

## Source of funding information

NIH (NIA)

## Total sum awarded (Euro)

204085.3211

## Start date of award

30/09/2016

## Total duration of award in years

1

## Keywords

Acquired Cognitive Impairment... Aging... Alzheimer's Disease... Alzheimer's Disease including Alzheimer's Disease Related Dementias (AD/ADRD)... Brain Disorders... Dementia... Neurodegenerative... Neurosciences... Prevention... Translational Research

## Research Abstract

Vivreon Biosciences – NIA SBIR # PA-16-091 Project Summary Vivreon Biosciences is pleased to apply for NIA SBIR Solicitation #PA-16-091. Vivreon Biosciences is an innovative life sciences company that is developing a series of novel small molecule, Ca<sup>2+</sup> channel inhibitors for the treatment of Alzheimer's disease (AD). Our lead compound series achieves

neuroprotection by an entirely new mechanism – inhibition of Ca<sup>2+</sup> release-activated Ca<sup>2+</sup> (CRAC) channels to block microgliosis. Vivreon seeks NIA funding to bridge the gap between discovery and development. We will optimize our promising lead compound series through medicinal chemistry. Upon successful completion of the program, our preclinical candidate will be the first to specifically target the CRAC pathway for neuroprotection in AD, thus comprising an entirely new tool in the battle against AD. Vivreon has discovered a lead compound series with oral bioavailability that penetrates into the central nervous system (CNS) very efficiently, shows no neurotoxicity in the Irwin test of CNS integrity, and demonstrates neuroprotection in a mouse model of microgliosis (experimental autoimmune encephalitis). The lead series inhibits microgliosis by blocking CRAC channel activity with nM potency; suppressing M1 NF- $\kappa$ B activity, while preserving M2 phagocytosis. We will improve on these favorable properties through medicinal chemistry lead optimization followed by biological screening. Our lead presents several routes for modification that could lead to improved drug-like properties, and these routes will be pursued to identify a preclinical candidate molecule suitable for future Investigational New Drug (IND)-enabling studies. The candidate will be identified using an animal model suitable for AD (5XFAD, Dr. Blurton- Jones, University of California, Irvine). The final aim for this proposal is synthesis and characterization of the first CRAC channel inhibitor for AD therapy.

**Further information available at:**

**Types:**

Investments < €500k

**Member States:**

United States of America

**Diseases:**

N/A

**Years:**

2016

**Database Categories:**

N/A

**Database Tags:**

N/A