

# Preventing Chickenpox/Shingles with NK-1R Antagonists

## Category

Pharmaceutic/Therapeutic

## Problem

Herpesvirus reactivation commonly leads to debilitating complications, postherpetic neuralgia, and myelitis

## Technology Overview

NK-1R antagonists as a novel antiviral drug to prevent the spread of herpesviridae

## IP Status

- ▶ Patent Pending
- ▶ Available for Exclusive or Non-Exclusive Licensing

## Value Proposition

- ▶ Development of treatments targeting NK-1R for symptoms or pathology associated with herpesviridae infection

## Market Attractions

- ▶ Global market for postherpetic neuralgia was estimated ~\$560 million for 2017
- ▶ Market expected to reach \$900 million by 2026
- ▶ US and France expected to account for over 50% of the market

## Contact

James Parrett

[James.parrett@ucdenver.edu](mailto:James.parrett@ucdenver.edu)

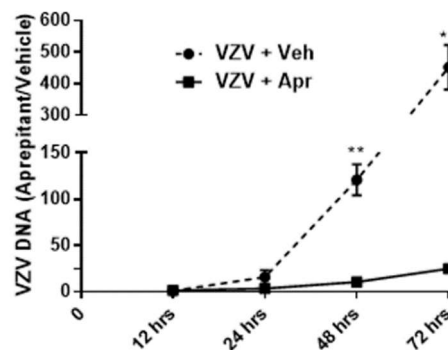
Ref# CU4707H

CU Innovations  
t. 303-724-0221

[innovations.ucdenver.edu](http://innovations.ucdenver.edu)

**Problem:** Varicella zoster virus (VZV), the herpesvirus underlying chickenpox and shingles, is an exclusively human neurotropic virus that, once acquired, can lay dormant throughout the neuraxis until reactivation with age or immunosuppression. Postherpetic neuralgia and myelitis are common complications of herpes zoster infection resulting from the reactivation of the virus. In immunocompromised individuals, myelopathy can be progressive and fatal with virus infection of the spinal cord, however the mechanisms are poorly understood and difficult to study.

**Technical Solution and Key Value Propositions:** A team of scientists at the University of Colorado have identified neurokinin-1 receptor (NK-1R) antagonists as a novel antiviral drug to prevent the spread of VZV and other herpesviridae. NK-1R signaling mediates the interaction between the immune system and nervous system, has been shown to be involved in RNA virus infection, and plays a role in measles virus spread in neurons. In a recent publication, Bubak and colleagues used primary human spinal astrocytes to study VZV infection in the context of VZV spinal cord disease. Their data indicates that cell-to-cell spread of VZV infection in primary human spinal astrocytes requires aberrant nuclear localization of NK-1Rs, which can be prevented by NK-1R antagonists (Bubak et al., 2018). Currently available NK-1R antagonists readily cross the blood-brain barrier and are used as an anti-emetic agent for patients undergoing chemotherapy. This novel treatment strategy may provide a much-needed alternative/adjuvant to treatment of recurrent or disseminated VZV infections, since the currently available drugs all share the same mechanism of action as nucleoside analog inhibitors of viral DNA replication.



*In-vitro* experiments show a marked decrease in VZV DNA concentration when VZV is in the presence of the NK-1R antagonist versus when with the spreading vehicle is uninhibited.

## Funding:

This research is funded by multiple NIH grants (NIH/NIA PPG, AG032958; NIH/NINDS R01, NS094758) and the Skaggs School of Pharmacy and Pharmaceutical Sciences Therapeutic Innovation Grant.

## Key Documents and Sources:

Provisional patent application available under CDA.

Bubak AN, Como CN, Blackmon AM, et al. (2018). Varicella Zoster Virus Induces Nuclear Translocation of the Neurokinin-1 Receptor, Promoting Lamellipodia Formation and Viral Spread in Spinal Astrocytes. *J Infect Dis.* 218(8):1324-1335. DOI 10.1093/infdis/jiy297.