Amyotropic Lateral Sclerosis – Parkinsonism Dementia Complex (ALS PDC) Model

CNS|CRO
Preclinical Neurological Disease Services

Model Overview

CNS|CRO's proprietary animal model of ALS PDC utilizes a neurotoxin that causes a slowly developing, progressive series of well-defined sequential deficits in mice. First documented in humans following dietary exposure to the cycad seed in the 1940's on the island of Guam, the neurotoxin was identified and patented by Neurodyn Life Sciences Inc, CNS|CRO's parent company.

Differentiation & Advantages

Slowly developing and progressive with well defined sequential deficits:

→initially, motor neurons (ALS) are affected
→followed by basal ganglia (PD), and
→finally cortex/hippocampus (Cognitive Decline)

Each stage is characterized by the behavioural and neuropathological hallmarks of disease observed in humans

Unlike end-stage models, candidate therapeutics can be investigated for all steps of disease development (e.g., candidate extension and repositioning, proof of concept, target identification, mechanism of action validation)

Validation

Construct validity:

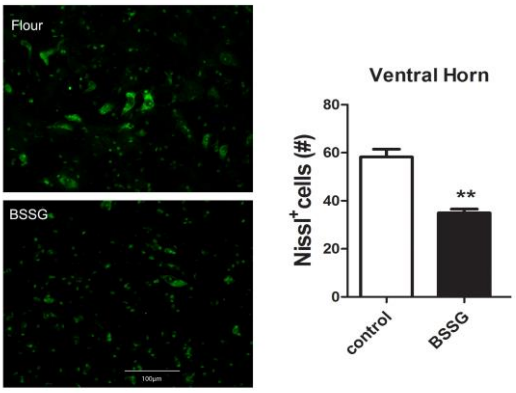
- this animal model is created using a neurotoxin to which ALS PDC patients had dietary exposure

Face validity:

- Neuroinflammation
- Oxidative stress
- Motor neuron loss
- Locomotor deficits

Predictive validity:

Minocycline failed in this model



Nissl Cell Counts in Mouse Spinal Cord. BSSG treatment induced a significant reduction in Nissl+ cells. **p<0.01, error bars represent SEM.

CNS|CRO
550 University Avenue
Charlottetown | Prince Edward Island
C1A 4P3

For more information: info@cnscro.com

CNSCRO.COM