Neuren's Partnering Update

5 December 2006: Mr Larry Glass, Executive Vice President of Neuren Pharmaceuticals Ltd (ASX:NEU) will this week be meeting with a variety of biotechnology companies in Europe to increase the awareness of the Company and to explore potential partnering opportunities for its drug candidates.

The attached presentation prepared for these meetings provides an overview of Neuren's drug pipeline.

A copy of this presentation is also available at www.neurenpharma.com under latest news.

About Neuren Pharmaceuticals:

Neuren Pharmaceuticals (ASX: NEU) is a biopharmaceutical company developing novel therapeutics in the fields of brain injury and diseases and metabolic disorders. The Neuren portfolio consists of six product families, targeting markets with large unmet needs and limited competition. Neuren has three lead candidates, Glypromate® and NNZ-2566, presently in clinical trials to treat a range of acute neurological conditions, and NNZ-2591 in preclinical development for Parkinson's and other chronic conditions. Neuren has commercial and development partnerships, including with the US Army Walter Reed Army Institute of Research, Metabolic Pharmaceuticals, UCLA Medical Center and the National Trauma Research Institute in Melbourne.

For more information, please visit Neuren's website at www.neurenpharma.com

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Pharmaceuticals

Disclaimer

This presentation includes forward-looking statements that are subject to risks and uncertainties. Such statements involve known and unknown risks and important factors that may cause the actual results, performance or achievements of Neuren to be materially different from the statements in this presentation.

Actual results could differ materially depending on factors such as the availability of resources, results of clinical studies, the timing and effects of regulatory actions, the strength of competition and the effectiveness of patent protection.

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Neuren Pharmaceuticals Ltd

- Spun off from the University of Auckland
- Listed on the ASX Feb 2005
- Excellent pipeline
  - 2 molecules in the clinic
  - 2 molecules in preclinical development
  - 3 families of compounds in discovery
- Discovery programs: neurology, growth and metabolism
- Clinical development focus: neurology
- Company operations
  - Auckland: HQ, R&D, Clinical Development
  - Maryland: regulatory, CMC, pharm/tox, business development
- Cost-effective discovery
- Experienced drug development team

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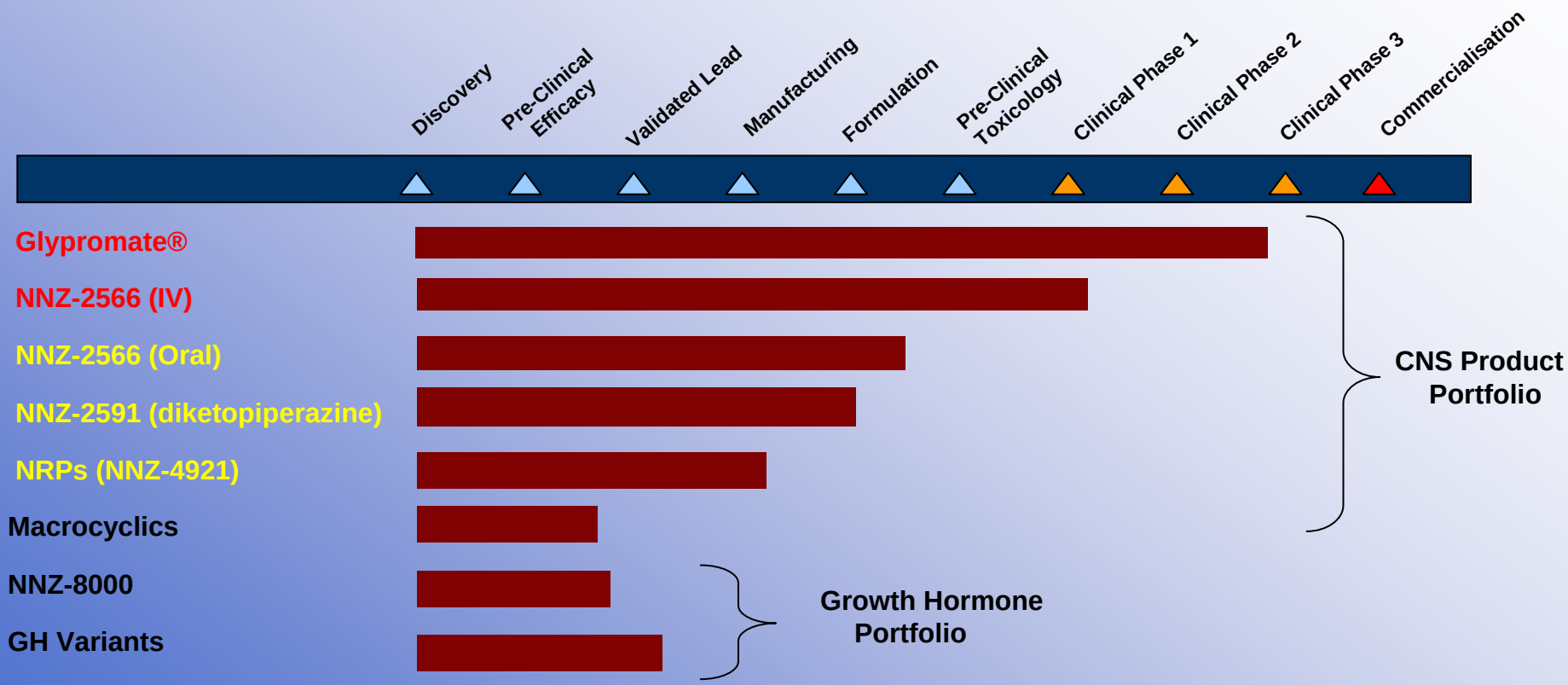
Product Pipeline



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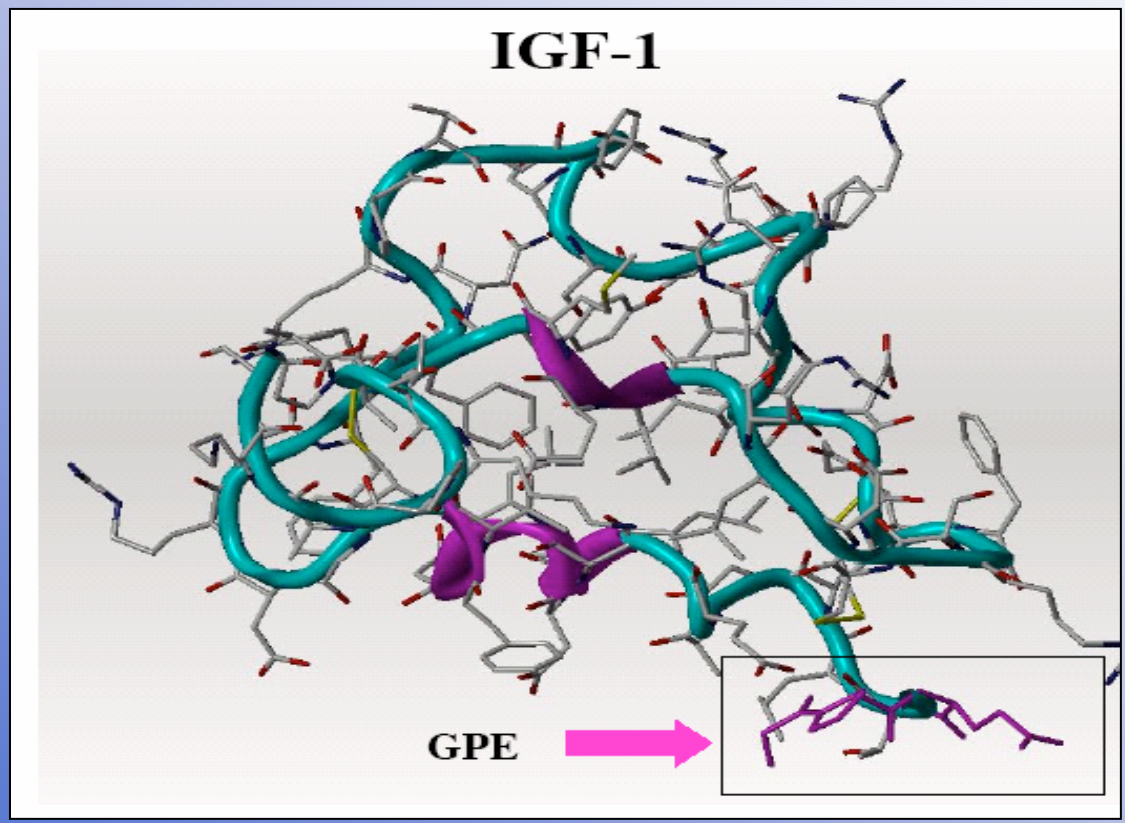
Glypromate®



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Glypromate®

- Drug-like molecule
  - Small molecule (MW=301.3 g/mol)
  - Highly neuroprotective at nM concentrations
  - Readily crosses the blood brain barrier
  - Rapidly cleared from circulation
  - Simple and inexpensive to synthesize
  - Simple and inexpensive formulation
- Preclinical efficacy
  - Global hypoxia-ischemia, focal stroke and MCAO models
  - Wide therapeutic window (100% effect at 7-11 hours)
  - Long term neuroprotection with single, 4-hour infusion
  - Improved histopathological, functional, behavioral outcomes
  - Multiple modes of action protect neurons and astrocytes

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Multiple Modes of Action

- Inhibition of caspase-3 dependent apoptosis
- Inhibition of microglial activation
- Prevention of delayed or secondary necrosis
- Protection of astrocytes and oligodendrocytes
- Protection of neurons and neuronal function

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Preclinical Safety and Toxicology

- Safety studies
  - No selectivity issues or unwanted secondary pharmacology predicted by wide binding
  - No inhibition of CYP-450 enzymes: low risk of drug-drug interactions
  - No *in vivo* effect on cardiovascular system observed with high i.v. doses
  - Clean result in GLP hERG testing
- Toxicology studies
  - No adverse effects at MFD (700 mg/kg in dogs)
  - No genotoxicity, mutagenicity, clastogenicity, chromosomal aberrations

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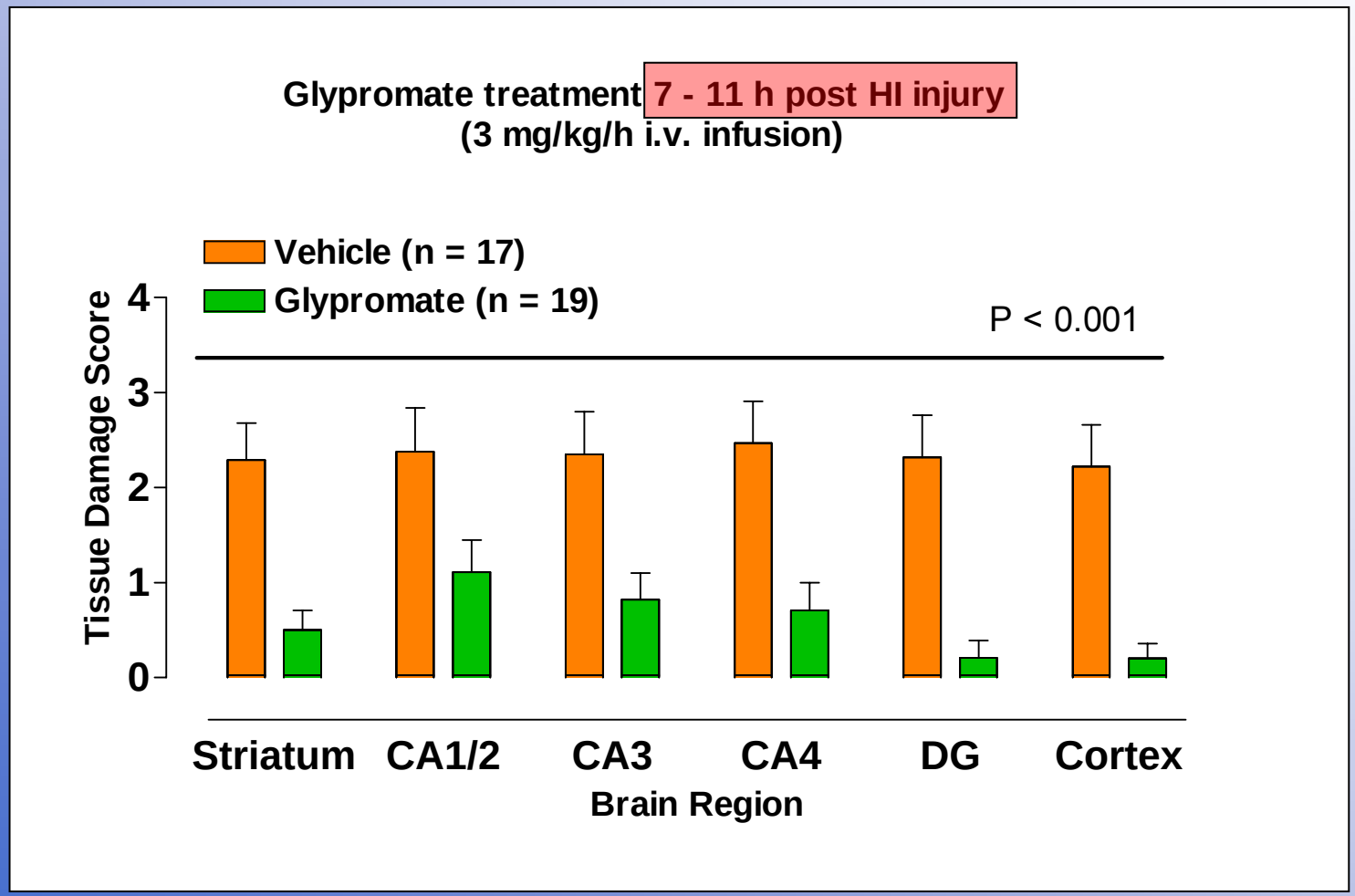
Glypromate is Neuroprotective After Acute Brain Injury



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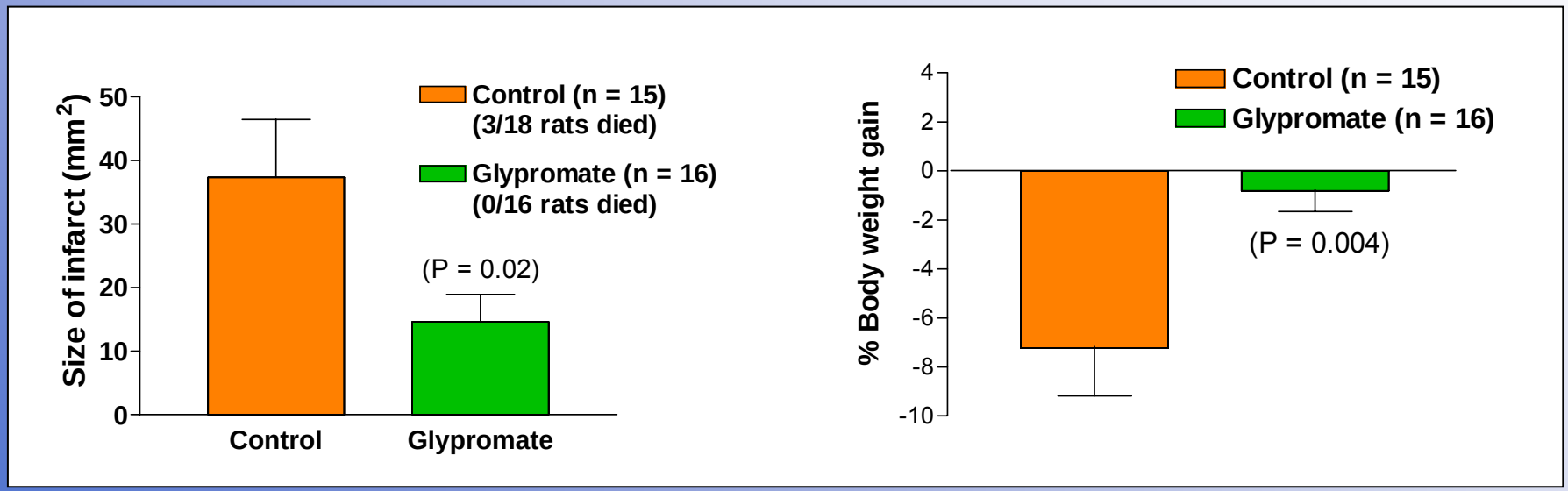
Effect of Glypromate in MCAO Model of Acute Brain Injury



3 mg/kg/h i.v. infusion

5 – 9 h treatment window

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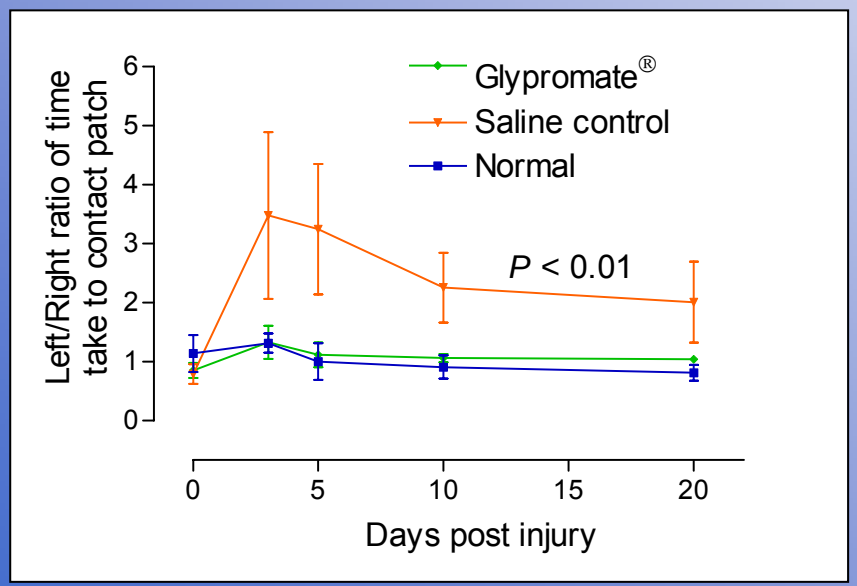
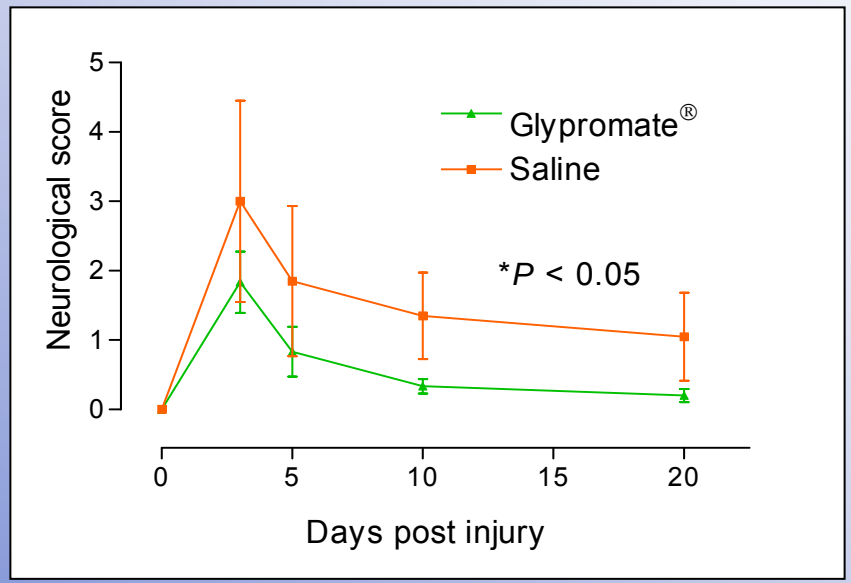
Long Term Improvements in Functional Recovery

- **Improved behavioral outcome**
- **Improved neurological outcome**
- **Improved histological outcome**





Glypromate 3 mg/kg/h i.v. 1-5 h post-injury
n = 8 per group

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Glypromate® Clinical Program

- Phase I
  - Single dose, double-blind, randomized, placebo-controlled, dose escalation
  - 30 healthy subjects
  - No SAEs; no drug-related AEs
- Phase IIa (CABG +/- valves)
  - Randomized, ouble-blind, placebo-controlled, multiple dose
  - 32 patients >60 years old (22 treatment, 10 controls)
  - No drug-related AEs or SAEs; linear PK
- Phase III (CABG +/- valves and valves alone)
  - Pre-IND meeting April 2005; Phase IIb designation changed to pivotal
  - 520 patients (CAGB, valve repair/replacement, both)
  - Drug administered as 4 hr infusion at end of surgery
  - Primary endpoint: change in composite neurocognitive function score
- Phase IIa (out of hospital cardiac arrest)
  - Army investigator-initiated study
  - Endpoints: mortality, neurological function, neurocognitive function

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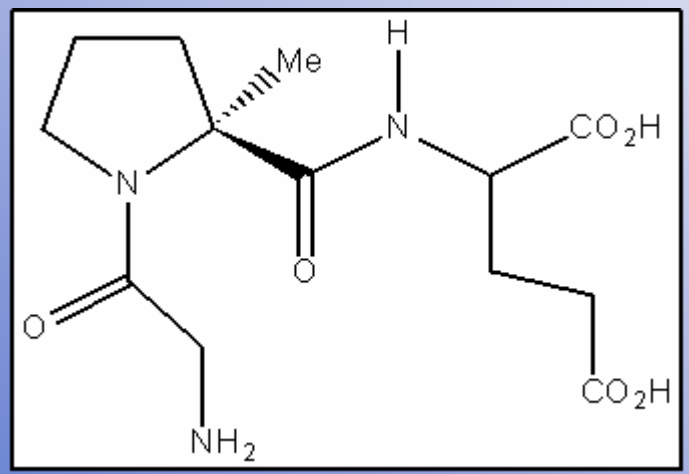
NNZ-2566



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NNZ-2566 (IV) Development

- GMP manufacturing process validated
- GLP acute toxicology program
  - Acute rat and dog and 14-day rat completed
  - 28-day dog scheduled for Q4 2006 (24-hr infusion)
- Phase Ia initiated April 2006 (bolus, dose-escalation)
- Phase 1b to be initiated Q4 2006 (continuous infusion)
- CRADA with US Army/WRAIR for TBI (including neurological, behavioral, functional and EEG endpoints)
- Agreement with UCLA and National Trauma Research Institute (Melbourne) for closed-head injury model
- No rights conveyed; no royalties due
- Phase II — two studies being planned
  - Open-label study in severe TBI patients
  - Randomized, double-blind study in mild-moderate TBI patients

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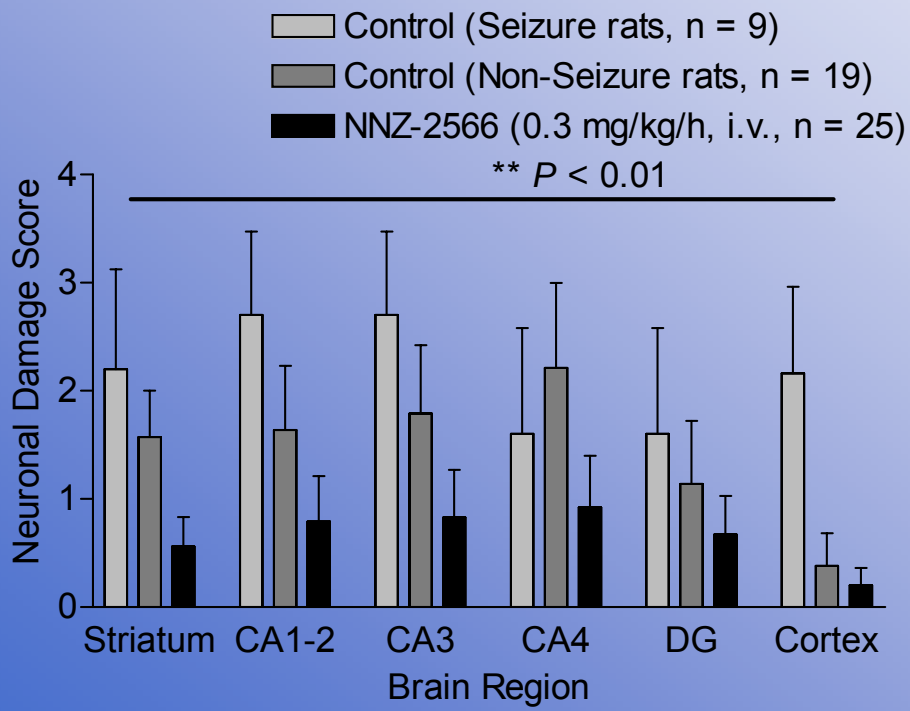
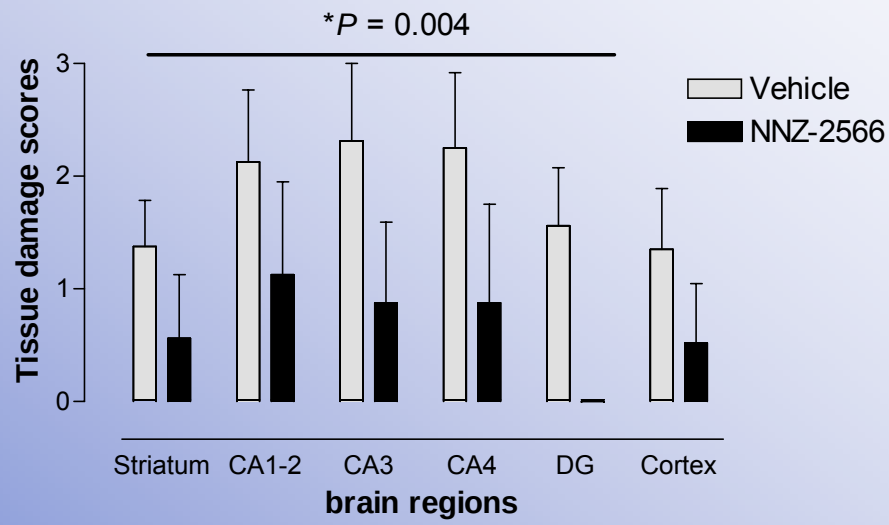
NNZ-2566 is highly neuroprotective



Global HI model; IV administration



MCAO model; subcutaneous administration

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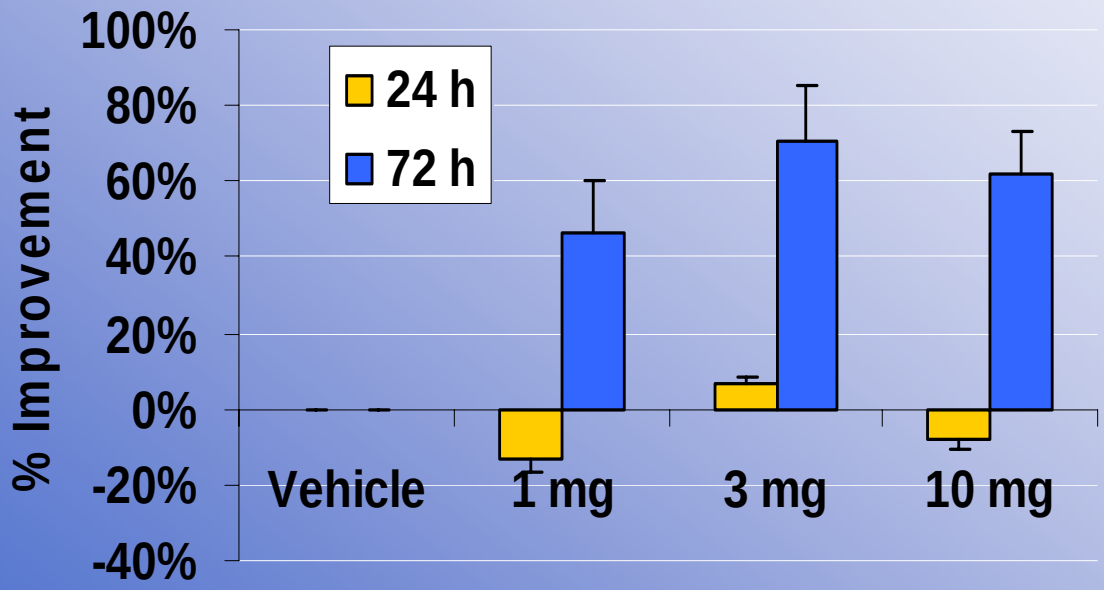
NNZ-2566 in a Penetrating Brain Injury Model

Balance Beam Test (Foot Fault Deficits)



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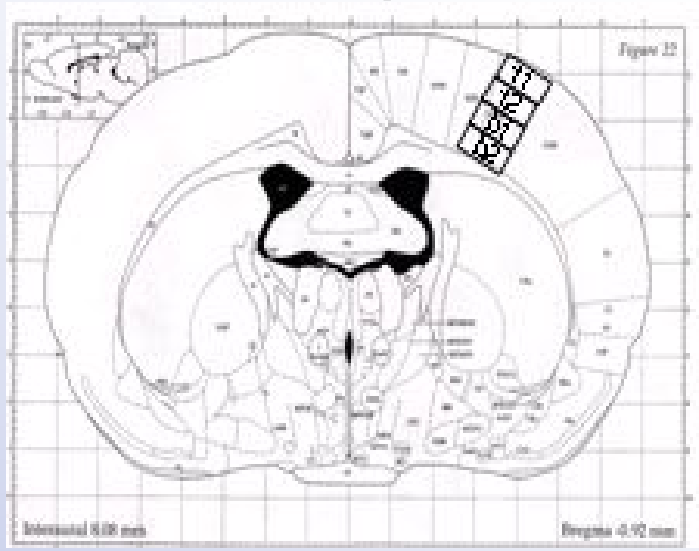
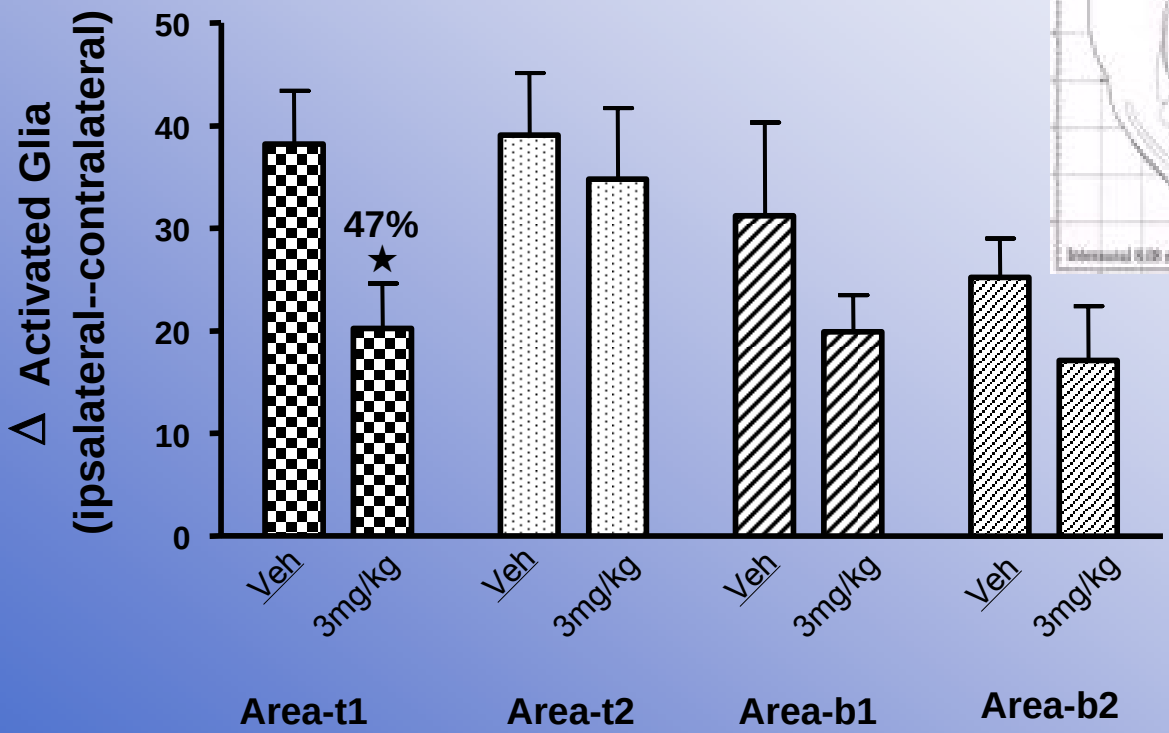
WRAIR TBI Model (12-hour infusion)

Microglial Cell Count (Ipsilateral minus Contralateral)





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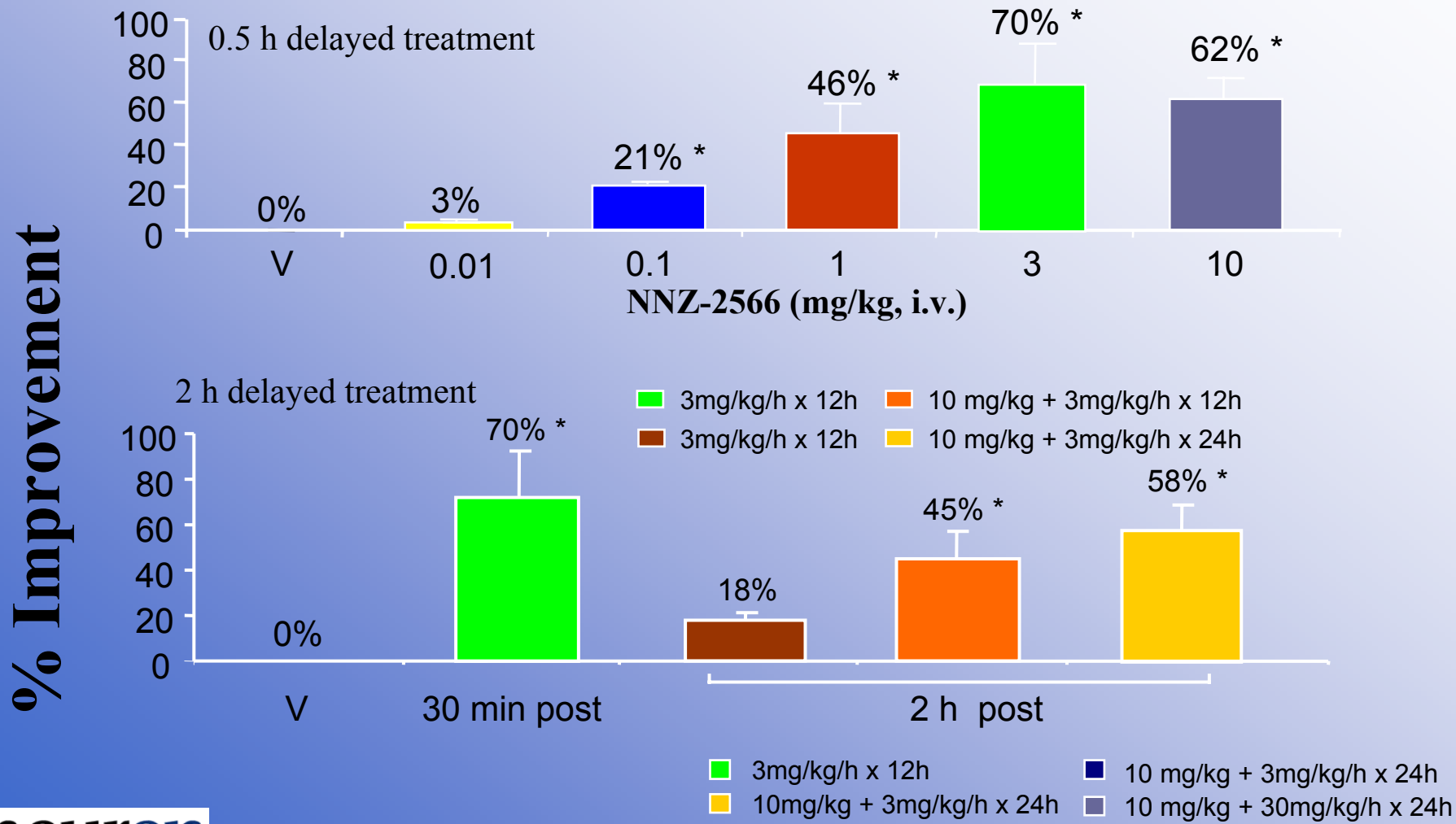
Balance Beam Performance



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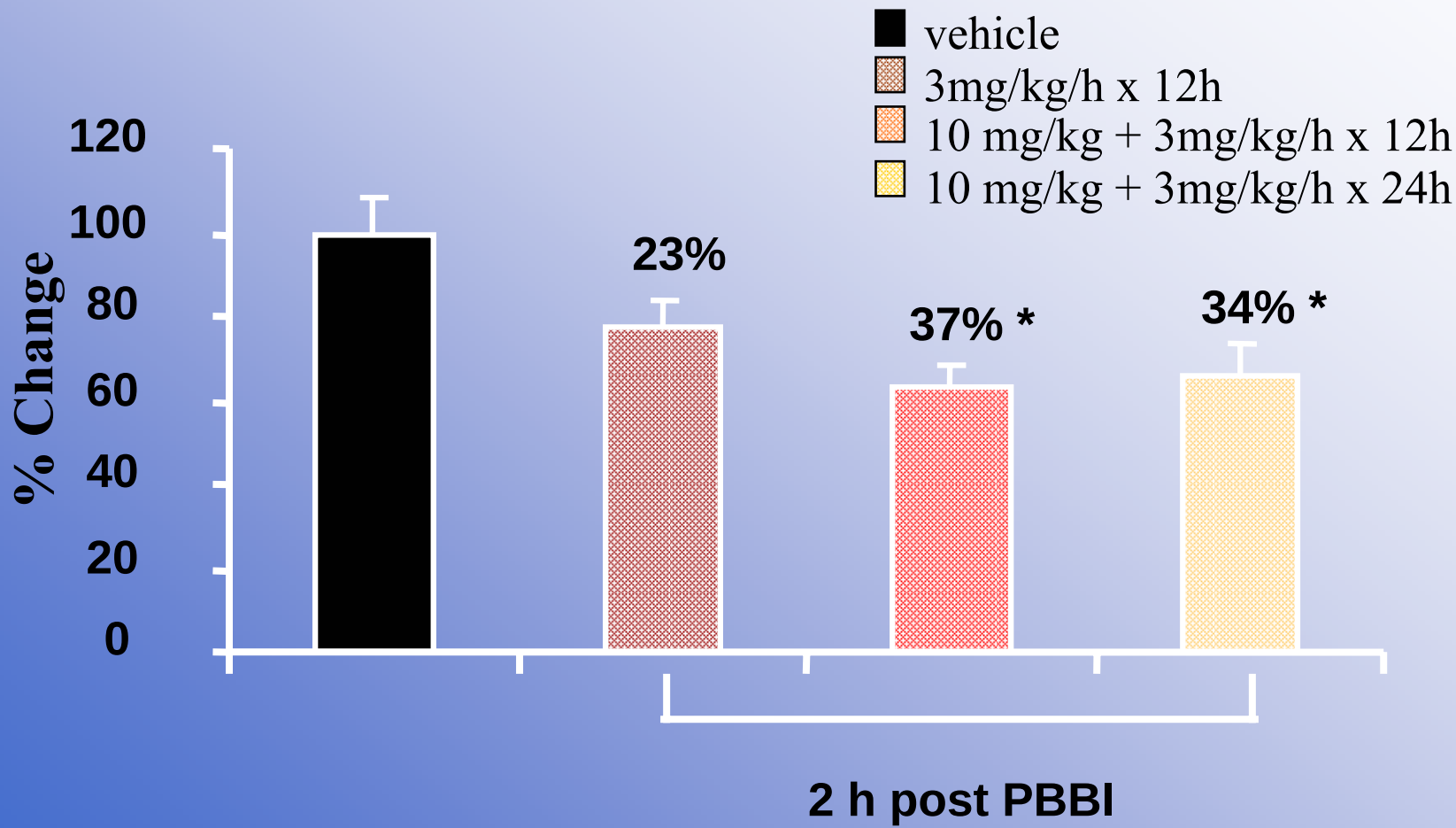
Injury Volume (H & E)



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Attenuation of Non-Convulsive Seizures

	Incidence	Total Time (sec)	Mean Time (sec)	Latency (min)
Vehicle	92%	1277	80.2	75.4
NNZ-2566	60%	555	48.7	208.7
Percent Change	-35%	-56%	-39%	+133%

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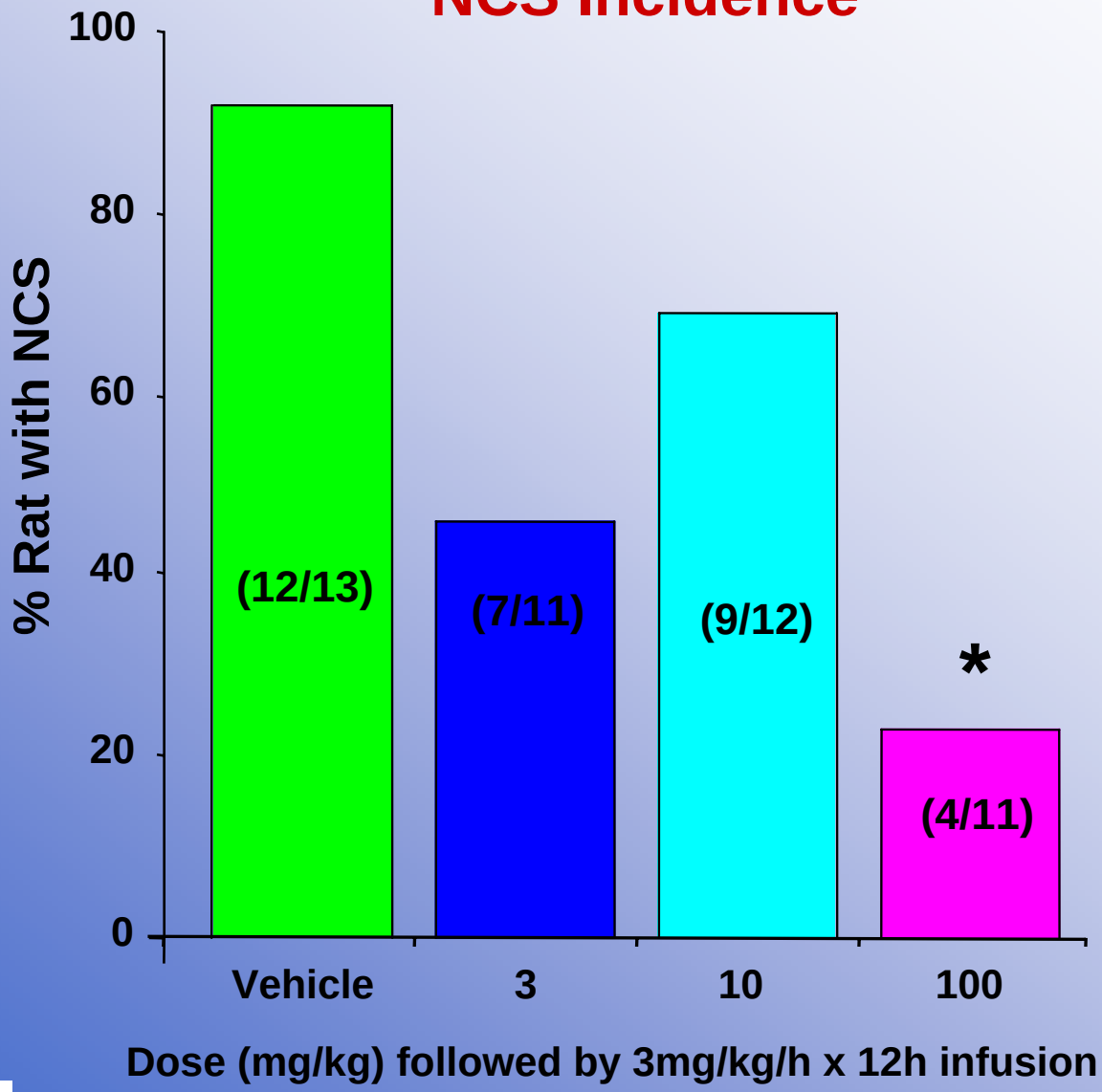
WRAIR MCAO Model
(bolus + 12-hour infusion)

NCS Incidence



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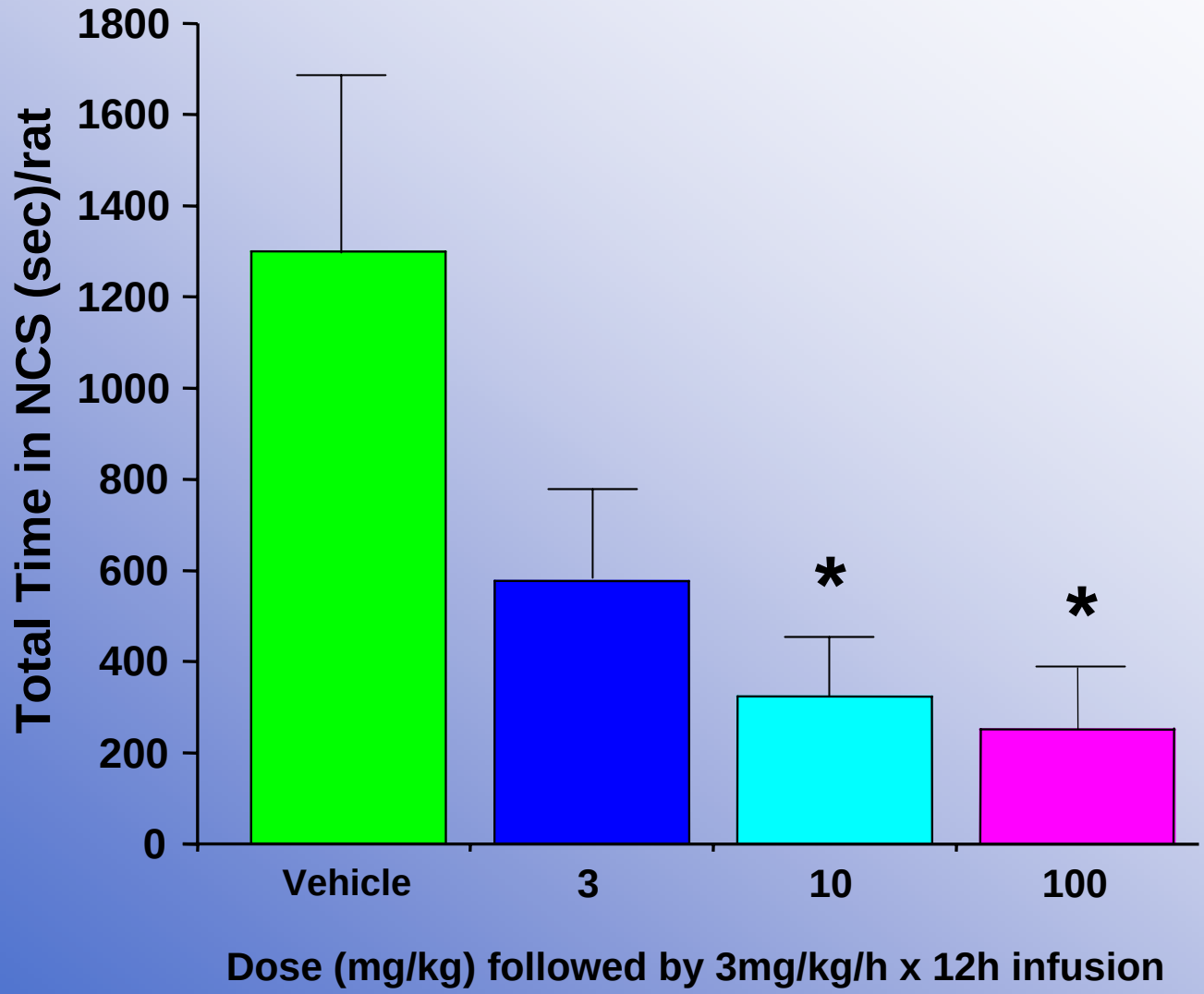
Total NCS activity



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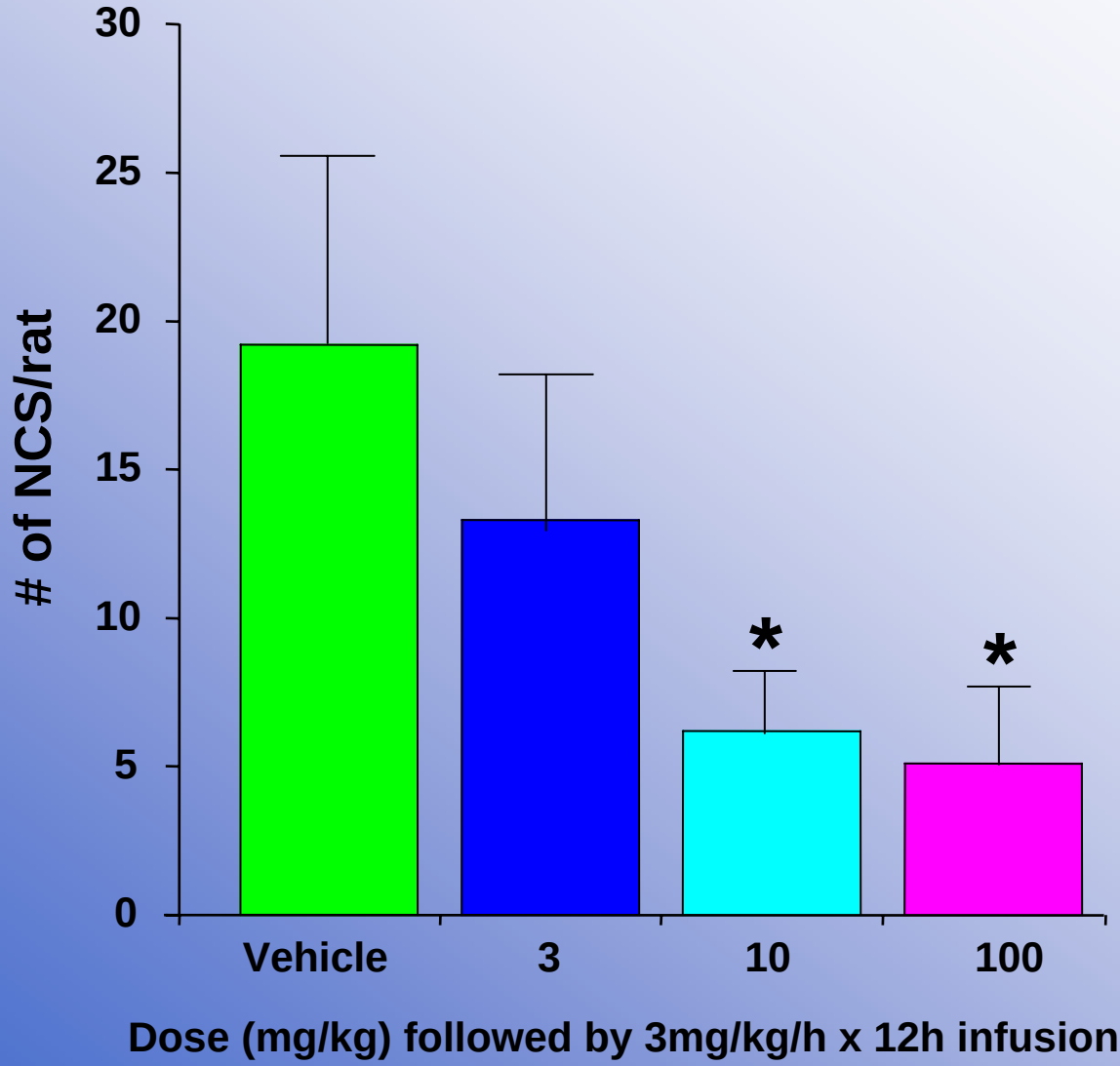
NCS frequency



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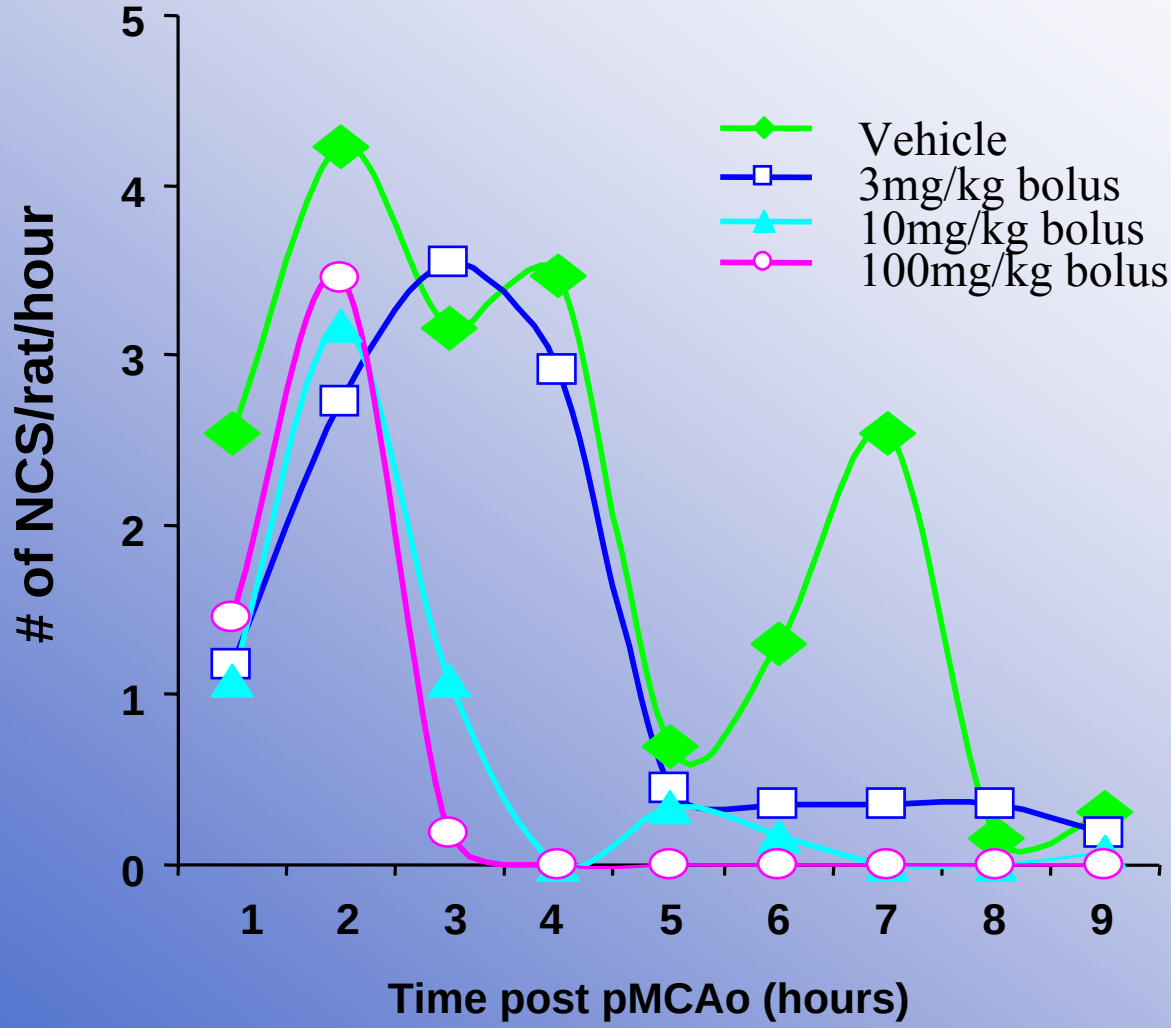
NCS time course



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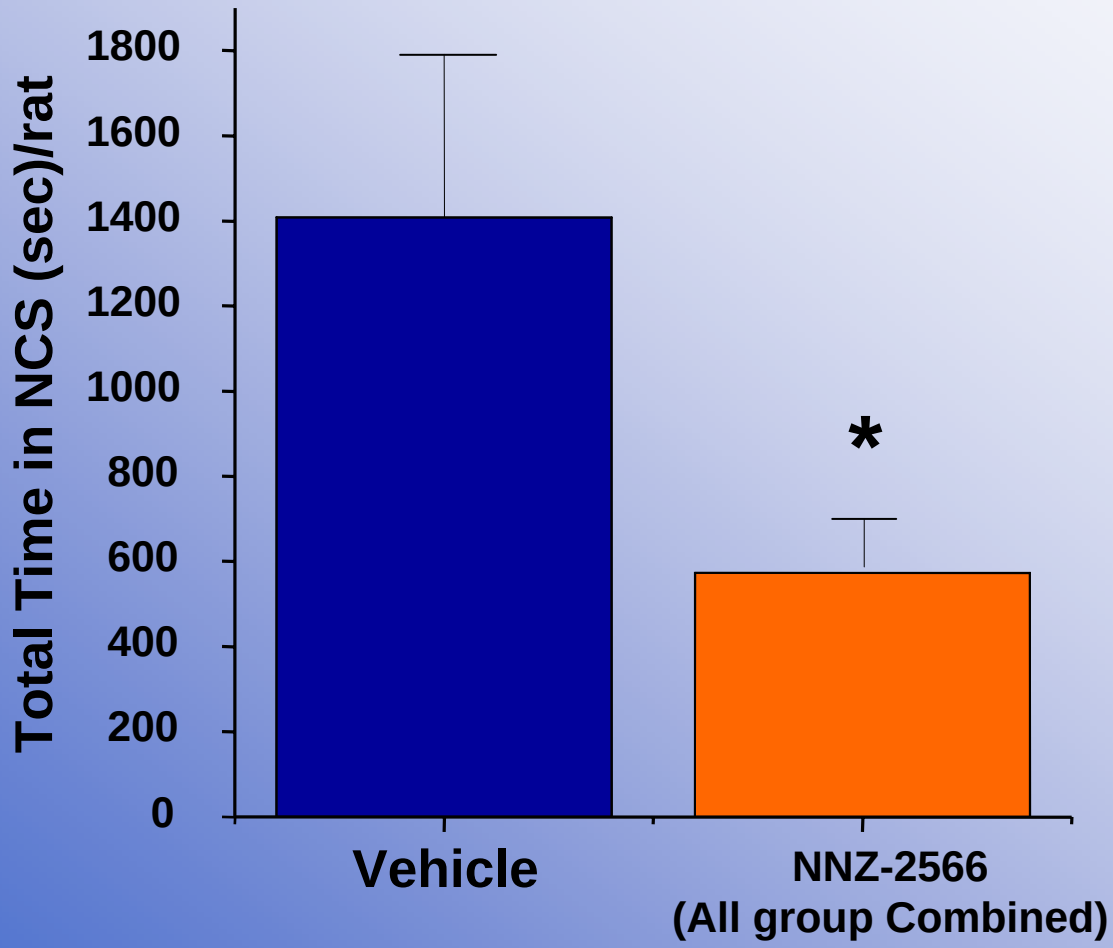
Total seizure time among all rats with seizures



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Inhibition of Gene Up-regulation

Gene	4 hours post-injury	24 hours post-injury
Apoptotic/Pro-apoptotic genes		
Bax	●	
Caspase-8	●	
Fadd	●	
Inflammatory cytokines		
IL-1β		●
IL-6		●
TNFα	●	

● = $p < .05$

Wei et al. NNZ-2566 treatment inhibits inflammatory and apoptotic gene up-regulation induced by experimental penetrating ballistic brain injury. ATACCC Conference, August 2006.

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Study 1: Phase IIa (severe)

- Open-label, observational study in TBI patients with GCS 4-8 to evaluate…
  - Safety
  - Penetration of drug to injured brain
  - Preliminary evidence of efficacy in modifying acute disease/injury processes
- Within-subjects and cohort comparison analyses
- Drug administered within 24 hours of injury as IV bolus followed by 72 hrs of infusion during days 1-5
- Planned enrollment: 50 closed head; 15 penetrating
- Stratification by closed (focal vs. diffuse) and penetrating

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Study 1 (severe): Within-subjects comparisons

- Incidence of non-convulsive seizures and epileptiform activity
- Mean global EEG percent alpha variability (PAV) score
- Biomarkers (measured in CSF at 24, 36, 72, 96 hrs)
  - Alpha II-spectrin breakdown product SBDP145 (calpain-mediated acute cell death / necrosis)
  - Alpha II-spectrin breakdown product SBDP120 (caspase-mediated delayed cell death / apoptosis)
  - Neuroinflammation marker IL-6
- Incidence of hypotension
- Percent time of elevated intracranial pressure
- Study drug concentration in brain microdialysis samples
- Mortality
- Safety (AEs and SAEs)

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Study 1 (severe): Cohort Comparisons

- Historical cohorts
  - Maas et al. Lancet Neurology 2006
  - Vespa et al. J Neurosurgery 2002
  - Vespa et al. J Neurosurgery 1999
  - Hebb et al. J Neurotrauma (submitted) 2006
- Mortality at 30 days
- GOSE at 6 months
- Percent time of elevated intracranial pressure
- Neuropsychological (CDR) test battery at 6 months
- Mean global EEG PAV during post injury days 1-5
- Percent time of microdialysis lactate:pyruvate ratio (LPR) > 25
- Incidence of electrographic epileptiform activity/NCS during study drug infusion compared with non-infusion period during post injury days 1-5
- Therapy Intensity Level values

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Study 2: Phase IIa (mild-moderate)

- Randomized, double-blind, placebo controlled, dose-escalation study in patients with GCS 9-15 to evaluate safety and efficacy
- Intended enrollment = 200 (3:2 active : placebo randomization)
- Interim analysis of efficacy and safety with ~50 completed patients
- Drug administered within 12 hours of injury as IV bolus followed by 8-24 hrs continuous infusion
- Primary outcome measures
  - Neurocognitive outcome at 6 months (CDR)
  - Hamilton depression scale at 6 months
  - Global quality of life measure (tbd) at 6 months
  - Safety (hypotension, mortality, AEs, SAEs prior to discharge)
- Secondary outcome measures
  - cEEG (PAV, epileptiform activity) to discharge
  - MRS N-acetylaspartate (NAA) at 6 months (on a stratified subset of patients)
  - MRI volume analysis at 6 months (on a stratified subset of patients)

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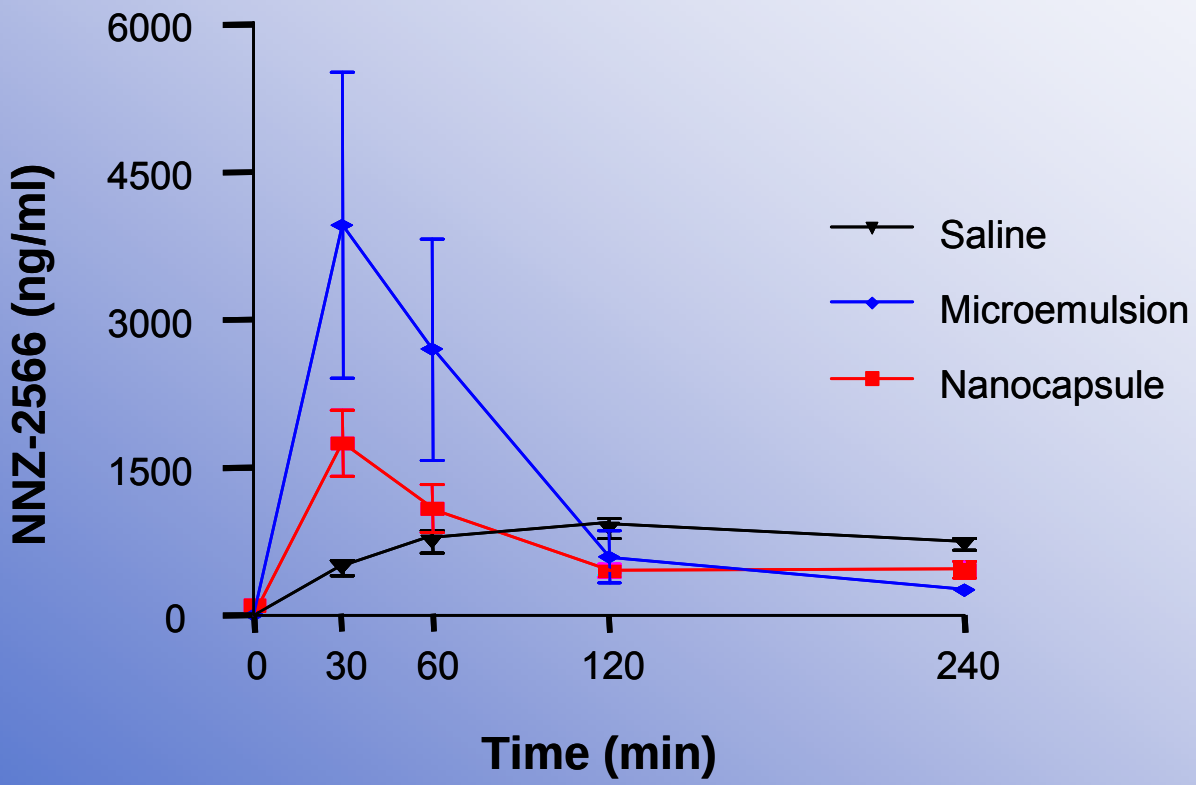
NNZ-2566 Oral: Bioavailability



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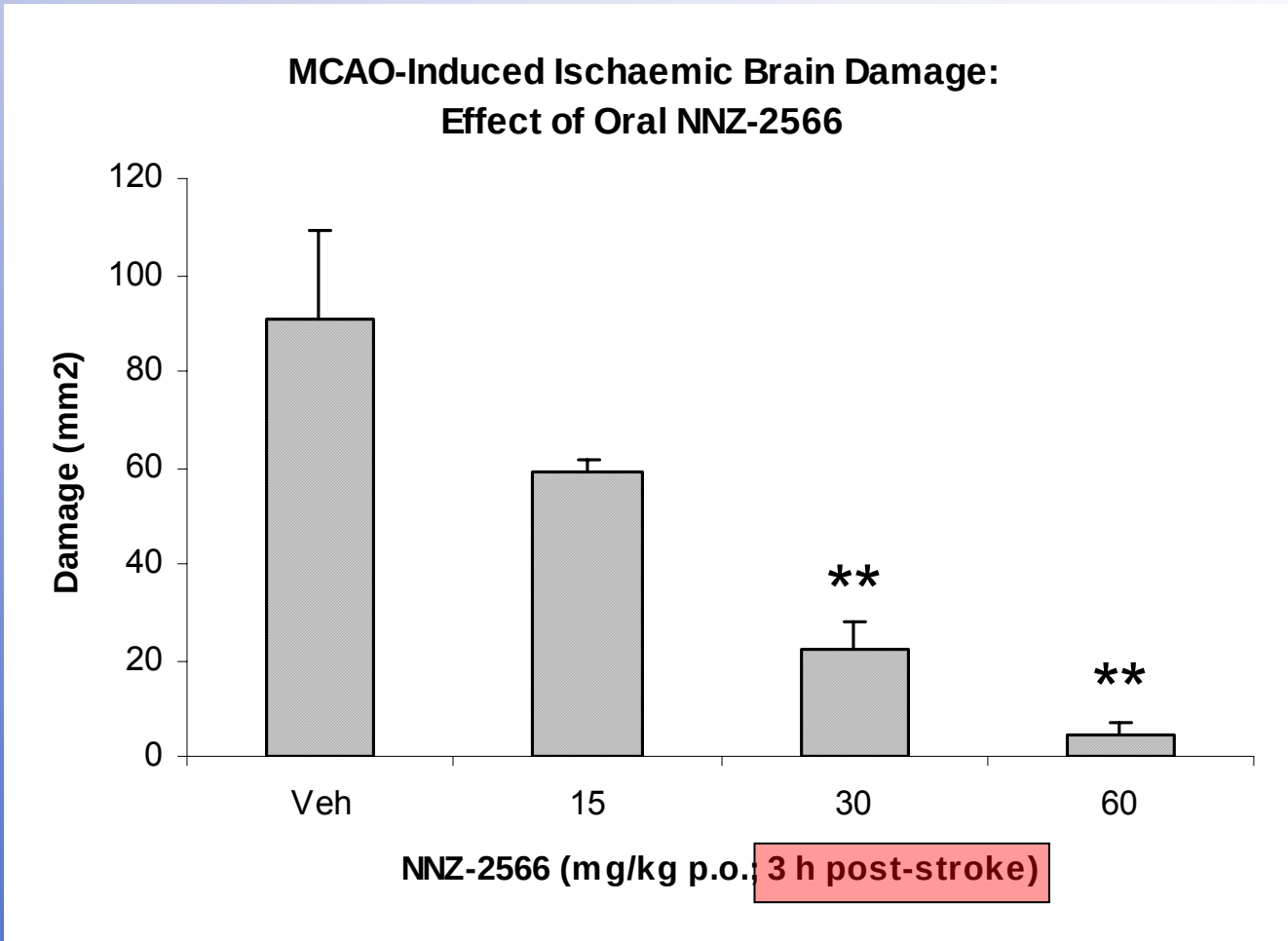
NNZ-2566 Oral (single dose)



** P < 0.01, ANOVA with Dunnett's post-hoc test. Group sizes: Veh (9), 15 mg/kg (8), 30 mg/kg (8), 60 mg/kg (7)

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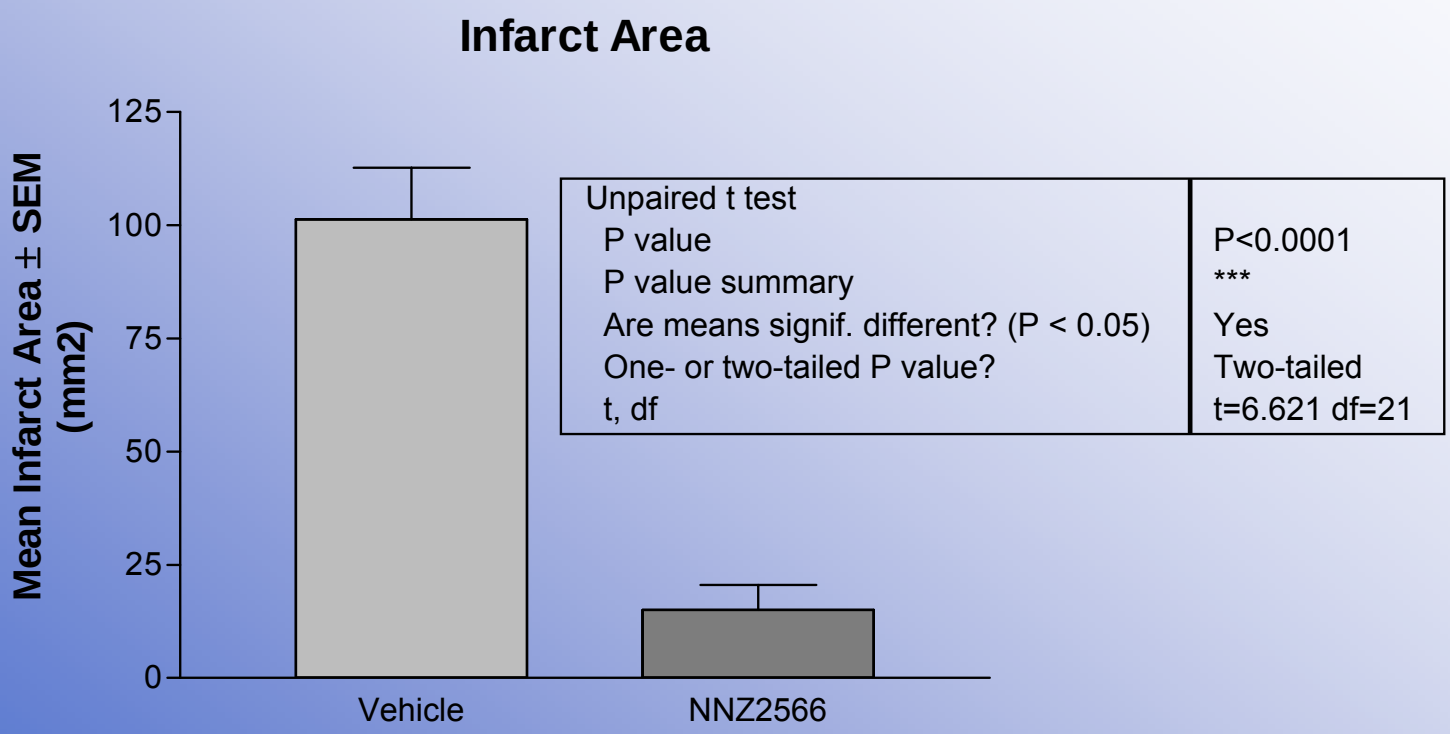
NNZ-2566 Oral (2 doses)



Effect of microemulsion NNZ2566 given orally at 2 and 4 hours post
Et-1 induced MCAO.Total dose is 80mg/kg. Vehicle (n = 12),NNZ2566 (n=11)

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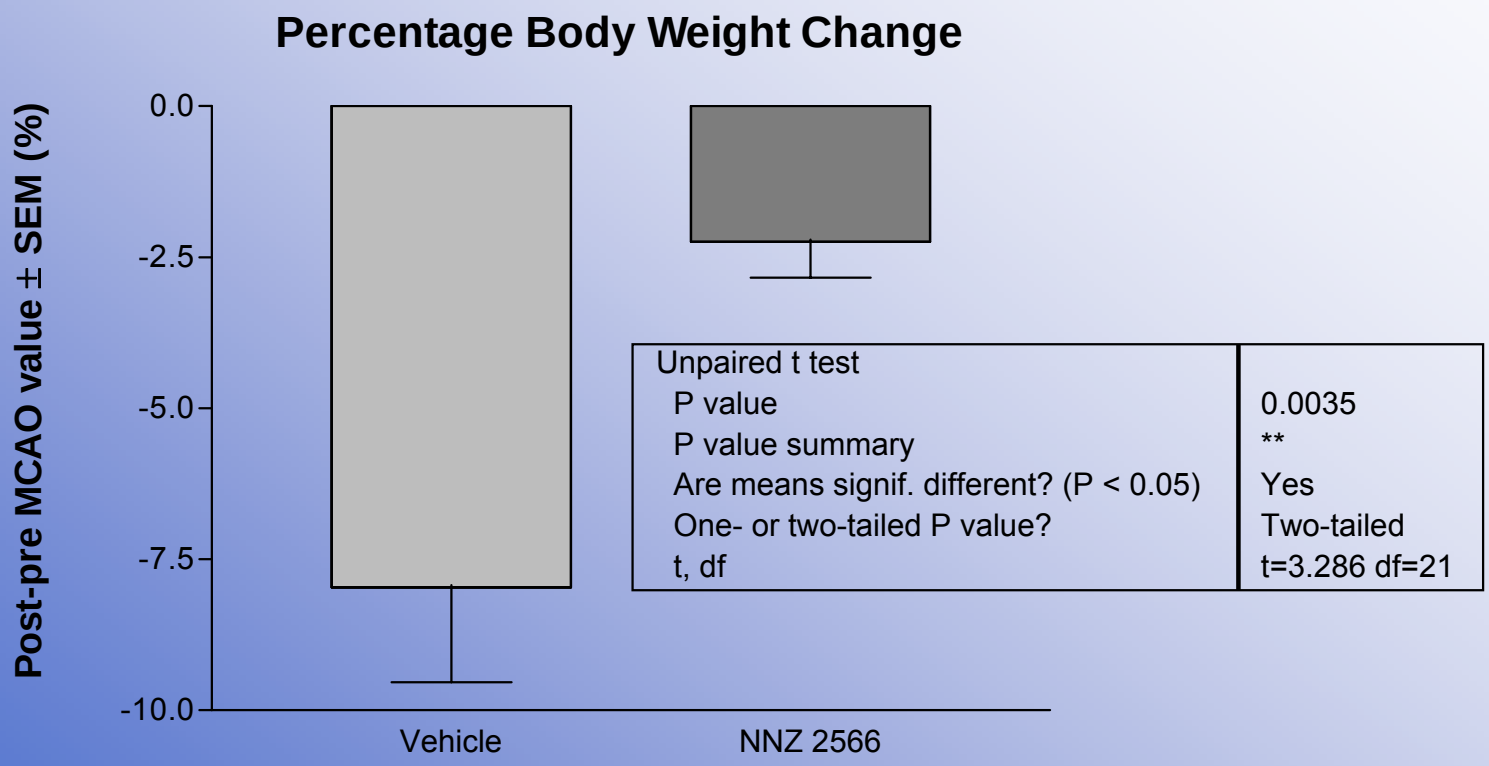
NNZ-2566 Oral (2 doses)



Effect of microemulsion NNZ2566 given orally at 2 and 4 hours post Et-1 induced MCAO.Total dose is 80mg/kg. Vehicle (n = 12); NNZ 2566 (n = 11)

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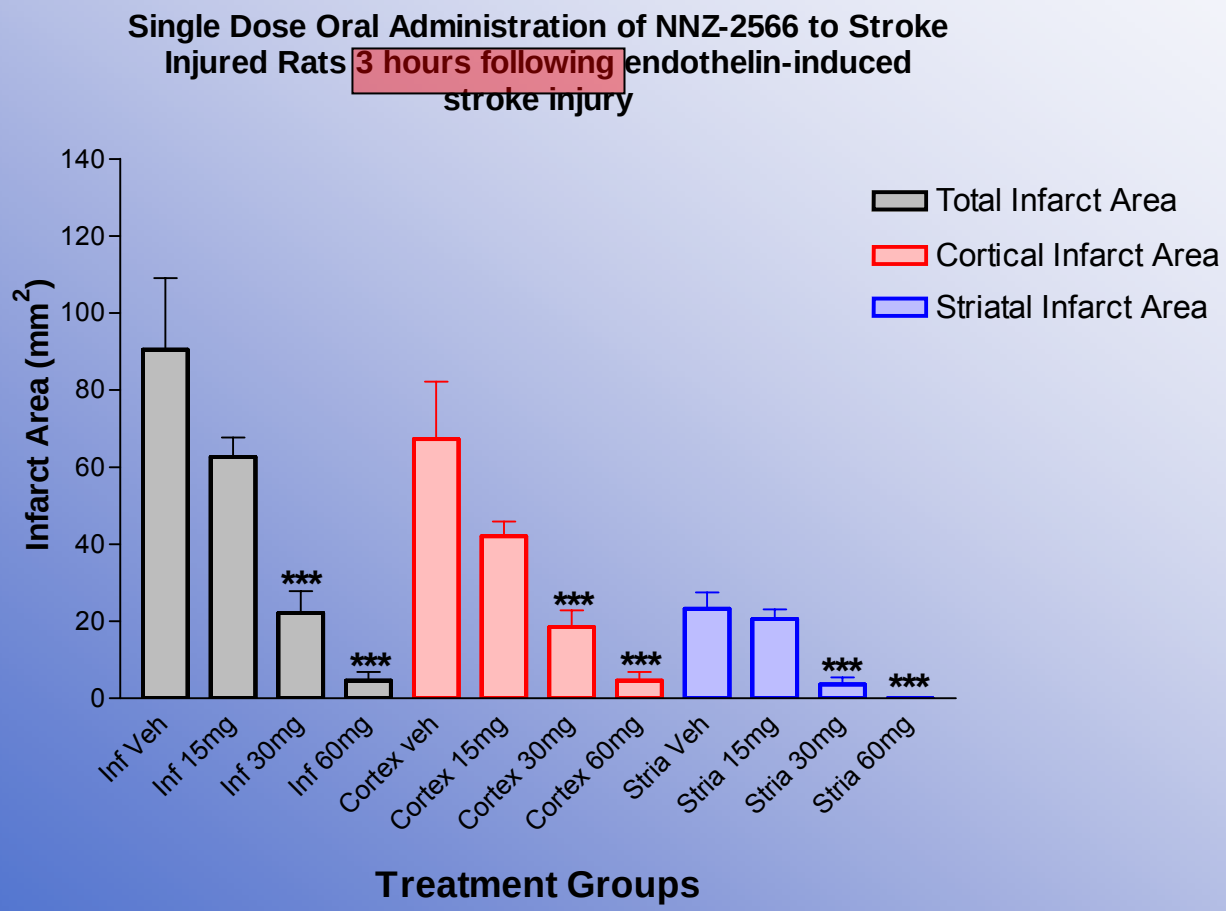
NNZ-2566 Protects Striatum and Cortex



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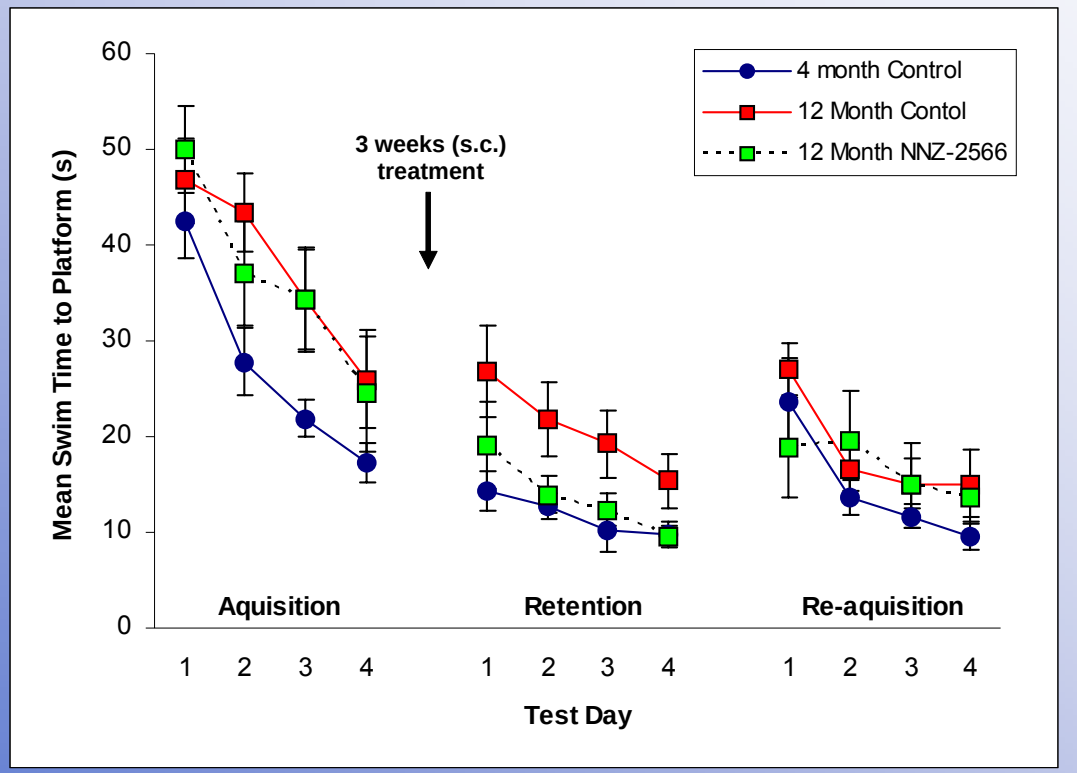
Chronic NNZ-2566 treatment improves memory retention



- Morris water-maze study; NNZ-2566 administered s.c. (4.8 mg/day, 3 weeks)
- Aged rats display slower acquisition and poorer retention of spatial memory
- NNZ-2566 significantly improves memory retention after chronic treatment

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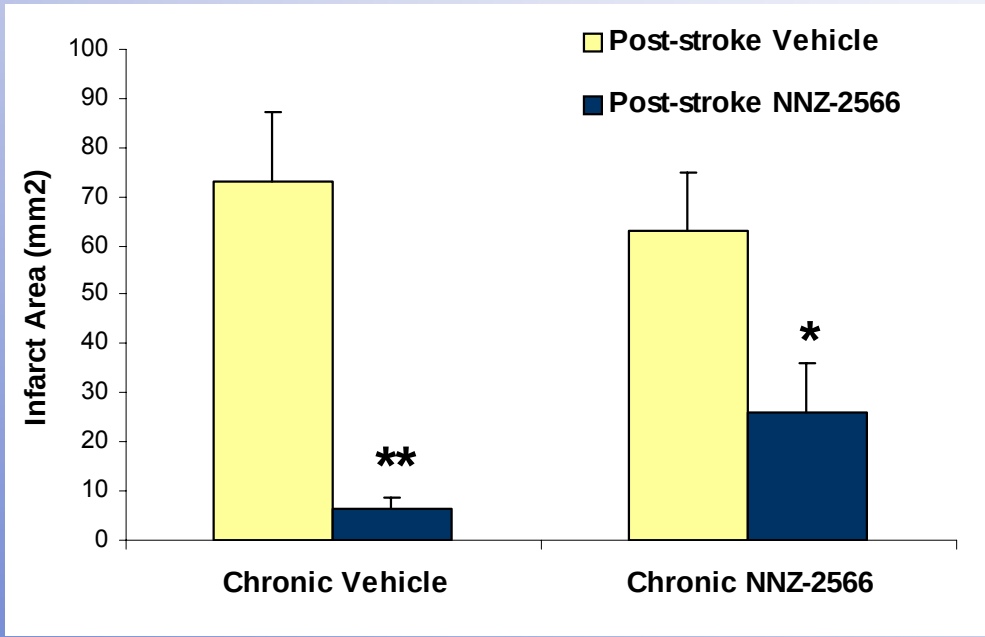
Neuroprotection retained in rats after chronic oral treatment



- Oral NNZ-2566 (30 mg/kg) retains efficacy in rat neurodegeneration model
- No statistical difference in effect of post-stroke NNZ-2566 whether previously treated with drug or water (13 days)
- Study supports viability of repeat treatment of NNZ-2566 in chronic disorders

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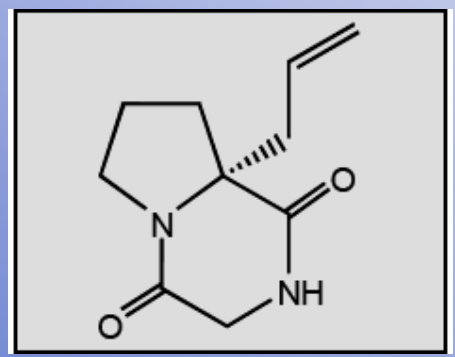
NNZ-2591
(Lead Diketopiperazine)



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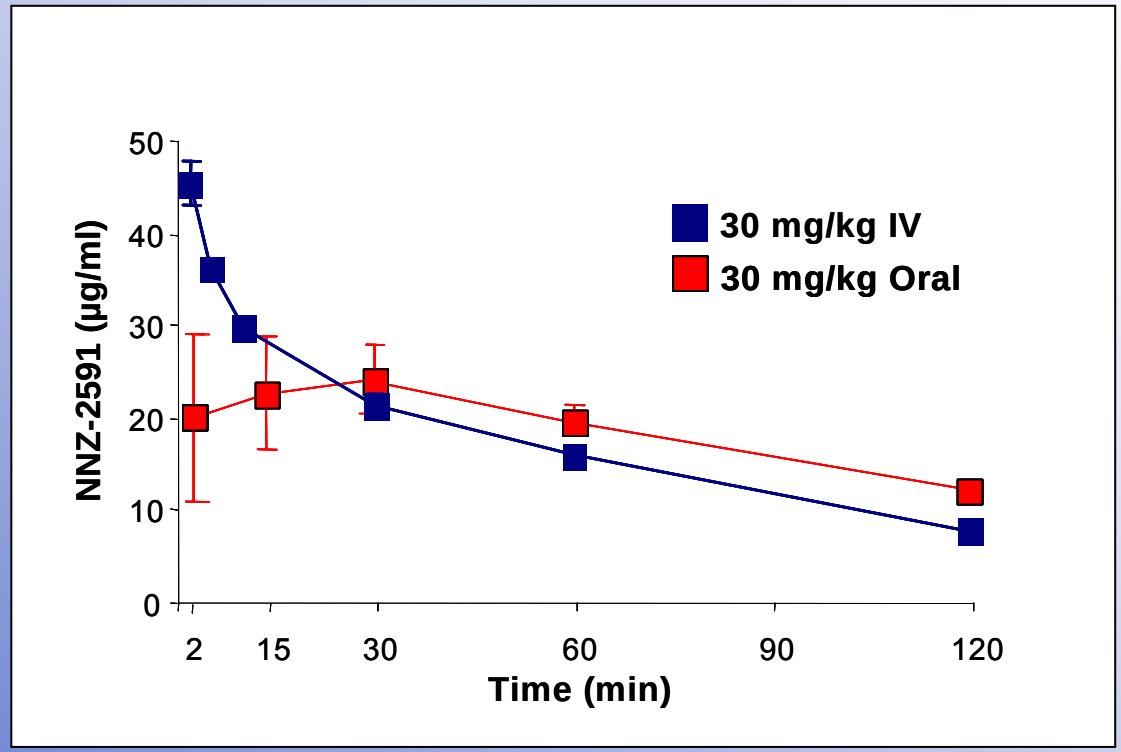
Oral bioavailability of NNZ-2591



- Oral bioavailability of aqueous NNZ-2591: **100%**
- Plasma half-life in rats: 50 minutes
- Excellent profile for chronic oral drug delivery

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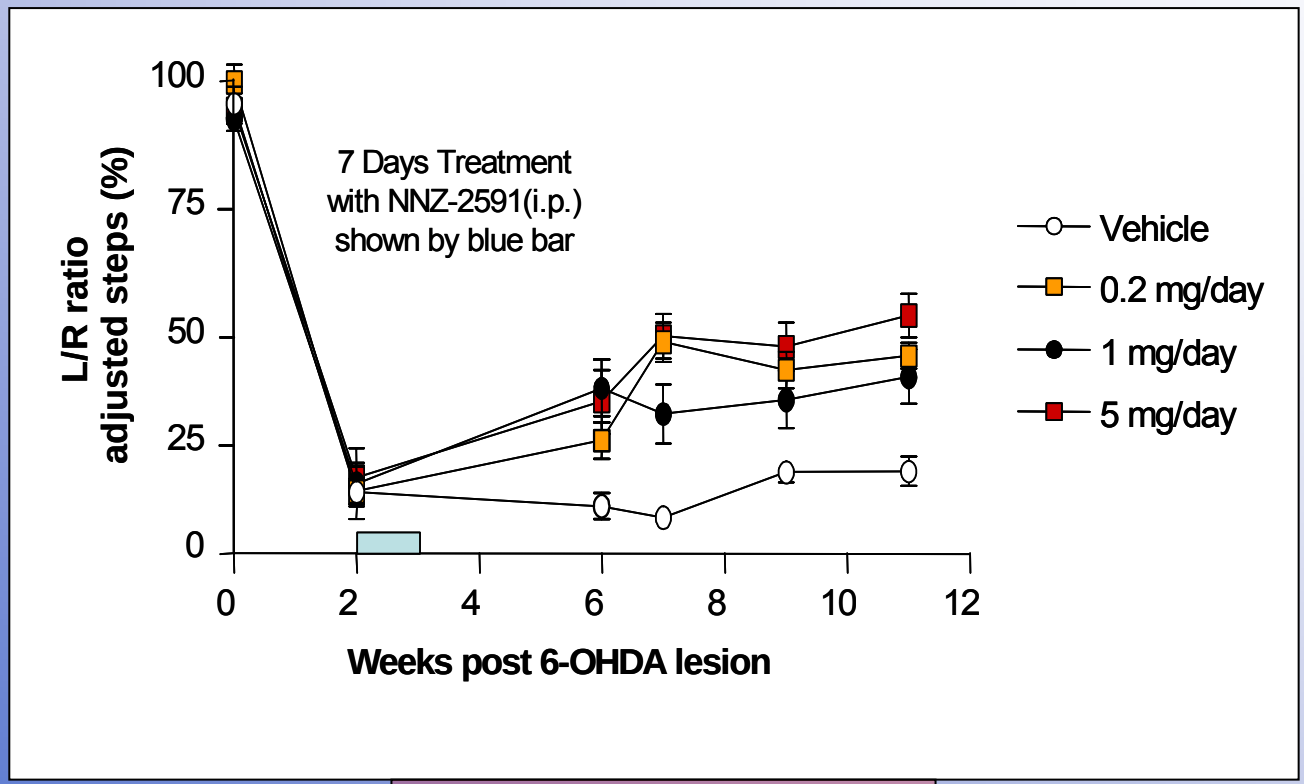
NNZ-2591: Chronic effects in Parkinson's disease



- NNZ-2591 given 2 weeks after striatal lesion
- 1 week treatment - prolonged behavioural improvement
- Anti-Parkinsonian effects at low systemic dose

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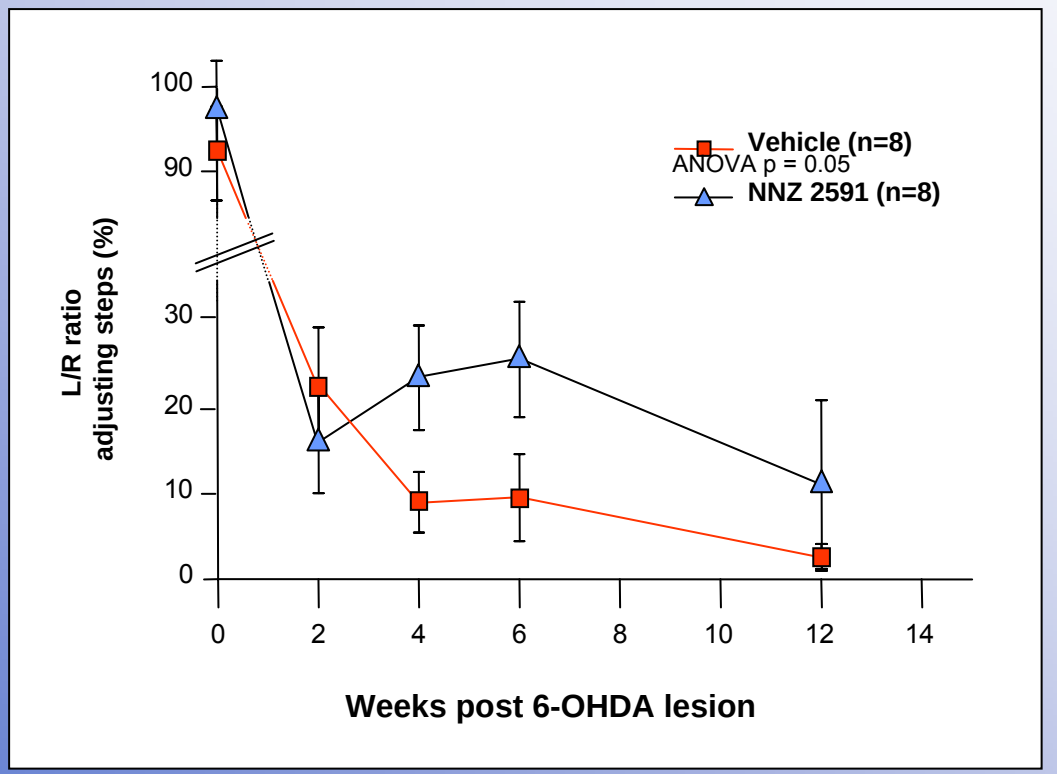
NNZ-2591 anti-Parkinsonian effect is centrally-mediated



- Parkinsonian disorder induced by intra-striatal 6-OHDA injection
- NNZ-2591 (20 ng) injected directly into rat cerebral ventricle 2 hours after 6-OHDA
- Partial restoration of motor function observed as in peripheral study

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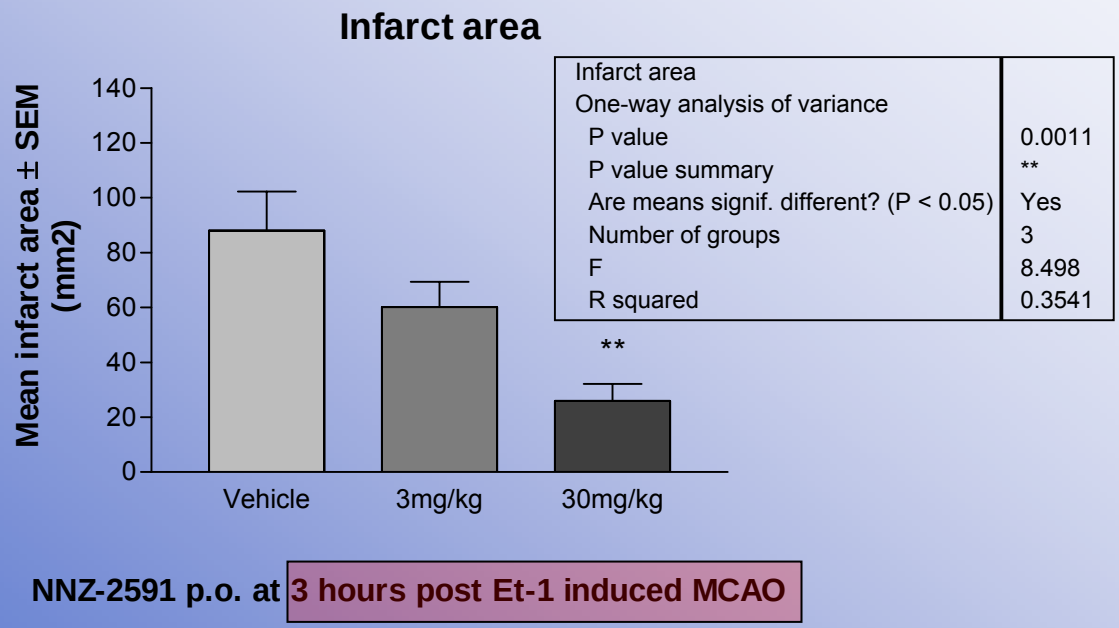
NNZ-2591 Oral Administration: Histopathology



NNZ-2591 p.o. at 3 hours post Et-1 induced MCAO

**P<0.01, ANOVA with Dunnett's post hoc test. Group sizes: Vehicle (12), 3mg/kg (11) , 30mg/kg (11)

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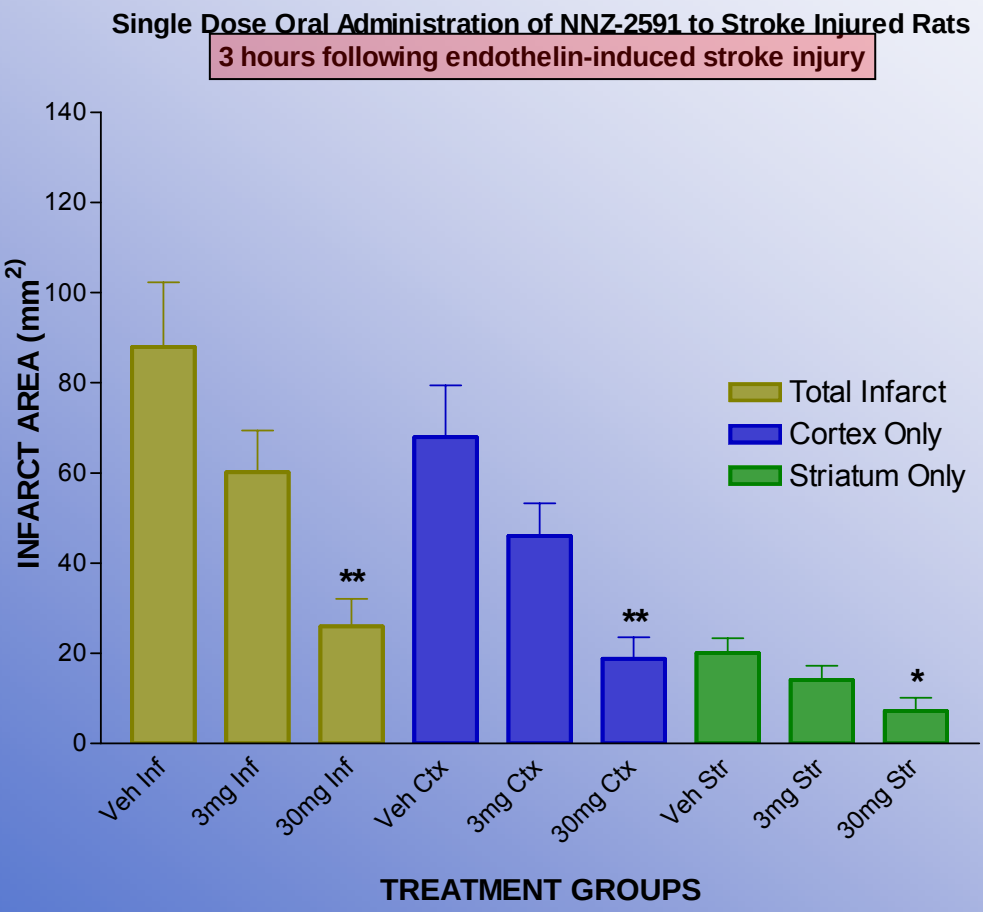
NNZ-2591 Protects Cortex and Striatum



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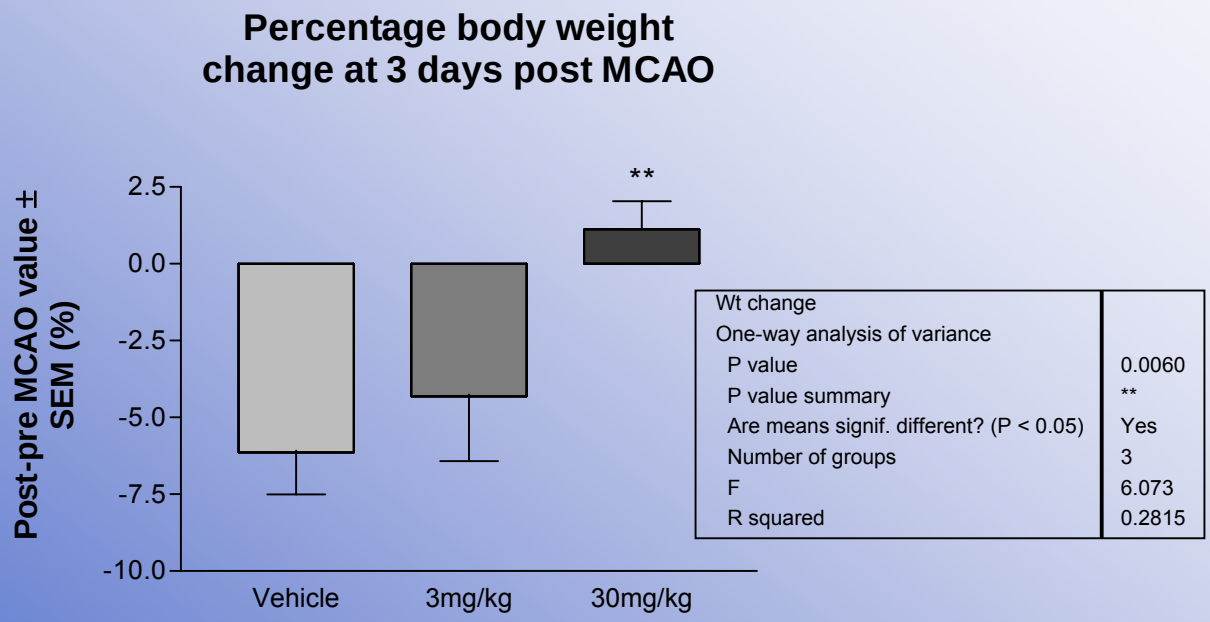
Oral Administration: Body Weight



NNZ-2591 p.o. at 3 hours post Et-1 induced MCAO

**P<0.01, ANOVA with Dunnett's post hoc test. Group sizes: Vehicle (12), 3mg/kg (11) , 30mg/kg (11)

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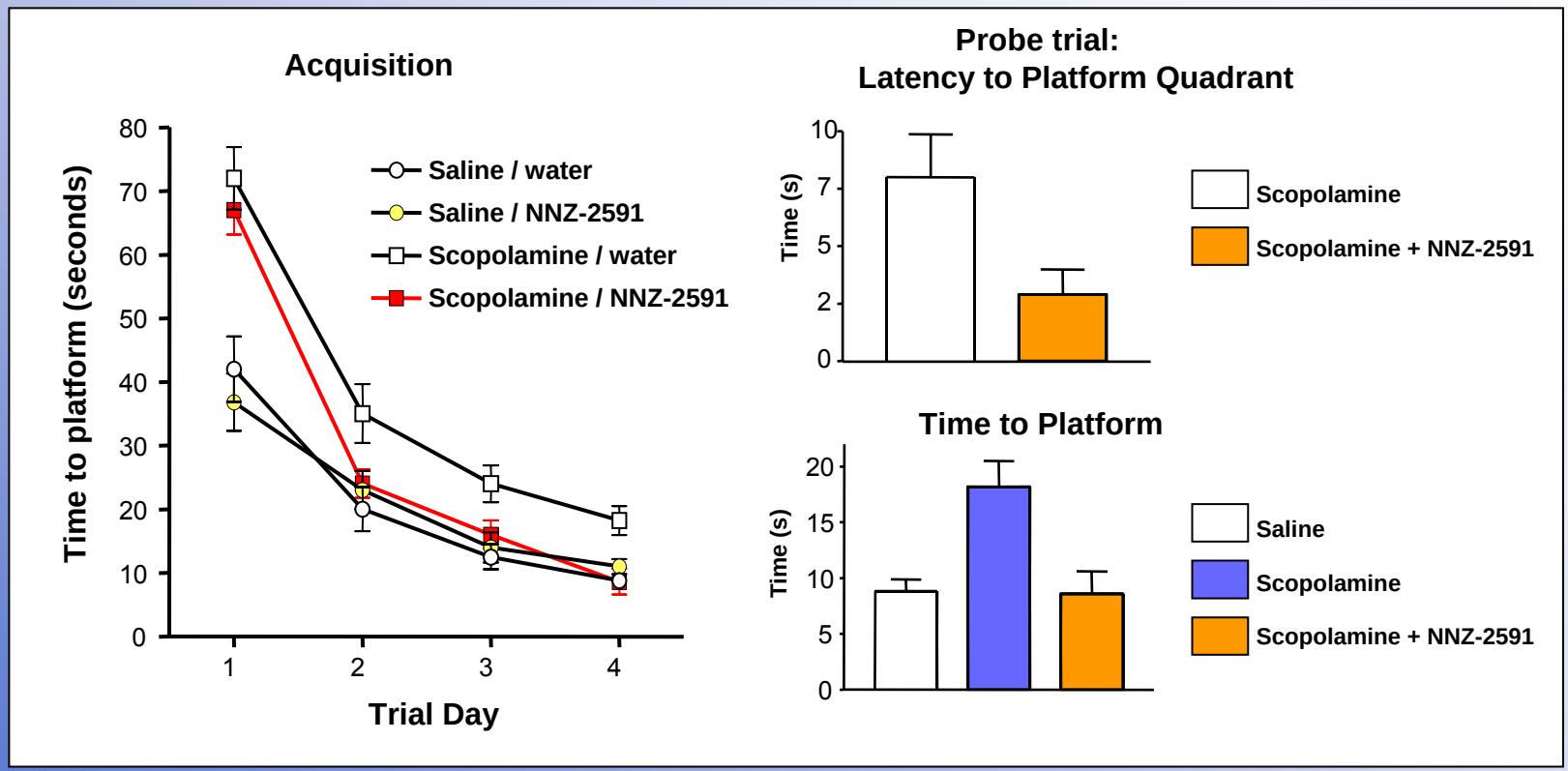
Oral Administration: Cognitive Performance



- Morris water maze study
- Scopolamine-impaired acquisition of spatial memory
- Oral NNZ-2591 (30 mg/kg; daily) completely reversed cognitive impairment induced by scopolamine

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Neural Regeneration Peptides

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Neural Regeneration Peptides – NRPs

- Neuren scientist discovered a novel mouse *Nrp* gene encoding a Neural Regeneration Protein
- Potent chemoattractive, neuronal survival-promoting factor
- Short peptide analogues (NRPs) synthesised (12-24 amino acids)
- Lead NRP: NNZ-4921
- Extremely potent
  - Highly active at 40 ng/kg
  - Minimum efficacious dose (further study): 40 pg/kg
- Multi-faceted pharmacology :
  - Promotes neuroprotection
  - Promotes neuronal migration and proliferation
  - Enhances neurite outgrowth
  - Promotes neuronal stem cell differentiation
- Targeted towards treatment of peripheral neuropathies

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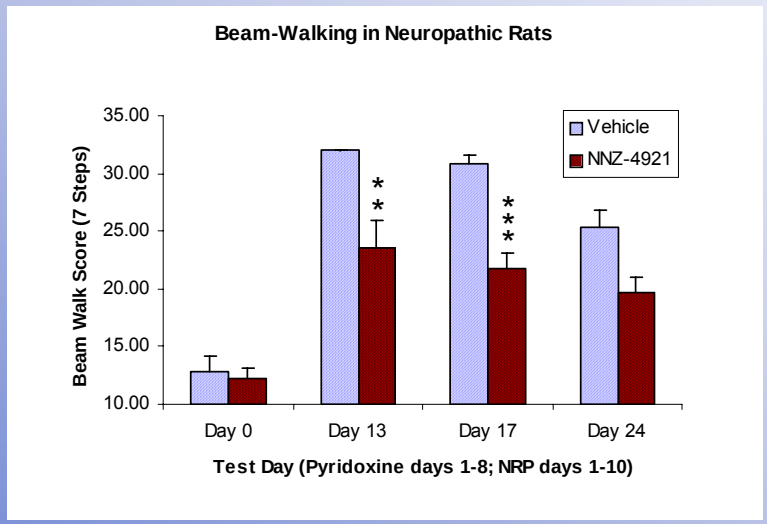
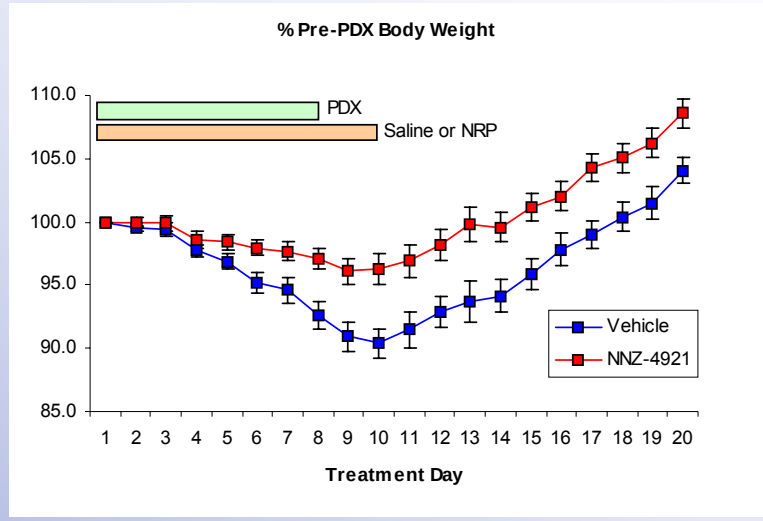
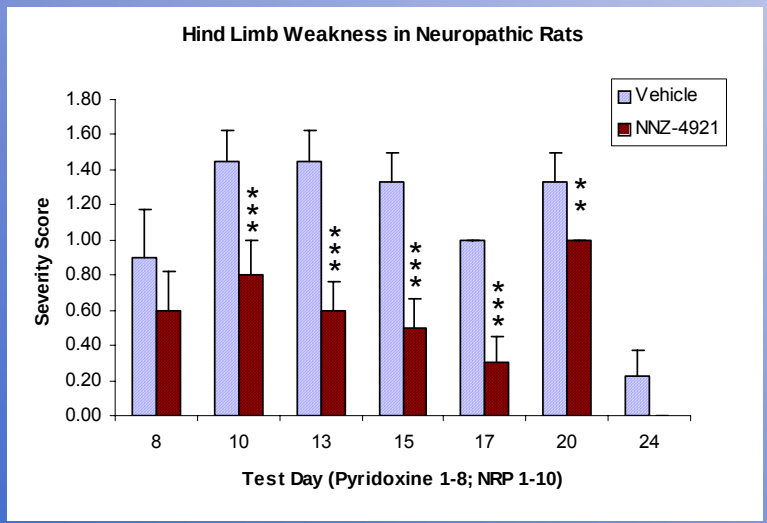
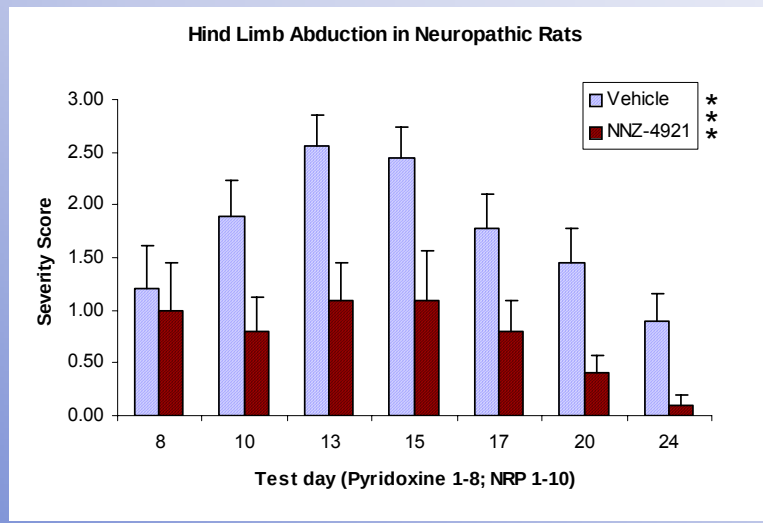
Positive Results in a Peripheral Neuropathy Model









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NNZ-8000

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NNZ-8000

- Growth hormone produced by cancer cells (autocrine GH) drives malignant phenotype, drug resistance
- NNZ-8000 inhibits extracellular signaling pathway (Trefoil Factors)
- In vitro and in vivo proof of principle with RNAi, peptidomimetic, polyclonal and monoclonal antibodies
- Antibodies targeted to conformational epitopes
- Therapeutic approach: fully human monoclonal antibody

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Pipeline Development Strategy

- Develop through registration
  - Glypromate®: cognitive decline post-CPB
  - Glypromate®: cardiac arrest
  - NNZ-2566 IV:
    - survival and neurological function in severe TBI
    - depression and memory in mild-moderate TBI
- Out-license
  - NNZ-2566 IV: acute stroke
  - NNZ-2566 oral: TBI/stroke recovery; chronic CNS
  - NNZ-2591: PD/PD dementia; other chronic CNS
  - NNZ-4921: peripheral neuropathy
  - NNZ-8000: GH-expressing cancers

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