Neurizon Therapeutics Limited
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Neurizon Presents Positive Preclinical Data on NUZ-001 at 7th Annual ALS Research Symposium

15 November 2024 – Melbourne, Australia: Neurizon Therapeutics Limited (ASX: NUZ & NUZOA) ("Neurizon" or "the Company"), a clinical-stage biotech company advancing treatments for neurodegenerative diseases, is pleased to announce that the Company's Managing Director and CEO, Dr. Michael Thurn, presented today at the **7th Annual ALS Research Symposium** hosted by ALS ONE. This prominent annual event brings together global experts, patients, and advocates to share the latest advances in Amyotrophic Lateral Sclerosis (ALS) research and treatment development.

The presentation, titled **"Exploring the Benefits of mTOR Inhibition in Patients with Amyotrophic Lateral Sclerosis,"** highlighted Neurizon's recent preclinical findings on its lead candidate, NUZ-001. These results demonstrate the therapeutic potential of NUZ-001 as an mTOR inhibitor to support neuronal health and slow disease progression, positioning NUZ-001 as a promising candidate in the fight against ALS.

Slides presented at the conference are available as an attachment to this announcement.

Key Preclinical findings presented:

- Positive preclinical results in human *in vitro* iPSC Motor Neuron models of Amyotrophic Lateral Sclerosis
- NUZ-001 and its major active metabolite significantly and dose-dependently reduced the aggregation of TAR DNA-binding protein 43 (TDP-43) by 50% and 65% respectively, in M337V Motor Neurons in response to a stressor
- Treatment with NUZ-001 and its major metabolite significantly improved electrophysiological dysfunction of TDP-43 mutated M337V Motor Neurons

About ALS Research Symposium

The ALS ONE 7th Annual ALS Research Symposium is recognized for fostering collaboration and patient-centered innovation in ALS research. The event brings together scientists, healthcare professionals, and advocates dedicated to advancing ALS treatments through cutting-edge research and shared knowledge.

-ENDS-

This announcement has been authorized for release by the Board of Neurizon Therapeutics Limited.

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About Neurizon Therapeutics Limited

Neurizon Therapeutics Limited (ASX: NUZ) is a clinical-stage biotechnology company dedicated to advancing treatments for neurodegenerative diseases. Neurizon is developing its lead drug candidate, NUZ-001, for the treatment of ALS, which is the most common form of motor neurone disease. Neurizon's strategy is to accelerate access to effective ALS treatments for patients while exploring NUZ-001's potential for broader neurodegenerative applications. Through international collaborations and rigorous clinical programs, Neurizon is dedicated to creating new horizons for patients and families impacted by complex neural disorders.

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We encourage you to utilise our Investor Hub for any enquiries regarding this announcement or other aspects concerning Neurizon.
This platform offers an opportunity to submit questions, share comments, and view video summaries of key announcements.

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Neurizon

7th Annual ALS Research Symposium

Exploring the Benefits of mTOR Inhibition in Patients with Amyotrophic Lateral Sclerosis

Dr Michael Thurn
Managing Director and Chief Executive Officer

15 November 2024

ASX: NUZ



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Product candidates for neurodegenerative diseases

Human Health Focus
Mid stage biotechnology company focused on large and growing markets in human health

Reformulating Monepantel
Reformulating an approved veterinary product – NUZ-001 – anthelmintic for sheep

Neurodegenerative Diseases
Exploiting autophagy as a hunter for toxic aggregates, a common pathology in neurodegenerative diseases

Motor Neurone Disease
Lead clinical program for the treatment of motor neurone disease/ Amyotrophic Lateral Sclerosis (MND/ALS)

Strong IP Position
Strong intellectual property with 6 patent families and protection beyond 2030

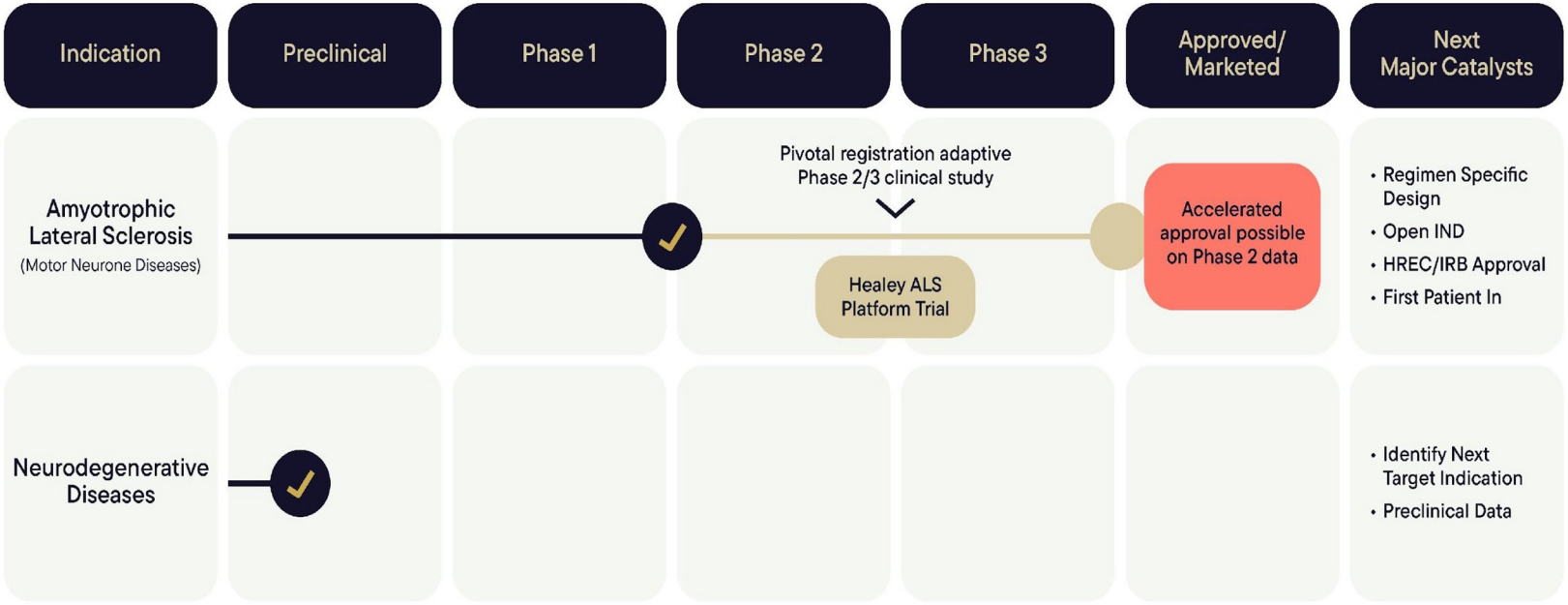
Pipeline Synergies
Pipeline synergies to leverage commercial infrastructure across development programs

Experienced Management
Experienced world-class Board and management team

Well-Funded
Healthy mix of loyal institutional and retail investors. Sufficient capital to support the pipeline

Pipeline

Multiple synergistic product opportunities in neurodegenerative disease by reformulating NUZ-001



- Single pivotal registration clinical study for ALS
- Targeting accelerated approval from Phase 2 data
- Selected into the HEALEY ALS Platform Trial reduces study cost and time, and increases patient participation rate

HREC – Human Research Ethics Committee; IND – Investigational New Drug; IRB – Institutional Review Board.

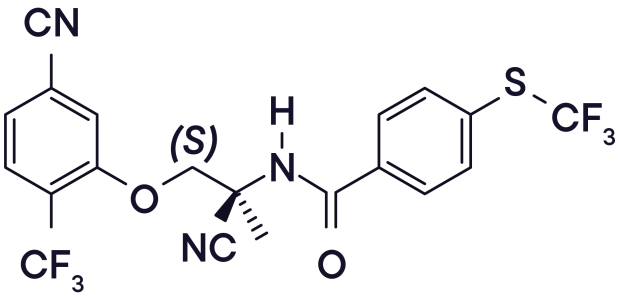
Monepantel (NUZ-001)

Active Substance, Pharmacological Class, History

Name: Monepantel (NUZ-001)
CAS No.: 887148-69-8

Pharmacological Class:
mTOR inhibitor

Monepantel S-enantiomer



Chemical Name

N-((1S)-1-Cyano-2-(5-cyano-2-trifluoromethyl-phenoxy)-1-methyl-ethyl)-4-trifluoromethylsulfanyl-benzamide

Structural Formula

(S)-monepantel enantiomer is used in the Drug Product for treatment of ALS

History

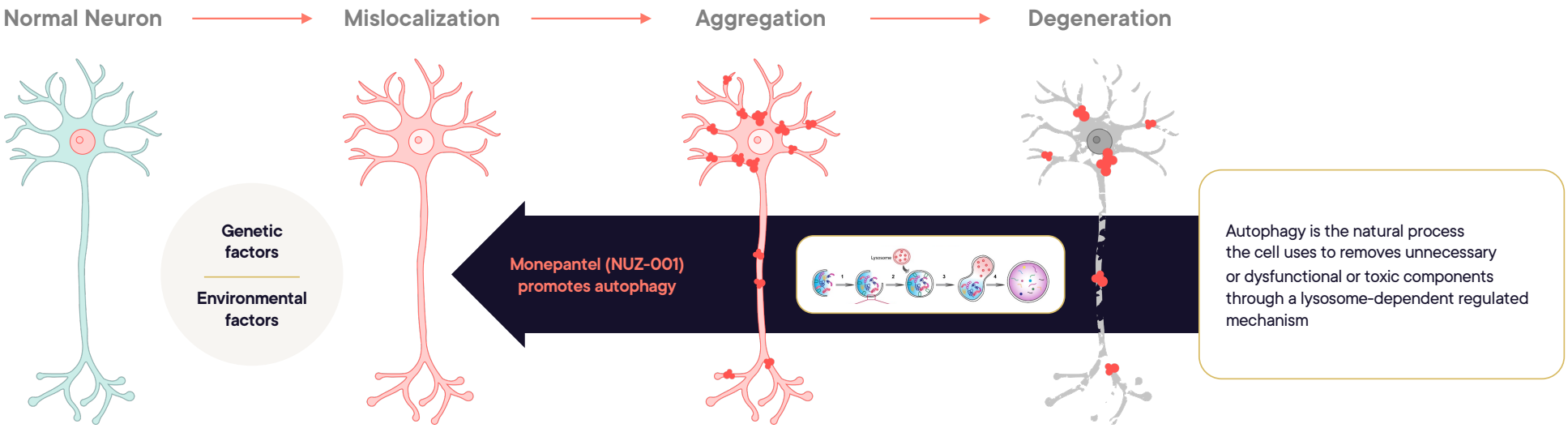
Monepantel (NUZ-001) was previously developed as the veterinary drug (Zolvix®), and has been approved since 2009 in Australia, New Zealand, and 36 other countries as an oral broad spectrum anthelmintic for the treatment of gastrointestinal roundworms in sheep. Mode of action as a de-worming agent is sheep-specific

MND/ALS
Mode of Action

NUZ-001 is an mTOR inhibitor

Nat Cell Biol (2019) Kim J et al./ J Mol Biol (2020) Djajadikerta A et al./Int J Molec Sci (2019) Thelling S et al.

- mTOR pathway is central to normal protein homeostasis in cells
- mTOR inhibition leads to reduced de novo net protein production and increased recycling of existing excessive or misprocessed cellular proteins
- Reduced phosphorylation of RPS6KB1 in PBMCs is a marker of mTOR inhibition

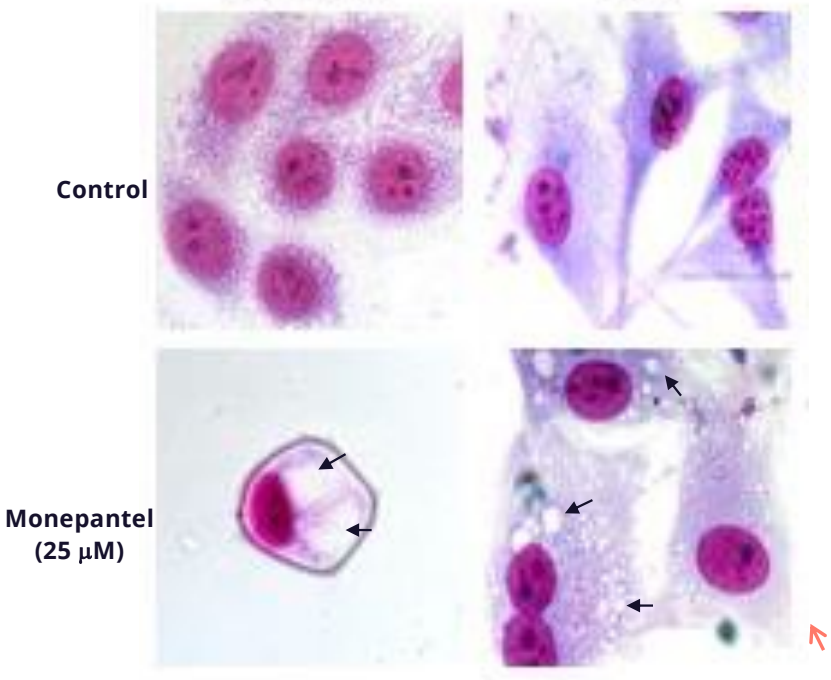


Nonclinical Data
NUZ-001 Induces Autophagy

Monepantel (NUZ-001) causes a clear development of intracytoplasmic cavities

Methods

- A2780 ovarian tumor cells, U87-MG glioma cells, and HOSE (ovarian surface epithelial) cells were examined after 72 hours of NUZ-001 treatment.
- Cavities were identified as lysosomal autophagic vacuoles using acridine orange staining.



Arrows depict autophagolysosomes (small lysosomal sacs or vacuoles that breaks down the cellular debris in cells during the process of autophagy)

Results

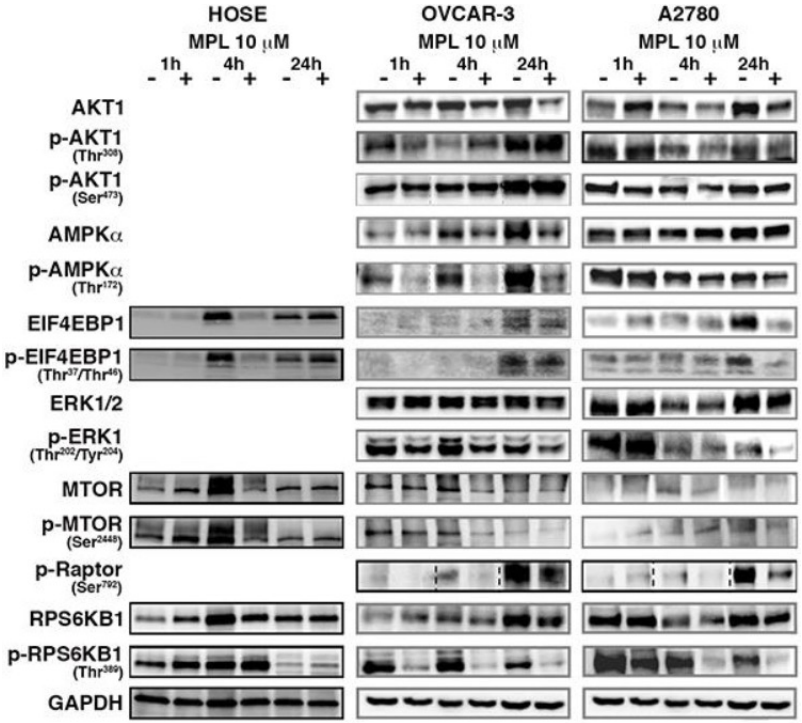
- **U87-MG cells: 20-fold increase at 72 hours**
- **A2780 cells: 100-fold increase at 72 hours**
- HOSE control cells: earlier increase (20-fold at 48 hours) that was no longer seen at 72 hours (conclusion: adaptation to culture conditions).

Nonclinical Data:
NUZ-001 Impacts Phosphorylation Status of Autophagy-Associated Proteins

Inhibition of mTOR results in changes in protein phosphorylation

Methods

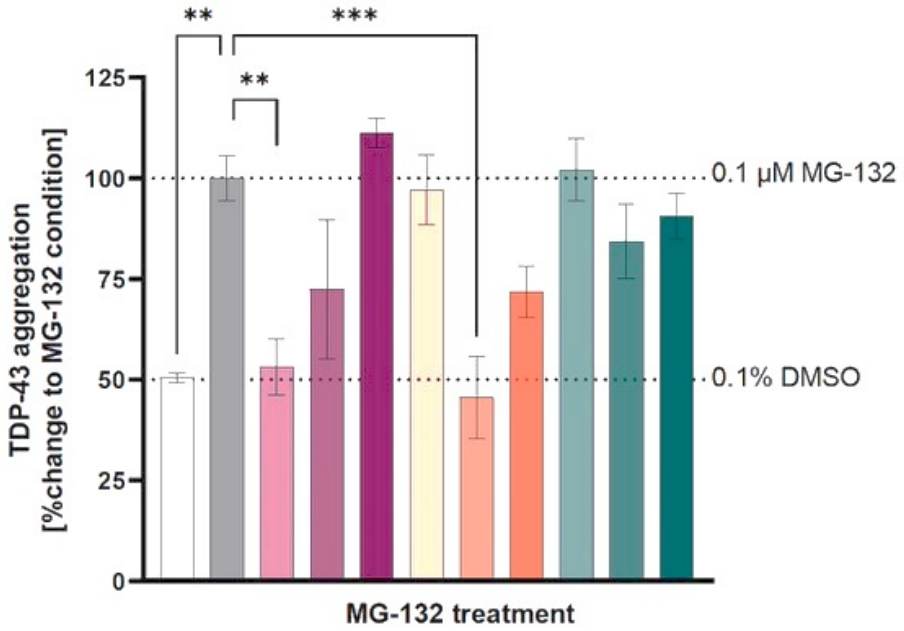
- Components of the PI3K/AKT/MTOR and (RAF)-MEK-ERK signaling pathways were investigated to determine changes in protein levels or phosphorylation status following NUZ-001 treatment.
- HOSE cells were controls as well as GAPDH protein.



Results

- NUZ-001 reduces RPS6KB1 and ERK1/2 protein phosphorylation, which are associated with increased autophagy
- Decreases RAPTOR phosphorylation, which is associated with inhibition of MTOR Complex I signalling and induction of autophagy

Nonclinical Data:
HTRF TDP-43 Aggregation Assay



- DMSO 0.1%
- MG-132 0.1 µM
- NUZ-001 0.625 µM
- NUZ-001 1.25 µM
- NUZ-001 2.5 µM
- NUZ-001 Sulfone 0.625 µM
- NUZ-001 Sulfone 1.25 µM
- NUZ-001 Sulfone 2.5 µM
- Rapamycin 0.625 µM
- Rapamycin 1.25 µM
- Rapamycin 2.5 µM

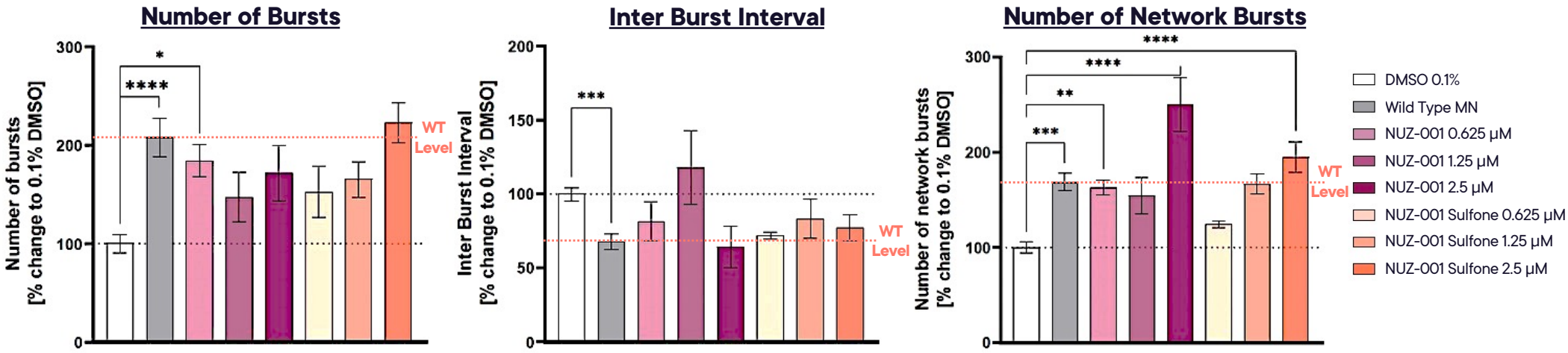
Methods

- Co-cultures of TDP-43 M337V Motor Neurons with astrocytes were treated with aggregation stressor (MG-132) and compounds for 7 days (from day 14 to day 21) at every medium change.
- At day 21 lysates were prepared from cultures and TDP-43 aggregation kit supplier's (Revity) recommendations were followed to perform the assay.

Dose-dependent effect of treatment was observed on TDP-43 aggregate reduction for:

- **NUZ-001 at 0.625 µM (50% reduction) and 1.25 µM (25% reduction)**
- **NUZ-001 Sulfone at 1.25 µM (statistically significant; 65% reduction) and 2.5 µM (25% reduction)**
- **Rapamycin at 3 µM (20% reduction)**

Nonclinical Data:
Multi-electrode Assay Activity



NUZ-001 and NUZ-001 Sulfone significantly improved the ALS TDP-43 M337V associated phenotype/MEA activity by increasing bursting (NUZ-001 0.625 µM p=<0.05) and network burst activity decreasing inter-burst interval (NUZ-001 0.625 µM p=<0.005; NUZ-001 2.5 µM p=<0.0005 and NUZ-001 Sulfone 2.5 µM p=<0.00005) at day 18 (corresponding to 4 days of treatment with compound).

Phase 1
ALS MEND Study Design

Patient Population:
11 Men, 1 Woman

- Adults with Familial or Sporadic ALS/MND
- 1st symptoms occurred < 3 years prior to screening
- Adequate bone marrow reserve, renal & liver function
- Seated Slow Vital Capacity ≥ 50% of predicted value
- **Median Age: 63.5 (42–78) years**

Intervention:
12 patients randomised

Open label, 24 hour escalating single dose PK study, followed by 4-week repeated escalating dose study

Treatment continued until dose escalation or End of Dosing

- **Cohort 1: 2 and 6 mg/kg/day dose levels**
- **Cohort 2: 4 and 10 mg/kg/day dose levels**

Primary & Exploratory Outcomes:

- Safety and tolerability
- Pharmacokinetic (NUZ-001 and its metabolite NUZ-001 sulfone in plasma and CSF) and pharmacodynamic (p-RPS6KB1 and p-EIF4EBP1 peripheral blood mononuclear cells)
- ALS Functional Rating Scale–Revised, ALS Quality of Life Questionnaire, Edinburgh Cognitive and Behavioural ALS Screen, slow vital capacity and biomarkers (serum neurofilament /light chain, CSF neurofilament/light chain and urinary p75 levels)

Study Objectives:

- Tolerability and safety of NUZ-001 with a goal of defining a maximally tolerated dose (MTD)
- Pharmacokinetics of NUZ-001 and its metabolite NUZ-001 sulfone in plasma and cerebrospinal fluid (CSF)
- Preliminary efficacy (ALSFRS-R)

Locations:

Two Centres in Australia

- Calvary Hospital Bethlehem, Melbourne
- Macquarie University, Sydney

Phase 1

ALS MEND: Demographics, Baseline Characteristics, ALS History, and Treatment Duration

- Most patients were male (91.7%)
- Median age was 63.5 years
- The most common site for the 1st disease onset was the Upper Limb region (58%)
- ALS diagnosis was considered Probable (58.3%) in most cases
- Median ALS duration (Time Since Symptom Onset) was 14.3 months
- Median Baseline ALSFRS-R was 39.5
- Median Pre-Baseline Slope was 0.74
- Median Treatment Duration was:
  - Dose Level 1 : 32.4 weeks
  - Dose Level 2 : 22.6 weeks
  - Dose Level 3 : 16.7 weeks
  - Dose Level 4 : 8.1 weeks

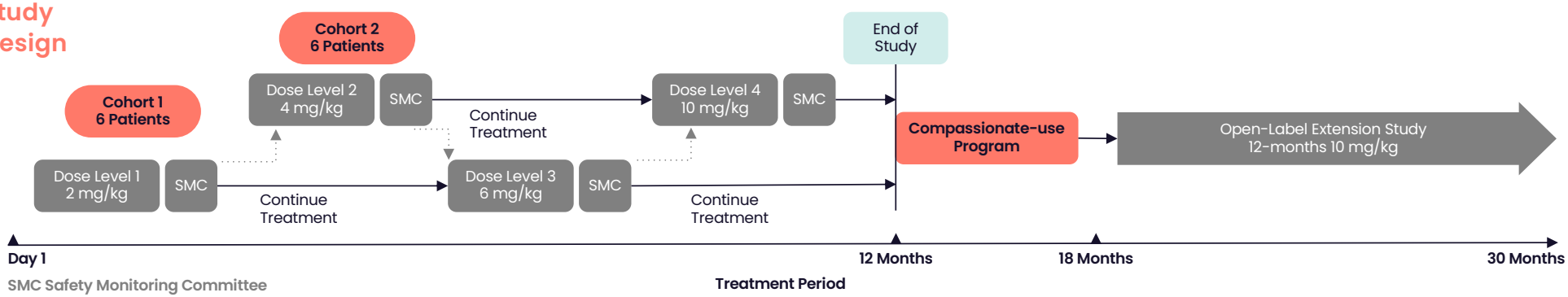
Gender, n (%)	
Female	1 (8.3)
Male	11 (91.7)
Age (years)	
Mean (SD)	61.9 (10.90)
Median (Min. Max)	63.5 (42,78)
Site of First Disease Onset, n (%)	
Lower Limb	2 (16.7)
Upper Limb	7 (58.3)
Limb Onset	1 (8.3)
Bulbar	2 (16.7)
ALS Diagnosis, n (%)	
Definite	5 (41.7)
Probable	7 (58.3)
Time since Onset (Months)	
Mean (SD)	14.7 (8.38)
Median (Min, Max)	14.3 (3.65, 34.0)

Baseline ALSFRS-R	
Mean (SD)	38.2 (5.10)
Median (Min, Max)	39.5 (28, 44)
Pre-Baseline Slope	
Mean (SD)	0.826 (0.461)
Median (Min, Max)	0.739 (0.210, 1.48)
Treatment Duration (weeks)	
Dose Level 1, Mean (SD)	28.1 (9.41)
Dose Level 1, Median (Min, Max)	32.40 (8.1, 34.1)
Dose Level 2, Mean (SD)	22.8 (2.86)
Dose Level 2, Median (Min, Max)	22.60 (20.1, 26.3)
Dose Level 3, Mean (SD)	17.0 (1.70)
Dose Level 3, Median (Min, Max)	16.70 (15.1, 20.1)
Dose Level 4, Mean (SD)	8.2 (0.13)
Dose Level 4, Median (Min, Max)	8.10 (8.1, 8.4)

Phase 1
ALS MEND Study

The Phase 1 MEND Study was an open label, multicentre study involving 12 patients with ALS with the goal of determining the recommended Phase 2/3 dose based on safety and preliminary efficacy

Study Design



Study Update



- Positive top-line data released in Q1 CY24
- 12 patients continued treatment with NUZ-001 under a compassionate-use program
- 10 patients rolled-over into 12-month Open-Label Extension Study. Treatment continues to be very well-tolerated

- Updated ALSFRS-R and Survival Analysis to be generated by Berry Consultants.
- First group of 6 patients are entering their 24th month of continuous treatment with NUZ-001
- Phase 1 and baseline OLE data used to design pivotal registration adaptive Phase 2/3 Study, to commence in H1 CY25

Phase 1
Safety and Tolerability Summary

No deaths, no Serious Adverse Events related to treatment and a very low incidence of Adverse Events

Incidence of Adverse Event (n)					
	Dose 1 (2 mg/kg)	Dose 2 (4 mg/kg)	Dose 3 (6 mg/kg)	Dose 4 (10 mg/kg)	Total
Adverse Events	29	6	12	9	56
Related to Treatment	2	1	–	–	3

- **Only 3 Adverse Events (mild in severity) possibly related to treatment**
  - Raised liver enzymes
  - Increased hair growth
  - Constipation
- **No deaths**
- **No patients withdrew or were discontinued from the study**
- **One Serious Adverse Events (SAEs) reported that was unrelated to treatment**
  - 1 patient (Dose Level 3-6 mg/kg)
  - Hospitalised for Intestinal dilatation and Pneumonia

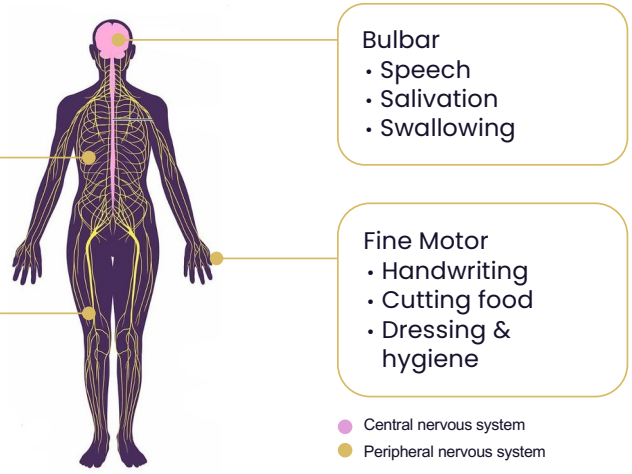


Phase 1

Preliminary Efficacy Amyotrophic Lateral Sclerosis Function Rating Scale – Revised (ALSFRS-R)

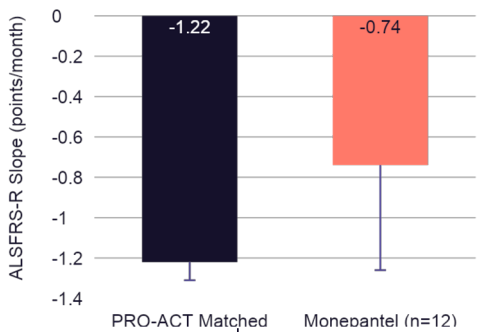
Treatment with NUZ-001 for up to 12 months slowed the progression of ALS in all 12 patients by 39% when compared to matched controls from the PRO-ACT database[1]

ALSFRS-R Domains Assessed



Each task is rated on a five-point scale from 0 = can't do, to 4 = normal ability.
Individual item scores are summed to produce a reported score of between 0=worst and 48=best.

NUZ-001 – 39% Slower Decline in ALSFRS-R



Northeast ALS Consortium (NEALS) clinically meaningful = 20% to 25% or greater change in the slope of ALSFRS-R[2]

Difference
0.48 points/month (p=0.08)

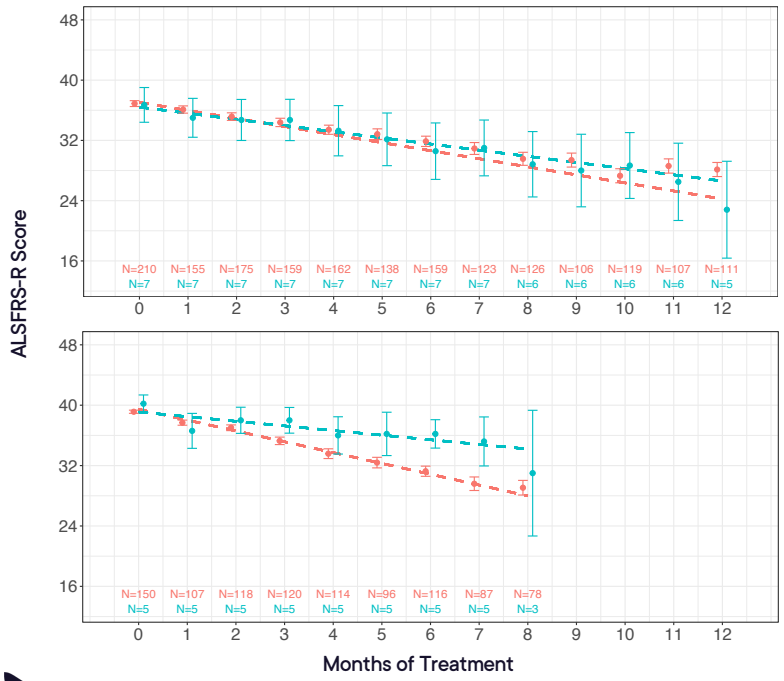
The PRO-ACT database is the largest publicly available repository of merged ALS clinical study data. Data were pooled from 16 completed Phase 2/3 ALS clinical studies and one observational study. Over 8 million de-identified longitudinally collected data points from more than 8,600 persons with ALS, including demographics, family histories, and longitudinal clinical and laboratory data.

1. Atassi N, Berry J, Shui A, Zach N, Sherman A, Sinani E, Walker J, Katsovskiy I, Schoenfeld D, Cudkowicz M, Leitner M. The PRO-ACT database: design, initial analyses, and predictive features. Neurology. 2014 Nov 4;83(19):1719-25. doi: 10.1212/WNL.0000000000000951.Epub 2014 Oct 8. PMID: 25298304; PMCID: PMC4239834.
2. Castrillo-Viguera C, Grasso DL, Simpson E, et al. Clinical significance in the change of decline in ALSFRS-R. Amyotroph Lateral Scler. 2010;11(1-2):178-180.

Phase 1

Preliminary Efficacy Amyotrophic Lateral Sclerosis Function Rating Score – Revised (ALSFRS-R)

Dose response observed between Cohort 1 and 2. Rate of disease progression in Cohort 2 was slowed by 58% compared to the PRO-ACT database matched controls



Cohort 1

-0.83 points per month

23% Slower Decline in ALSFRS-R

Cohort 2

-0.60 points per month

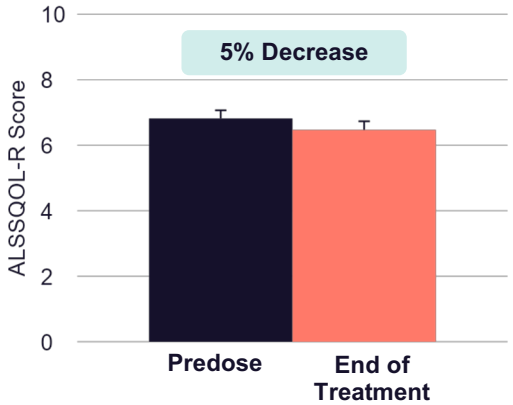
58% Slower Decline in ALSFRS-R

Neurizon

Phase 1
Exploratory Endpoints

No significant difference in ALS Quality of Life Questionnaire, Edinburgh Cognitive and Behavioural ALS Screen, and Slow Vital Capacity between predose and end of treatment

ALSSQOL-R
Quality of Life Questionnaire

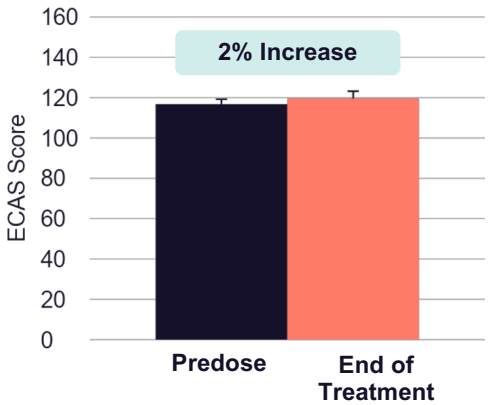


Small (non-statistically significant) numerical reduction in mean ALSSQOL-R score for All Subjects

This stable result appears to represent a slower decline for study subjects compared to what is expected without treatment.

The mean pre-study screening ALSSQOL-R score for All Subjects was 6.829, and the mean end of dosing ALSSQOL-R score was 6.464

Edinburgh Cognitive and Behavioural ALS Screen

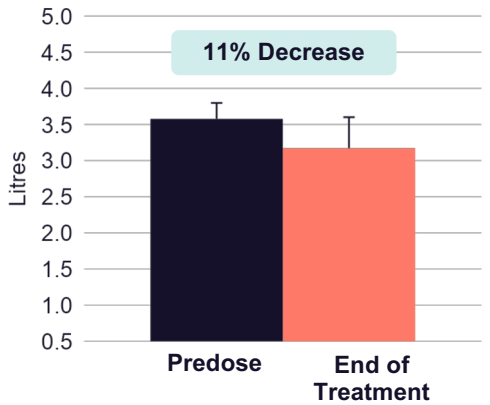


Small (non-statistically significant) numerical increase in mean ECAS score for All Subjects

The small increase/stable result over the treatment period may indicate a protective effect on cognitive function

The mean pre-study ECAS score for All Subjects was 116.5, and the mean end of dosing ECAS score was 119.3

Slow Vital Capacity



Small (non-statistically significant) numerical decrease in mean SVC measurement for All Subjects

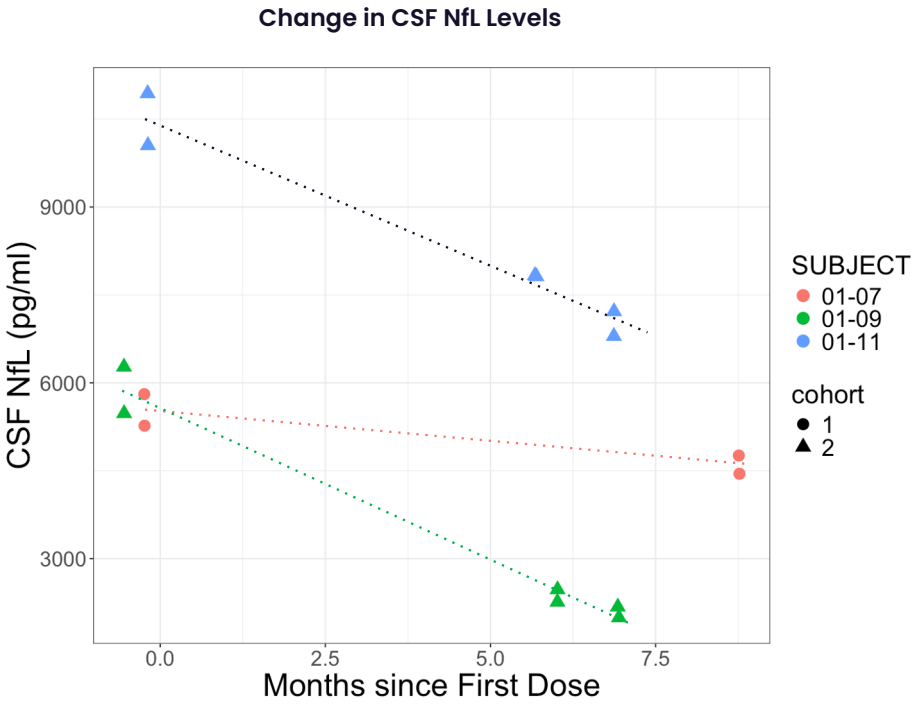
The small decrease/stable result over the treatment period may indicate a protective effect on respiratory function

The mean pre-study screening SVC for All Subjects was 3.508 L, and the mean end of dosing SVC was 3.026 L

Phase 1
Exploratory Biomarkers Assessment

Neurofilament levels correlate with disease progression rate in ALS and higher levels of neurofilament are associated with faster/greater decline of ALSFRS-R over time[1]

- Marked decreased NfL cerebrospinal fluid levels in 3 patients
- Decline of 6.9% (14.0 %, 0.9%) per month in CSF NfL levels
- Predose values ranged from 5540.53 to 10500.13 pg/mL and had decreased at Day 29 of their second Dose Level for All Subjects (reduction ranged from 17.0 to 64.5%)
- Although limited data, the CSF NfL data is encouraging
- NfL samples continue to be collected in the OLE study with results due in H2 CY24



1. Brodovitch A, Boucraut J, Delmont E, Parlanti A, Grapperon A-M, Attarian S, et al. Combination of serum and CSF neurofilament-light and neuroinflammatory biomarkers to evaluate ALS. Sci Rep. 2021;11(1):703. doi: 10.1038/s41598-020-80370-6.

Phase 1
Exploratory Biomarkers Assessment

No significant difference in biomarkers serum neurofilament /light chain (NfL) and urinary p75^{ECD} levels between pre-dose and end of treatment

Neurofilament Light Chain Serum

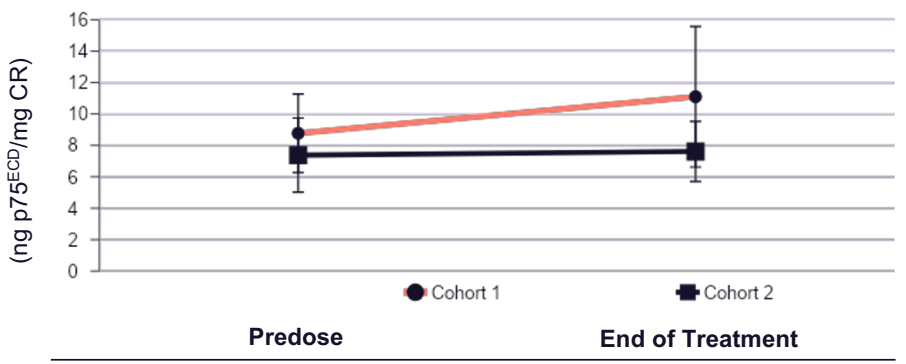


Mean predose levels of serum NfL were 51.62 pg/mL for All Subjects, remaining relatively steady to 52.56 pg/mL at End of Dosing.

For the 3 CSF subjects, NFL serum levels were:

- *01-07 – Pre-dose 37.39 pg/mL; EoD 41.29 pg/mL*
- *01-09 – Pre-dose 25.99 pg/mL; EoD 23.42 pg/mL*
- *01-11 - Pre-dose 108.37 pg/mL; EoD 90.58 pg/mL*

Urinary p75^{ECD}/Creatinine



Mean predose urinary p75^{ECD} levels were 8.357 ng p75^{ECD}/mg creatinine for All Subjects, remaining relatively steady to 9.821 ng p75^{ECD}/mg creatinine at end of dosing, and remained stable in Cohort 2 subjects.

Urinary p75^{ECD} levels increase on average 0.19 ng/mg of creatinine per month as the disease progresses.

Phase 1
Conclusion

Positive Phase 1 study results showed that NUZ-001 has an excellent safety profile and the ability to slow the progression of MND/ALS

Highlights

Primary Objectives

- Excellent safety and tolerability profile

Preliminary efficacy data

- Slows the rate of progression by 58% (Cohort 2 High Dose)

Blood Brain Barrier

- NUZ-001 and its active metabolite, NUZ-001 Sulfone, detectable in cerebrospinal fluid

Competitive Advantage

- Superior safety, tolerability and preliminary efficacy profile to other FDA approved drugs

Survival

- Favourable comparison to historical control subjects from PRO-ACT database

Phase 2/3 Dose

- Optimal dose selected for pivotal Phase 2/3 clinical study

Phase 1

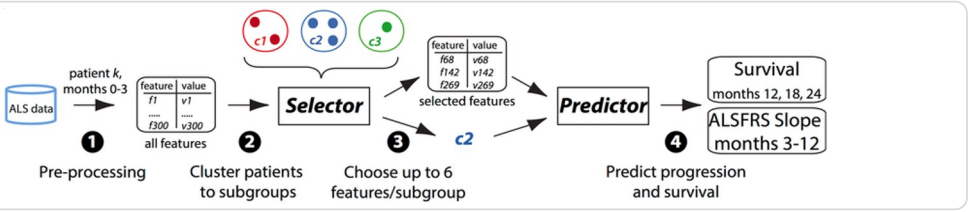
ALS Open Label Extension Study

Compared to matched controls from the PRO-ACT Historical Database, treatment with NUZ-001 significantly (X^2=12.83, p=0.00034) prolongs the survival of patients with ALS

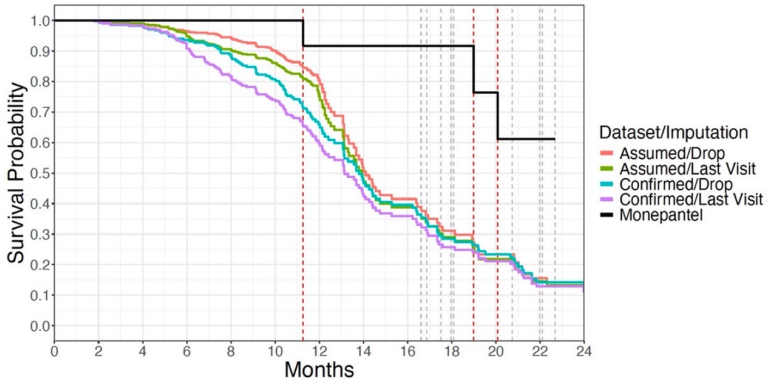
Berry Consultants Statistical Analysis

- Berry's analysis involved comparing patients in the PRO-ACT database with similar characteristics to those in Neurizon's Phase 1 MEND Study adjusting for differing diagnosis durations
- Hazard ratio of 0.197 (95% CI: (0.062, 0.627), p = 0.0059) indicating that treatment with NUZ-001 reduces the risk of death by 80.3%

Analysis Method		Log-Rank Test		Cox Proportional Hazards Model		
Dataset	Death Time Imputation	χ^2	p-value	Hazard Ratio	95% CI	p-value
Assumed Survival	Leave out	12.82	0.00034	0.197	(0.062, 0.627)	0.0059
	Last Visit	14.07	0.00018	0.187	(0.059, 0.593)	0.0044
Confirmed Survival	Leave out	14.52	0.00014	0.186	(0.059, 0.586)	0.0024
	Last Visit	16.35	0.00005	0. 173	(0.055, 0.546)	0.0015

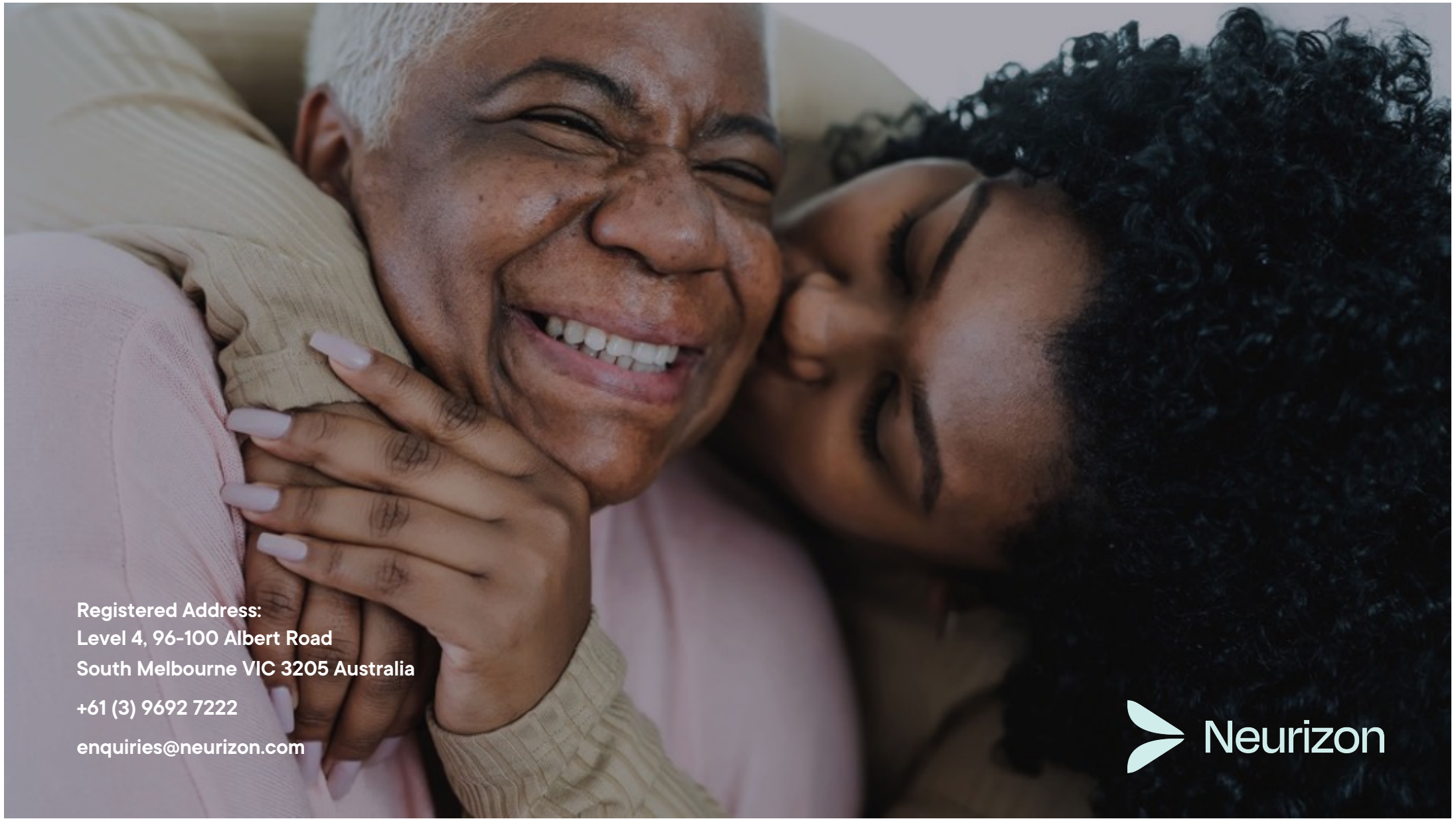


Overall Survival Probability



The PRO-ACT database is the largest publicly available repository of merged ALS clinical study data. Data were pooled from 16 completed Phase 2/3 ALS clinical studies and one observational study. Over 8 million de-identified longitudinally collected data points from more than 8,600 persons with ALS, including demographics, family histories, and longitudinal clinical and laboratory data.

[1]Atassi N, Berry J, Shui A, Zach N, Sherman A, Sinani E, Walker J, Katsovskiy I, Schoenfeld D, Cudkowicz M, Leitner M. The PRO-ACT database: design, initial analyses, and predictive features. Neurology. 2014 Nov 4;83(19):1719-25. doi: 10.1212/WNL.0000000000000951.Epub 2014 Oct 8. PMID: 25298304; PMCID: PMC4239834.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