Trickle Research

Every raging river, every great lake, every deep blue sea starts … with a trickle

Initiating Research Coverage

Neurizon Therapeutics Limited

(OTC: NUZTF)

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12- 24 month Price Target: USD$.32

Allocation: 4

Closing Stock Price at Initiation (Closing Px: 03/20/26): USD$.11

(All $ references in this report are in Australian Dollars {"AUD"} unless otherwise noted. However, because the shares trade on the U.S. OTC market, the closing price information above as well as the price target information is in U.S. Dollars)

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Company Overview

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 neuron disease. Neurizon's strategy is to accelerate access to effective ALS treatments for patients while exploring the potential of NUZ-001 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. NUZ-001 is an investigational product and is not approved for commercial use in any jurisdiction.

We're focused on optimizing quality of life for people living with neurodegenerative diseases and moving forward at pace to make innovative treatments available sooner. Our scientific expertise is driving this momentum. After a decade of research and development, we are pioneering novel applications for NUZ001 (S-Monepantel). Long-term safety, tolerability, and strong signals of efficacy point to the real possibility that a life-changing new treatment for Amyotrophic Lateral Sclerosis (ALS) is on the horizon. Our work also holds promise for other neurodegenerative diseases, including Alzheimer's, Parkinson's and Huntington's Disease, opening new possibilities for millions of patients worldwide.

At Neurizon, everything we do is guided by our mission: to bring forward next-generation treatments that makea real difference in the lives of patients and their loved ones living with neurodegenerative diseases. Our goal is to create hope by delivering meaningful progress for people living with neurodegenerative diseases, and to advance innovative, disease-modifying therapies that address the underlying pathology and drivers of neurodegenerative diseases like Amyotrophic Lateral Sclerosis (ALS), the major form of Motor Neurone Disease (MND), Huntington's Disease (HD), and others. We are building more than a biotech company – we are creating a patient-centered organization grounded in scientific credibility, transparency, and authentic partnership with the communities we serve. With strong global partnerships and a clear clinical pathway, we are building momentum toward regulatory milestones and long-term value creation.

The strength of our clinical foundation and increased level of scientific confidence is best evidenced through the successful completion of our Phase I trial and Open-Label Extension study using NUZ-001, leaving the company well-positioned for participation in the HEALEY ALS Platform Trial initiated in February of this year. This platform provides a globally recognised, patient-centric and capital-efficient pathway to the delivery of Phase 2 data – the next key step in our clinical development pathway, prior to broader commercialisation opportunities.

We were introduced to Neurizon by our colleagues at Integrous Communications (Home | Integrous Communications), who we have known for many years. As generalist, we review a fair number of biotech companies, as well as some others in healthcare research (devices for instance), and we have historically put a few of these under coverage. However, we submit we are typically quite cautious about the space. On one hand, we are especially attracted to companies/ideas that we think can create meaningful contributions towards making the world a better place. Small healthcare related companies trying to develop therapies to help people who are suffering and/or dying from rare diseases certainly fit those criteria. On the other hand, our research approach is also unapologetically capitalist, and while that does not mean we will cover *anything* we think will make money regardless of its social impact, finding ideas that work is always near the top of the page. With that in mind, small companies trying to develop therapies or devices to address disease face a very challenging road on a variety of fronts, and we will address some of those challenges throughout this report. That said, for several reasons we will also cover in this report, Neurizon has managed to position itself in a way that should help them avoid some of those typical challenges or at least mitigate them vis-à-vis many of the other small biotech enterprises we have evaluated in the past, and that may be

true with respect to their position relative to other failed ALS drugs as well. That posture is the basis for our thesis and enthusiasm for Neurizon Therapeutics Limited.

Neurizon is based in Melbourne, Victoria, Australia listed on the Australian Securities Exchange (ASX: NUZ) and recently began trading in the U.S. on the OTCQB under the symbol NUZTF. The Company was founded in 2000 under the name PharmAust and changed its name to Neurizon Therapeutics Limited in October 2024.

Industry Overview

- **What is ALS**

From Cleveland Clinic (Amyotrophic Lateral Sclerosis (ALS): What It Is & Symptoms):

Amyotrophic Lateral Sclerosis ("ALS") is a neurodegenerative condition that affects how nerve cells communicate with your muscles. It leads to muscle weakness that gets worse over time. Symptoms can affect how you move, speak and breathe. ALS treatment includes therapies and medications to manage symptoms and slow the progression of the disease.

You might notice muscle weakness and muscle twitching that affect your ability to walk independently, reach for objects, chew food and talk. ALS eventually causes your muscles to waste away (atrophy). Atrophy can make it harder to breathe and may lead to life-threatening outcomes. Although there's no cure, treatments are constantly improving. The right combination may slow the progression of the disease and improve quality of life.

There are two types of ALS based on their cause:

- *Sporadic ALS. The most common type makes up about 90% of cases. It occurs randomly and isn't inherited.*
- *Familial ALS. It makes up about 10% of cases. It's caused by inherited gene changes passed down from a parent.*

ALS symptoms include:

- *Muscle weakness (arms, legs and neck)*
- *Muscle cramps*
- *Twitching in your hands, feet, shoulders and/or tongue*
- *Stiff muscles (spasticity)*
- *Speech challenges (slurring words, trouble forming words) • Drooling*
- *Unintentional emotional expressions (like laughing or crying)*
- *Fatigue*
- *Trouble swallowing (dysphagia)*
- *Early symptoms are usually muscle weakness or stiffness in your arms and legs, as well as trouble with speech and swallowing. These can make everyday tasks like writing or eating more challenging. Over time, symptoms typically spread throughout your body. The speed at which symptoms progress varies from person to person.*
- *As symptoms get more severe, you may have trouble breathing, standing or walking. You might experience significant weight loss. Contact your healthcare provider if symptoms worsen, and seek emergency care if you're having trouble breathing.*

Researchers don't know what causes amyotrophic lateral sclerosis. They believe it's a combination of the following factors:

- *Genetics. About 70% of familial cases and 5% to 10% of sporadic cases involve gene changes. They're most often in the C9orf72, SOD1, TARDBP and FUS genes. There are more than 40 related genes.*
- *Environment. Exposure to toxins (like lead or mercury), viruses or trauma may also play a role.*

What researchers do know is that the disease targets motor neurons. These regulate your voluntary movements, like talking, chewing, moving your limbs and breathing.

Your neurons communicate with your muscles to tell them to move. ALS disrupts communication, like bad phone reception. The messages sent from neurons to muscles break up and don't get through clearly. This eventually causes the call to end. As a result, neurons can't take any new calls, causing you to experience symptoms.

Some types of amyotrophic lateral sclerosis are genetic. You can inherit genetic changes that cause ALS from your biological parents. Inherited ALS isn't common, though. Sometimes, genetic changes happen randomly, without a history in your biological family.

Risk factors for amyotrophic lateral sclerosis include:

- *Age. You're most likely to develop symptoms between the ages of 55 and 75.*
- *Race and ethnicity. White (non-Hispanic) people are most likely to get ALS.*
- *Sex. For cases that occur before age 55, men are at higher risk.*
- *Veterans. Military veterans may be at higher risk. Researchers suggest it's from environmental exposure (toxins or pesticides) or physical trauma.*

- **History of ALS**

From NIH, (Jean-Martin Charcot: The Father of Neurology - PMC):

"ALS was identified in 1869 by French neurologist Jean-Martin Charcot, who is often viewed as the "Father of Neurology". ..."Similar to Charcot's other discoveries, it was through careful clinical observation and meticulous work in the laboratory detailing the pathophysiology that allowed for establishment of amyotrophic lateral sclerosis (ALS) as a separate disease. He described and diagnosed the first cases of ALS as a specific neurological disease associated with a distinct pathology. Studies conducted between 1865 to 1869 by Charcot and his colleague Joffroy found that lesions within the lateral column in the spinal cord resulted in chronic progressive paralysis and contractures (no atrophy of muscles), while lesions of the anterior horn of the spinal cord resulted in paralysis without contractures (with atrophy of muscles).

These findings supported his hypothesis at the time that the motor component of the spinal cord consisted of a two-part system, and that the location of the lesion results in a varying clinical presentation. While the term "amyotrophic lateral sclerosis" was not offered by Charcot until 1874 when his lectures were compiled into a collection of his work titled "Oeuvres Completes," ALS is still referred to as Charcot's disease in many parts of the world. While numerous molecular and genetic discoveries have allowed for a greater understanding of this disease, his original descriptions of the associated clinical and pathological findings of ALS have remained virtually unaltered..."

From the ALS Association, (History of ALS | The ALS Association):

ALS became more widely known internationally on June 2, 1941, when it ended the career of one of baseball's most beloved players, Lou Gehrig. For many years, ALS was commonly known as Lou Gehrig's disease. Born in New York City to the son of German immigrants, Heinrich and Christina Gehrig, Gehrig attended Columbia University on a football scholarship to study engineering, yet he also played the sport for which he is best known during his tenure at the Ivy League school. Gehrig played with the New York Yankees for 17 years and received the moniker "The Iron Horse" due to his ability to play baseball despite suffering from a variety of injuries.

Gehrig was diagnosed with ALS on his 36th birthday during a visit with his wife Eleanor to the Mayo Clinic in Rochester, Minnesota, on June 19, 1939. Prior to his diagnosis, Gehrig noticed several of the disease's symptoms while playing on the field, including a loss of strength, slipping, falling and loss of coordination.

Charcot's discoveries provided the groundwork for a better understanding of ALS, as he recognized some of the common characteristics ("*clinical presentation*") that would ultimately define the disease, and those included things like spinal lesions, motor neurons degeneration, progressive muscle wasting and ultimately death.

Since Chalcot's discoveries, the overall understanding of the disease now includes a much clearer picture of its biological and molecular characteristics, unfortunately, that understanding has resulted in only limited success in the development of treatments for the disease. For instance, there are currently only three FDA approved ALS drugs on the market today, and they collectively provide modest benefit for ALS patients. Specifically, Riluzole (now sold generically by various manufacturers under different brand names) typically extends survival 2 to 3 months, Edaravone/Radicava (primarily marketed by the Japanese pharmaceutical company Mitsubishi Tanabe Pharmaceutical, which was acquired by Baine Capital in 2025 for $3.4 billion) provides modestly slower disease progression in some patients, and Tofersen (Biogen Inc. -Nasdaq: BIIB) has exhibited efficacy in a subset (SOD1) of the *familial* ALS population, which represents 1%- 2% of the *overall* ALS population. Specifically, a 2014 Lancet study (Clinical trials in amyotrophic lateral sclerosis: why so many negative trials and how can trials be improved? - ScienceDirect) notes that *"more than 50 randomised controlled trials ("RCT") of proposed disease-modifying drugs have failed to show positive results in the past halfcentury. In the past decade, at least 18 drugs have been tested in large phase 2 or 3 RCTs"*. That is a sobering reality for those facing the disease. On the other hand, the healthcare industry *has provided* major improvements in terms of supportive care, which have collectively improved patient lives. In our view, there are several challenges that have contributed to the lack of clinical success, and we will cover some of those further in this report.

- **ALS Statistics**

From St. Louis University, (ALS By The Numbers : SLU - Saint Louis University):

- *Every 90 minutes, someone is diagnosed with the disease and someone passes away from the disease.*
- *Most people who develop ALS are between the ages of 40 and 70, with an average age of 55 at the time of diagnosis. However, cases of the disease do occur in people in their twenties and thirties.*
- *ALS is 20 percent more common in men than in women. However, with increasing age, the incidence of ALS is more equal between men and women.*
- *About 90 percent of ALS cases occur without a family history, which is known as sporadic ALS. The remaining estimated 10 percent of ALS cases are inherited through gene disorders, known as familial ALS.*
- *ALS occurs worldwide with no racial, ethnic, or socioeconomic boundaries.*
- *It affects as many as 30,000 individuals in the U.S., with 5,000 new cases diagnosed each year.*

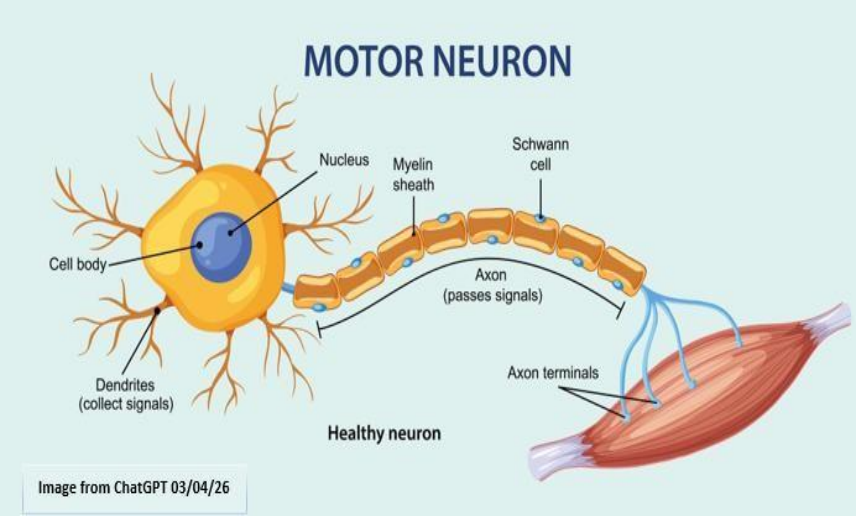
- *Estimates suggest that ALS is responsible for as many as five of every 100,000 deaths in people aged 20 or older.*
- *The incidence of ALS is five times higher than Huntington's disease and about equal to multiple sclerosis.*
- *The average life expectancy of a person with ALS is about 2-5 years from the time of onset of symptoms.*
- *Military veterans are up to twice as likely to be diagnosed with the disease as the general public. The reasons are unknown.*
- *More than half of all ALS patients live more than three years after diagnosis; 20 percent live five years or more, up to 10 percent live more than 10 years, and about five percent live 20 years or more.*
- *ALS does not affect mental ability. Although, some patients may have frontotemporal dementia affecting executive functioning.*
- *ALS patients burn calories faster than non-sufferers and, as a result, are often underweight.*

- **ALS Pathology**

As a preface to an overview of the pathology of ALS, we would reiterate something we noted above, *"…since Chalcot's discoveries, our overall understanding of the disease now includes a much clearer picture of its biological and molecular characteristics, unfortunately, that understanding has resulted in only limited success in the development of treatments for the disease…".* To reiterate something else we noted above, we are generalist equity analysts, so describing the pathology of ALS in detail is far beyond our associated aptitudes. That is, we certainly do not pretend to have some insight into a problem that has been evading clinicians for decades. However, there are some elements of ALS pathology that we *are able* to understand and articulate, and we think that understanding, albeit rudimentary on a clinical level, is quite helpful in understanding how Neurizon's technology is approaching the disease, and why that might be successful, which is of course the goal of this report.

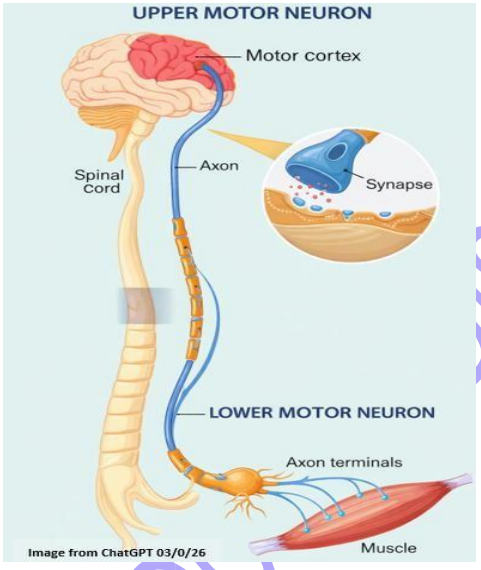
ALS appears to "start" in "motor neurons" from one of two but perhaps related areas of the body, the brain ("upper motor neurons") or the brain stem/spinal cord ("lower motor neurons").
Motor neurons are specialized nerve cells that control muscles. Motor neurons carry electrical signals from the nervous system to the muscles that move arms and legs but also control breathing, swallowing and other voluntary movements. Without properly functioning motor neurons and/or the network that supports them, muscle cannot do their jobs. **Table 1.** below illustrates that modality:

Table 1.



The human body's 1 million+ motor neurons are among the largest and most structurally demanding cells in the human body and the system that controls them is vast and complex. For instance, if you want to reach for a glass of water, neurons in the motor cortex of your brain generate signals that travel down the spinal cord. These signals ultimately activate motor neurons that control the muscles needed to perform the movement. Because reaching for a glass of water involves coordinated action of multiple muscles, such as those in the shoulder, arm, hand, and even the eyes, the nervous system coordinates many motor neurons simultaneously. In this pathway, an "upper" motor neuron in the brain connects to a "lower" motor neuron in the spinal cord. The signal is transmitted between them at a junction called a synapse. Table 2 provides an illustration of that relationship:

Table 2.



Motor neurons are much larger than other cells in the body. For instance, *a single motor neuron may extend from the spinal cord all the way to the foot, in which case its axon can be over 1 meter long. Further, a motor neuron that size could measure up to 1 million microns, which is 33,000 times the size of a skin cell (30 microns) and 125 times larger than a red blood cell (8 microns).* Among other things, because of their relatively large size, motor neurons create unique complexities and some of those complexities also make them particularly vulnerable, which in turn make ALS difficult to identify, treat and (hopefully someday) cure.

To reiterate, motor neurons are among the longest cells in the human body so maintaining such a long structure requires a continuous system for transporting proteins, energy-producing mitochondria, and other cellular components between the cell body and the distant end of the axon where the neuron attaches to the muscle. This process, known as "Axonal Transport", must operate reliably for decades in order to keep the neuron functioning. With that in mind, even relatively small disruptions to this transport system can have meaningful consequences for the health of the cell.

In addition to their structural complexity, motor neurons also have substantial energy requirements. These cells transmit information through electrical impulses, and each impulse requires the movement of charged ions across the neuronal membrane. After each signal is generated, energy-dependent pumps must restore the proper ion balance so that the neuron can fire again. Moreover, the neuron must supply energy not only to its cell body but also to the long axon and the neuromuscular junction where it communicates with muscle fibers. As a result, motor neurons rely heavily on efficient mitochondrial function and other cellular maintenance mechanisms to provide that considerable energy.

Finally, motor neurons differ from many other cells in the body in that they generally cannot be replaced once lost. Neurons do not readily divide in adulthood, and each motor neuron forms highly specific connections within neural circuits and with the muscle fibers it controls. Recreating these connections would require rebuilding extremely precise biological wiring that the adult nervous system has limited capacity to regenerate. Consequently, when motor neurons degenerate, as occurs in ALS, the body has little ability to restore them. As we will cover further in this report, processes such as "autophagy" play an important role in maintaining neuronal health over time, but when these systems fail the consequences can be catastrophic.

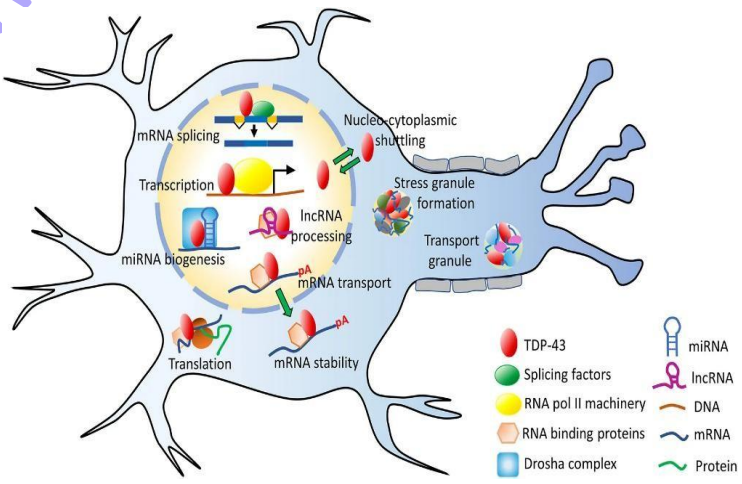
Taken together, the unusual length, high metabolic demand, the need for precise coordination across and within the motor system and limited regenerative capacity of motor neurons help explain why they represent a critical point of vulnerability in diseases that degrade that network.

While the above provides a high level view of the vulnerabilities that exist in the circuitry and pathways within the human neural network, a more granular understanding of the interworking of the actual cell body, may provide some additional clues about the origins and causes of ALS.

While the precise cause of ALS remains an active area of research, most investigators now believe the disease arises from a combination of interacting cellular stresses that ultimately impair the health and survival of motor neurons. At a high level, many of these mechanisms converge on disruptions in how neurons process RNA and maintain protein quality within the cell.

Under normal conditions, genetic information stored in DNA is transcribed into RNA, which acts as a working template for building proteins. This process requires careful regulation, particularly in neurons, which rely on thousands of RNA molecules to coordinate the production of proteins needed for signaling, transport, and cellular maintenance. A key regulator of this process is the protein TDP-43, which plays an important role in controlling RNA splicing, transport, and stability. In healthy neurons, TDP-43 is located primarily in the nucleus, where it helps ensure that RNA molecules are properly processed before they are exported to the rest of the cell. However, TDP-43 is also found outside the nucleus in the surrounding cytoplasm where it assists in RNA transport granules that move RNA through the neuron and also helps regulate when RNA is translated into proteins. Recognize, the nucleus of the cell body stores and transcribes information which is ultimately transported to the cytoplasm where the proteins get constructed. TDP-43 helps shuttle the RNA instructions to the cytoplasm, then, under normal conditions, it returns to the nucleus to again assist RNA processing, and ultimately (again) RNA transport back to the cytoplasm. **Table 3.** from Frontiers in Molecular Neuroscience (Frontiers | Molecular Mechanisms of TDP-43 Misfolding and Pathology in Amyotrophic Lateral Sclerosis) illustrates the process:

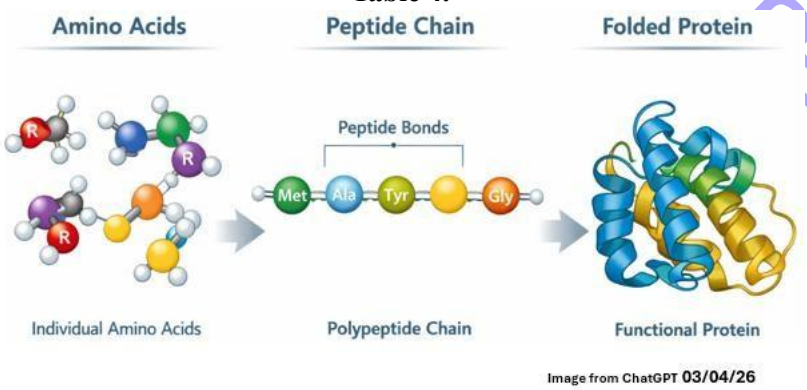
Table 3.



In many ALS cases, however, TDP-43 becomes "mislocalized" and *accumulates* in the cytoplasm rather than returning to the nucleus. When this occurs, two problems may arise simultaneously. First, the nucleus loses an important regulator of RNA processing, which can lead to errors in how RNA molecules are produced and managed. Second, the TDP-43 proteins that accumulate in the cytoplasm can begin to clump together and form "*aggregates*". These aggregates interfere with normal cellular processes and may disrupt the transport and translation of RNA needed to maintain neuronal function. While it is unclear what makes TPD-43 mislocalize and clump in the cytoplasm, **the phenomenon occurs in roughly 95% to 97% of patients with ALS**, making it perhaps the telling connection amongst them.

Compounding the problem is the phenomenon of protein misfolding. **Table 4**. illustrates the process by which proteins are folded and by extension uniquely formed.

Table 4.



Briefly, amino acids are bound together into specific peptide chains which are then folded in a particular way to create a specific protein. The body creates/contains thousands of proteins, each with a specific function. TDP-43 is one of those proteins and again, its function is to assist in the binding and regulation of RNA.

Again, proteins must be folded into *precise* three-dimensional shapes in order to function correctly. Various cellular stresses, including oxidative stress, genetic mutations, or errors in RNA processing, can cause proteins to fold incorrectly. Misfolded proteins sometimes stick to one another and form aggregates that can disrupt and/or clog the internal machinery of the neuron.

At the same time, the body sometimes also forms "stress granules". These are referenced in **Table 3** above. Stress granules are a temporary structure that forms in the cytoplasm of a cell when the cell experiences stress. Their primary function is to pause and reorganize protein production so the cell can focus on surviving the stressful condition.

Under normal circumstances, messenger RNA (mRNA) molecules are continuously being translated into proteins by ribosomes. When a cell is exposed to stress, such as oxidative stress, heat shock, or disruptions in energy supply, the cell temporarily slows or stops this translation process. The unused mRNA molecules and several RNA-binding proteins are then gathered into stress granules. This process is a sort of safety valve or a breaker, that protects mRNA from degradation while the cell is under stress by temporarily halting protein synthesis, conserving cellular energy. Importantly, stress granules are normally reversible structures. Once the stressful condition passes, they typically dissolve and the stored RNA returns to normal translation.

However, in certain neurodegenerative conditions, including ALS, stress granules may persist longer than intended. Proteins that regulate RNA, including TDP-43, can accumulate within these structures, and over time this accumulation may contribute to the formation of abnormal protein aggregates that interfere with normal cellular function. In short, stress granules are normally protective as a temporary cellular response to stress, but when they fail to dissolve properly, they may aggregate and contribute to the cellular dysfunction observed in diseases such as ALS. Further, as we noted, misfolded proteins may carry the same negative implications, and *they* sometimes combine within aggregated stress granules.

Under normal circumstances, cells possess systems designed to identify and remove damaged or misfolded proteins. One of the most important of these systems is "autophagy", which acts as a form of intracellular waste removal. Through autophagy and related protein-quality control pathways, cells attempt to isolate and break down harmful protein aggregates (misfolded proteins and stress granule aggregates for instance) before they accumulate. However, in ALS it appears that the buildup of abnormal proteins, including aggregates containing TDP-43, can overwhelm these cellular cleanup systems. When the capacity of the neuron to remove damaged proteins is exceeded, aggregates may accumulate within the cell, disrupting normal functions such as RNA processing, protein transport, and energy production. Over time, these stresses can impair the neuron's ability to maintain its long axon and synaptic connections with muscle, ultimately leading to degeneration of the motor neuron itself. That brings us to research.

- ALS Research and Treatment

Over the past several decades, researchers have proposed a number of theories regarding the biological mechanisms that may contribute to ALS. While these theories have evolved as scientific understanding has improved, it is fair to say that the precise cause of the disease remains incompletely understood. As a result, ALS research has historically pursued several different biological pathways, many of which have produced promising hypotheses but ultimately limited clinical success.

Earlier research focused heavily on the concept of excitotoxicity, which proposed that excessive stimulation of neurons by the neurotransmitter glutamate could damage motor neurons over time. This theory led to the development of the drug Riluzole, which modestly reduces glutamate signaling and remains one of the few therapies approved for ALS. While Riluzole demonstrated that modifying neurotransmitter activity could modestly slow disease progression, the clinical benefits proved limited and suggested that excitotoxicity alone was unlikely to explain the broader disease process.

Subsequent research expanded to include several additional mechanisms that may contribute to motor neuron degeneration. These include oxidative stress, mitochondrial dysfunction, impaired axonal transport, and disruptions in the cellular systems responsible for clearing damaged proteins. Many experimental therapies targeting these pathways entered clinical trials over the past two decades. However, despite encouraging results in laboratory models, most of these therapies failed to demonstrate meaningful clinical benefit in human trials. These setbacks have led many researchers to conclude that ALS likely arises from a convergence of multiple cellular stresses rather than a single dominant mechanism.

More recently, scientific attention has increasingly focused on abnormalities in RNA regulation and protein homeostasis within neurons. One of the most consistent pathological findings in ALS involves the mislocalization and aggregation of TDP-43, we discussed above. This discovery has contributed to a growing view that disruptions in RNA metabolism, protein folding, and cellular cleanup systems such as autophagy may play a central role in the disease.

While these insights have helped refine the scientific framework for understanding ALS, translating this knowledge into effective therapies has proven challenging. Numerous drug candidates targeting oxidative stress, inflammation, mitochondrial dysfunction, and protein aggregation have entered clinical development

only to fail in late-stage trials. These outcomes reflect both the complexity of the disease, and the difficulty of intervening once substantial motor neuron degeneration has already occurred.

Taken together, the history of ALS research illustrates both the persistence of investigators and frankly various advocacy groups, seeking to understand the disease and the considerable challenges involved in developing effective treatments. Although many early hypotheses proved incomplete, each wave of research has contributed incremental insight into the biological processes that may ultimately drive motor neuron degeneration. The emerging emphasis on RNA regulation, protein aggregation, and cellular stress responses represents the most recent step in that evolving scientific framework.

As we noted, today, there are only three approved drugs available for ALS patients and they include, Riluzole, Edaravone and Tofersen, which collectively provide relatively nominal support. That said, we believe Neurizon is on a path that may address some of the known pathology of ALS, specifically the clearing of TDP-43 and other harmful aggregates, as well as perhaps some of the challenges associated with applicable clinical trials, which brings us to our next major observation.

From another perspective, the development of therapies for ALS has historically been complicated by several practical and scientific challenges associated with clinical trials. While the disease has been the subject of significant academic and industry research over the past several decades, translating promising biological hypotheses into effective therapies has proven difficult in part because of the way ALS progresses and the relatively small patient population available for study.

One of the most immediate challenges relates to patient enrollment. As some of our subscribers may recognize, we have provided some prior research on the growing difficulties faced by the clinical trial industry and specifically enrollment. We think it is fair to say that those problems are more acute in ALS research than in perhaps many other diseases. ALS is considered a rare disease, with a relatively limited number of diagnosed patients at any given time. Moreover, the disease progresses rapidly, which means patients often enter clinical trials at different stages of progression. This variability can complicate the interpretation of trial outcomes, as patients with similar diagnoses may experience meaningfully different rates of decline. As a result, assembling patient cohorts that are both large enough and sufficiently similar to produce statistically meaningful results can be difficult.

A second challenge involves the selection of appropriate clinical endpoints. Because ALS primarily affects motor function, many trials rely on functional rating scales that attempt to measure changes in a patient's ability to perform everyday activities such as walking, speaking, swallowing, and breathing. The most widely used measure is the ALS Functional Rating Scale, which tracks functional decline across several domains. While this tool provides a standardized method for evaluating disease progression, the rate at which patients decline can vary considerably, which can make it difficult to detect modest treatment effects within the time frame of a clinical trial.

In addition to these challenges, ALS trials often face difficulties related to disease heterogeneity. The condition may arise from several different biological mechanisms, including genetic mutations and disruptions in cellular processes such as RNA regulation and protein homeostasis involving proteins like TDP-43. Because these mechanisms may differ among patients, a therapy that targets one pathway may benefit only a subset of the overall ALS population. Identifying the appropriate patient population for a given therapy therefore represents another important consideration in trial design. We alluded to this above with respect to the current drug Tofersen, which has "exhibited efficacy in a subset (SOD1) of the Familial ALS population", **which represents only 1%- 2% of the overall ALS population**, which helps illustrate the problem.

Taken together, these factors; limited patient populations, variability in disease progression, and challenges associated with selecting reliable clinical endpoints and others, have historically complicated the development of new therapies for ALS. As a result, researchers and regulators have increasingly explored alternative trial designs and biomarkers that may allow investigators to detect therapeutic signals more efficiently in future studies.

Lastly, as we will cover briefly in the Technology Overview below, there may be other neurodegenerative diseases related to TDP-43 folding or other associated mechanisms that Neurizon's technology may be applicable to.

Technology Overview

Neurizon's technology is referred to as "NUZ-001" and it appears to influence signaling pathways associated with autophagy, the process by which cells identify and remove damaged proteins and other cellular debris. By modulating components of the mTOR/p70S6K pathway, the therapy is believed to enhance the cells' natural ability to clear toxic protein aggregates that can accumulate within neurons. In the context of ALS, this is particularly relevant because abnormal aggregation of proteins such as TDP-43 is observed in the majority of patients.

That said, the underlying concept behind NUZ-001 is not necessarily to target a single genetic mutation or isolated biological mechanism, but rather to improve the broader cellular environment within stressed neurons. If the therapy is able to enhance protein clearance and reduce cellular stress, it could theoretically slow the cascade of damage that ultimately leads to motor neuron loss. That is also why it may be relevant to other neurological diseases.

While the clinical effectiveness of NUZ-001 remains to be determined, the drug's mechanism reflects a growing interest in therapies that seek to restore fundamental cellular housekeeping processes. In that sense, NUZ-001 represents an attempt to address one of the underlying biological themes that appears to recur across many neurodegenerative diseases: the progressive inability of neurons to properly manage and dispose of damaged proteins.

Beyond the specifics of NUZ-001, as we illustrated in the **Industry Overview** above, there are a few characteristics of the ALS ecosystem (disease pathology, patient individualities, researchers, clinical trial processes and others), that make solving its challenges unique. We will reiterate some of the more cogent of those challenges below as a setup to Neurizon's technology, clinical path and prospects for FDA approval and commercial application.

1. *"…despite encouraging results in laboratory models, most of these therapies failed to demonstrate meaningful clinical benefit in human trials. These setbacks have led many researchers to conclude* ***that ALS likely arises from a convergence of multiple cellular stresses rather than a single dominant mechanism****…"* As we will illustrate, NUZ-001 involves a protein that is prevalent in nearly all ALS patients.

2. Many drugs (ostensibly 30% to 40%) fail in Phase 1 clinical trials because they demonstrate poor safety data that may not have presented in pre-clinal animal studies. NUZ-001 is a derivative of a drug that has been used for some time to safely treat animals, so it is a known quantity in that sense. As we will delineate, we believe the Company's clinical results have established the safety of NUZ001 in humans as well, which is the first big clinical hurdle.

3. Neurizon/NUZ-001, has been selected to participate in the HEALEY ALS Platform Trial. We will expand upon this below, but in our view and for various reasons, outside of successfully completing Phase 1 safety trials, this may be the single most important event in the Company's history. Absent this step, we would likely not be initiating this coverage. In our view, this is a watershed event for Neurizon.

4. In addition to the HEALEY Platform, the Company has been granted Orphan Drug status by the FDA and has achieved similar designations from other applicable international regulatory bodies. We think *demonstrated success* in HEALEY could result in additional FDA designations that could shorten their path to regulatory approval and commercial launch.

5. As noted, there may be other neurodegenerative diseases related to TDP-43 folding or other associated mechanisms that Neurizon's technology may be applicable to, and the Company's has commenced pre-clinical studies in at least one of those that we know of.

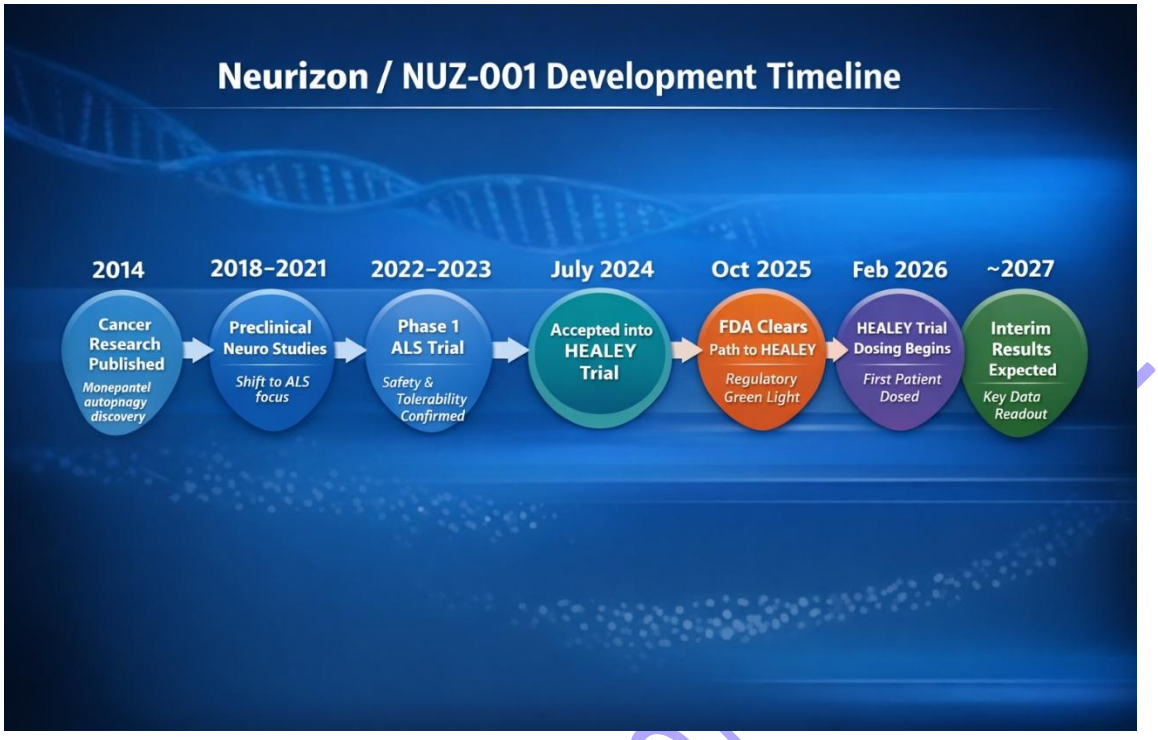
Neurizon's lead drug candidate NUZ-001 is more typically known as "Monepantel". Monepantel was developed in the early 2000's by Novartis Animal Health, which was acquired by Eli Lilly and Company (NYSE: LLY), and later spun out as Elanco Animal Health Incorporated (NYSE: ELAN). It was originally developed as a treatment for drug resistant parasitic worms in livestock. The molecule addresses "nematode nicotinic acetylcholine receptors", which are common to certain parasites, but are not found in mammals. That factor is important to the drug's favorable safety profile in humans. After years of clinical work around the potential repurposing of Monepantel for ALS, Neurizon acquired an exclusive licensing agreement with Elanco for the (potential) use of the drug in humans.

As a bit of history, it is important to note that prior to October 10, 2024, the Company was called PharmAust Limited and thereafter became Neurizon Therapeutics Limited. PharmAust's initial interest in Monepantel was aimed at repurposing the drug to address (specifically) human ovarian cancer cells. Those efforts included initial preclinical trials during which they discovered that Monepantel (referred to as "MPL") induced autography. Here is a short excerpt from a (2014) study we believe the Company collaborated in (Monepantel induces autophagy in human ovarian cancer cells through disruption of the mTOR/p70S6K signalling pathway - PubMed),

> "*…accumulation of acidic vacuoles with distinct chromatin morphology and an increase in punctuate localization of green fluorescent protein-LC3B, and MPL-induced changes in the expression of SQSTM1/p62 were all indicative of MPL-induced autophagy. Consistent with this, we found inhibition of mTOR phosphorylation leading to suppression of the mTOR/p70S6K signalling pathway. Our findings provide the first evidence to show that MPL triggers autophagy through the deactivation of mTOR/p70S6K signalling pathway".*

We believe this discovery, in conjunction with the researchers' apparent sense of the role that autophagy inducement might play in ALS, led them to switch gears and pursue the current path. That path has since included preclinical and Phase 1 clinical trials and an Open Label Extension study that demonstrated that the drug was well tolerated (no serious adverse event) and suitable for chronic dosing, as well as promising initial efficacy potential. That success has led to the current clinical trial which is being conducted on the HEALEY ALS Platform. **Table 5**. provides a timeline of the Company's progress to the point. We would add, as of 12/31/25, the Company reflected an Accumulated Deficit of just under AUD $83 million, so they have spent considerable resources getting to this point.

Table 5.



To reiterate something we alluded to above**, setting aside their collective success in advancing the drug to the current trial status**, we believe the Company's acceptance onto the HEALEY ALS Platform Trial is perhaps the Company's most notable milestone to date. Here is some high level information regarding the platform that we think helps validate that view.

The HEALEY ALS Platform was started by Sean M. Healey, who was a Managing Director of Advent International, a large private equity investment firm located in Boston, Massachusetts. Mr. Healey was diagnosed with ALS in 2016 at the age of 45, and along with his wife Caroline, became advocates for the acceleration of ALS research and development, specifically clinical trials, through collaboration with physicians and researchers at Massachusetts General Hospital. As a result, today Massachusetts General Hospital (which is also affiliated with Harvard Medical School) is one of the leading ALS research facilities in the world, especially with respect to clinical trial innovation.
Unfortunately, Mr. Healey passed away in 2017, but not before establishing the Healey ALS Platform Trial, which subsequent to his death, his family continued to build and support. That said, here is a brief overview of the platform.

The HEALEY ALS Platform Trial is a relatively new approach to clinical drug development designed to accelerate the testing of potential treatments for ALS. Historically, ALS drug development has faced a number of structural challenges. The disease affects a relatively small patient population, and individual clinical trials have traditionally required large numbers of participants, separate infrastructure, and lengthy recruitment periods. As a result, many promising therapies have taken years to evaluate, while others have struggled to advance simply because the companies developing them lacked the financial resources to run large standalone trials.

The idea behind HEALEY was to fundamentally rethink how ALS trials are conducted. Rather than each company running an independent study, the platform allows multiple experimental drugs to be tested simultaneously within a single master clinical trial. Participating therapies share a common network of

clinical sites (70 ALS centers across the nation), centralized data systems, and a unified statistical framework. Perhaps most importantly, the platform also uses a shared placebo group, which significantly reduces the number of patients required for each individual study arm and allows a greater proportion of participants to receive an experimental therapy.

The trial also incorporates an adaptive design. This means that therapies showing little evidence of benefit can be removed from the study relatively quickly, while promising treatments can expand enrollment and transition toward later-stage development. New therapies can be added to the platform over time without having to build an entirely new trial from scratch.

Participation in the HEALEY ALS Platform Trial offers several important advantages, particularly for smaller biotechnology companies (Neurizon) developing new therapies. The most immediate benefit is access to an established clinical trial infrastructure. Rather than building a full trial network from scratch, participating companies can use the existing system of ALS clinical sites, investigators, patient registries, and data management already established through the HEALEY platform. This significantly reduces both the cost and the logistical complexity of launching a clinical trial.

As we alluded to above, another key advantage is the use of a shared placebo group. Because multiple experimental drugs are evaluated within the same master protocol, all participating therapies share a common placebo arm. This reduces the total number of patients required for each study and increases the likelihood that participants will receive an active therapy. For patients with a rapidly progressing disease like ALS, this improves recruitment and helps trials enroll more quickly.

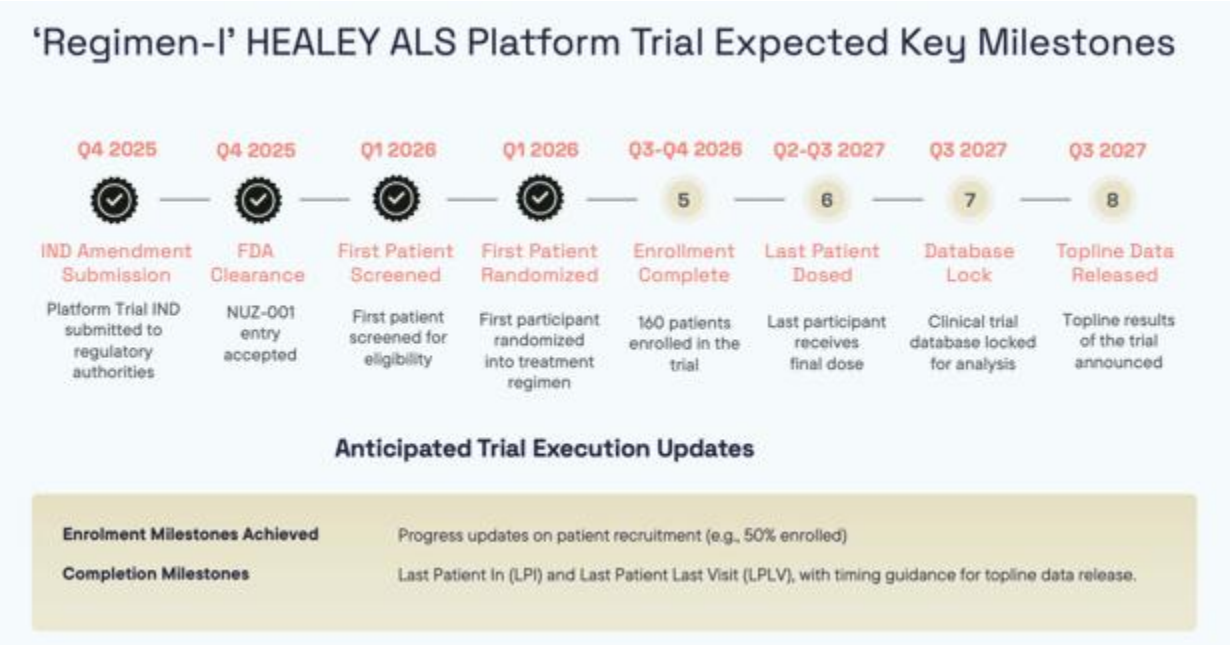
The platform's adaptive design also makes it more efficient than traditional clinical trials. Treatments that show little evidence of benefit can be removed from the study relatively early, while therapies that demonstrate promising signals can expand enrollment or move more rapidly toward later-stage development. This flexibility reduces the time required to identify whether a therapy is worth pursuing further.

For smaller biotechnology companies, these features can dramatically lower development costs. Running a standalone Phase 2 or Phase 3 neurological trial can cost tens of millions of dollars and take years to organize. By leveraging the HEALEY platform, companies can evaluate their therapies using an existing clinical infrastructure and a streamlined trial design, allowing them to reach meaningful clinical data more quickly and with substantially less capital.

Succinctly, the HEALEY platform allows multiple companies to evaluate new ALS therapies simultaneously, accelerates the pace of research, and helps promising treatments reach patients faster than would be possible under the traditional one-drug, one-trial development model.

To reiterate, Neurizon's acceptance into the HEALEY trial is in our view a validation of their prior progress, as we do not believe they would have been accepted into the trial had that work failed to demonstrate the potential to ultimately become a viable treatment for ALS patients. From another perspective, one of the major advantages of HEALEY is to more quickly identify therapies that are not demonstrating meaningful efficacy, so time and resources are not wasted on an ineffective candidate. Moreover, that "fail fast" approach allows other potentially promising solutions to replace those ineffective candidates and get into the clinic more quickly. To that end, we believe the platform's first 3 drug candidates commenced trials in mid-2020. Since that time 7 or 8 have participated in total, of which 2 have shown enough promise to move toward Phase 3 trials. That said, **Table 6** below was excerpted from one of Neurizon's recent presentations, and it reflects their expectations with respect to the timeline and/or *anticipated* milestones of their HEALEY trial. To clarify, the trial is dubbed "Regimen-I" and it is a Phase 2/3 trail.

Table 6.



In our view, the "map" from **Table 6** above is a relatively tight window for expectations/milestones among most of the companies we have followed and/or ultimately covered. Further, keeping in mind the "fail fast" nature of the platform, as we understand it, unlike many clinical trials HEALEY trials do not typically provide for the release of preliminary data, but they do often include "futility reviews", which serve as a basis of identifying (early) therapies that are ostensibly failing. We believe the exact schedule varies by trial and at pre-specified thresholds as determined in the protocol. That being the case, while we would prefer to be able to see some preliminary data along the way, in this case, no news may be good news. Regardless, as we look at the timeline above (recognizing that the first patient in the trial was dosed a few weeks ago), we would expect them to have some top line data on initial patients before year end (calendar 2026). Again, no news on that front will be good news, because any announced news would likely reflect poor initial data and by extension termination of the trial.

Switching gears, one of the advantages of developing therapies for diseases with relatively small patient populations, especially those for which there are few if any available treatments, is that regulatory pathway is often more accommodating. That is, when a disease affects a limited number of patients but carries a high degree of medical urgency and few available treatment options, regulators have historically shown a willingness to provide pathways intended to accelerate the development and review of potential therapies.

One of the most important of these mechanisms is Orphan Drug designation, which applies to diseases affecting fewer than 200,000 patients in the United States. This designation can provide several meaningful incentives for drug developers, including tax credits for certain clinical trial expenses, waiver of certain FDA fees, and perhaps most importantly, seven years of market exclusivity following approval. That exclusivity can be particularly valuable because it protects the drug from direct competition even if the underlying compound itself does not enjoy strong patent protection. To date, this is the one designation Neurizon has been provided by the FDA, and it has been granted similar designations by other equivalent international regulatory bodies. However, as we will delineate below, there are other accelerated FDA pathways that the Company could potentially be granted if the current trial reflects favorable ongoing data. Companies developing therapies for serious conditions with limited treatment options may also qualify for the FDA's **Fast Track designation**. This program allows for more frequent interaction between the

company and the FDA during the development process and permits what is known as a rolling review of the regulatory filing, meaning portions of the application can be submitted and reviewed before the entire submission is complete. The practical effect is that the regulatory review process can begin earlier and potentially proceed more quickly.

Another pathway that can be particularly relevant is **Breakthrough Therapy designation**, which is granted when early clinical evidence suggests that a therapy may offer a substantial improvement over existing treatments. Drugs that receive this designation benefit from more intensive guidance from the FDA and often experience a more streamlined development process as regulators work closely with sponsors to design efficient clinical trials.

In addition, the FDA may grant **Accelerated Approval** for therapies targeting serious or life-threatening diseases based on surrogate endpoints that are reasonably likely to predict clinical benefit. In neurodegenerative diseases such as ALS, this could include measures of functional decline or biomarker data rather than waiting for longer-term outcomes such as survival. While companies receiving accelerated approval are typically required to conduct confirmatory trials after approval, the pathway can allow promising therapies to reach patients sooner.

Finally, patients suffering from serious diseases with limited treatment options may gain access to investigational therapies through the FDA's **Expanded Access Program**, often referred to as "**compassionate use"**. This program allows physicians to request access to experimental drugs for patients who are not eligible for clinical trials when the potential benefits are believed to outweigh the risks.

Taken together, these regulatory pathways reflect a broader effort by the FDA to encourage the development of therapies for rare but serious diseases that might otherwise be overlooked. For biotechnology companies operating in areas such as ALS, these programs can shorten development timelines, reduce certain regulatory costs, and provide meaningful commercial protections if a therapy ultimately reaches the market.

Lastly, as we noted earlier, the primary therapeutic rationale behind NUZ-001 centers on its apparent ability to influence cellular pathways associated with protein clearance, particularly through modulation of the mTOR signaling pathway and the activation of autophagy. Generally speaking, impaired autophagy and the accumulation of misfolded proteins are believed to play a central role across several neurodegenerative diseases including ALS. As a result, while Neurizon Therapeutics is currently focused on ALS, it is worth recognizing that the biological mechanism being targeted may have relevance across a broader set of neurological conditions.

More specifically, several neurodegenerative diseases are characterized by the abnormal accumulation of toxic protein aggregates within neurons. In ALS, this pathology most commonly involves the mislocalization and aggregation of the TDP-43 protein, which is observed in the vast majority of cases. However, other disorders exhibit similar patterns of protein accumulation, albeit involving different proteins. For instance, Alzheimer's disease is associated with the buildup of beta-amyloid plaques and tau tangles, Parkinson's disease involves the aggregation of alpha-synuclein, and Huntington's disease is characterized by mutant huntingtin protein accumulation. While the specific proteins differ, the underlying challenge, the inability of neurons to effectively clear damaged or misfolded proteins, appears to be a recurring theme across these conditions.

That said, therapies capable of enhancing autophagic processes have increasingly attracted interest as a potential strategy for addressing these diseases at a more fundamental level. If a therapy can restore or enhance the cell's ability to remove toxic protein aggregates, it could theoretically slow the progressive neuronal damage that ultimately drives disease symptoms. In this context, the ability of NUZ-001 to influence pathways linked to autophagy has prompted researchers to consider whether the compound might

have broader applicability beyond ALS. To that end, we believe Neurizon is in various stages of pre-clinical research in Huntington's Disease, Alzheimer's Disease (specifically Frontotemporal Dementia-"FTD", Parkinson's Disease, and potentially others). Again, our expectation is that positive results from the HEALEY Regimen-I trial could perhaps accelerate the Company's work in some of these other diseases, providing additional potential valuation legs to the story.

Lastly, while this may be a bit conceptual, as we understand it, the Company currently has patients from the Phase 1 trial who have been taking NUZ-001 for over 3 years. That may demonstrate its safety as a potential chronic therapy. That said, again conceptually, if NUZ-001 proves effective in upregulating autophagy, it could potentially slow the progression of the disease much earlier if diagnostic advances could enhance earlier detection. That type of scenario could expand the addressable market of the drug.

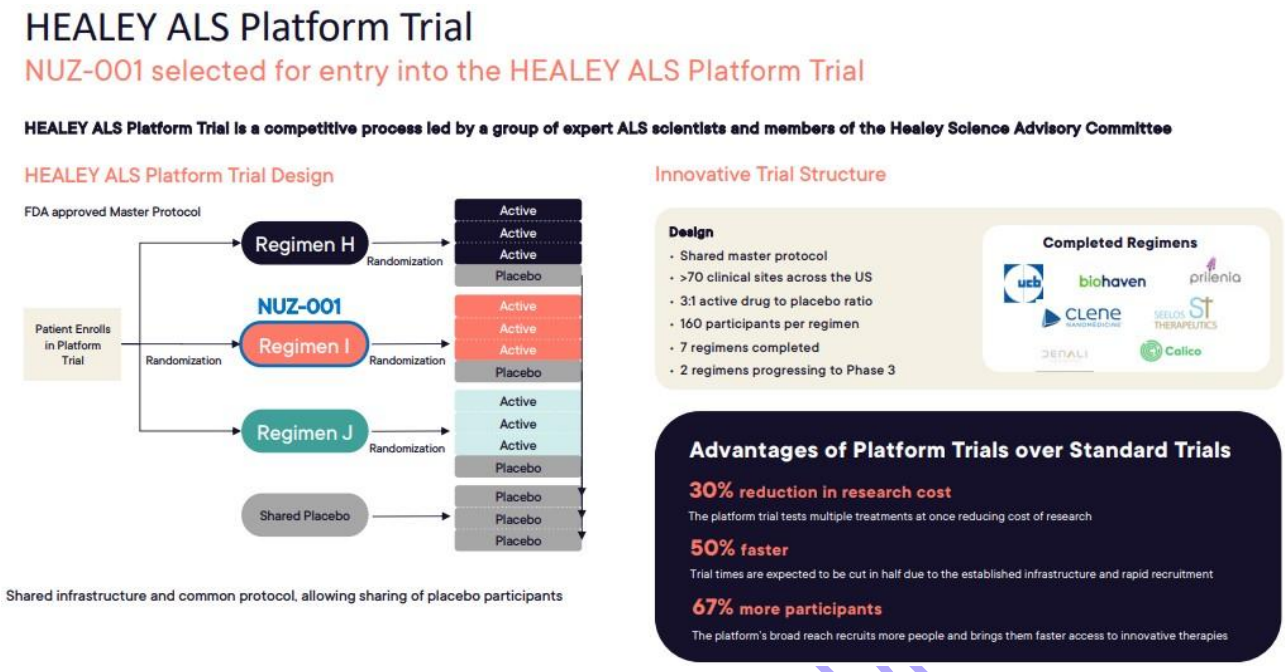
Operating/Clinical Overview

Clearly, an Operating Overview for a pre-revenue company is less topical than for those generating revenues. Specifically, this analysis hinges more on attempting to estimate the cash required to get to commercialization, or some other liquidity event that rewards shareholders for the work done along the way. As an extension, that includes (and perhaps most importantly) estimating those capital needs in the context of the equity dilution that will almost certainly accompany it, and that assume the technology proves viable. **To that end, everyone should recognize the likely outcome if the technology does not do well in the clinic.**

The above noted, here is our sense of the capital requirements going forward, which we submit, is subject to adjustment.

First, the Company has provided guidance around what they believe their expenses will be through the HEALEY trial period, but before we get to that, we will provide a bit more detail regarding the trial itself that may be helpful. Specifically, the trial is a randomized, double-blind, placebo-controlled study, which means that patients are randomly assigned to receive either the investigational drug or placebo. "Double blind" refers to the fact that neither the patients nor the physicians/investigators know who is receiving the active drug and who is receiving placebo, while an independent monitoring group ("the Data Safety Monitoring Board") has access to the unblinded data. Obviously, the treatment group is compared against a placebo control group, which allows researchers to measure whether the therapy is providing benefit vs. the placebo. Importantly, in the HEALEY platform that placebo group is shared across the other drug regimens in the trial, which is one of the design features that reduces the number of placebo patients required. For instance, Neurizon's trial includes a 3:1 randomization ratio, which means 75% of the enrolled patients receive an investigational therapy, while the others 25% receive a placebo. That high ratio helps enrollment, as it increases the likelihood that patients receive an active therapy (versus a typical 1:1 ratio, where 50% receive an active therapy). To that end, while the first patient was enrolled a few weeks ago (02/25/26), our understanding is that early enrollment has outpaced their expectations. Neurion's/NUZ001's arm is called "Regiment I" and it is designed to enroll 160 patients in over 70 clinical sites across the U.S, and the treatment will be administered and then evaluated over a 36 week period. **Table 7** below is the Company's illustration of the trial design which we think is helpful.

Table 7.



To be clear, Regiment H and Regimen J involve other drugs to be tested on the platform under the exact same protocol: double blinded, 3:1 randomization, 160 participants each, conducted at 70 clinics across the U.S. and administered and evaluated over at 36 week period. Also, the platform was intentionally designed to test drugs that target **different biological pathways involved in ALS**, rather than multiple drugs that all do roughly the same thing. As **Table 7** also reflects, and to our point regarding the advantages of the HEALEY platform, this approach provides for marked reductions in typical research costs (30%), much faster completion (50%) and more robust enrollment (67%). That brings us to Neurizon's guidance on outlays and capital.

Table 8 provides some of their guidance (again in AUD$) around both expected sources and uses of capital through the horizon of the completion of the current Phase II trial period.

Table 8.

Sources & Use of Funds

Neurizon is focused on efficiently delivering an outcome from the HEALEY ALS Platform Trial, including significantly reducing spend on non-core costs. The below table, excluding any funds raised from the Entitlement Offer, shows the sources and uses of funds to the expected topline readout[1].

Sources of Funds	**(A$m)**
Existing Cash	3.0
Placement	7.3
Research & Development Tax Rebate[2]	22.6
Obsidian Convertible Note Facility[3]	20.0
TOTAL Sources of Funds (A$)	**52.9**
Use of funds[1, 3]	**(A$m)**
Research & development:	
1. HEALEY ALS Platform Trial	35.8
2. Other R&D – Other Clinical, Preclinical, Clinical Manufacturing	6.6
Other Spend:	
1. Regulatory & Commercial Preparation	4.6
2. Governance, Finance, Other Working Capital	4.6
Expenses of the Offer[4]	1.3
TOTAL Uses of Funds (A$)	**52.9**

[1] See also risk titled 'Future funding risk' on slide 42.
[2] The amounts reported as R&D Tax Rebate include both the FY2025 R&D receivable (net of financing) as well as forecast R&D receipts (inclusive of potential financing where appropriate to do so).
[3] Assumes full draw down under the Convertible Note Facility.
[4] The expenses of the Offer, includes costs associated with both the Offer and Convertible Note Facility (assuming a full draw down of its capacity) as well as an estimate of advisory fees.

We will unpack **Table 8** from the bottom up.

We believe the capital requirement of AUD$52.9 million reflects their projected needs through the expected top line trial results in Q3F27 (see **Table 6** above), or roughly the next 18 months. Our projected operating model reflects that guidance, and we have spread that spend over the relevant period in a relatively linear way. Obviously, it may be more variable from one period to the next than we are reflecting. Our charge will be to monitor it to try to determine if the original assumption ($52.9 million) remains reasonably accurate as we move forward.

The source of funds analysis has a few contingencies that require some attention. Over the past few months, the Company has engaged three separate funding vehicles.

1) In December (2025) the Company completed a private placement raising AUD$7.1 million at AUD$.08 through the sale of approximately 89 million shares.

2) Thereafter, but prior to the end of calendar 2025, the Company commenced an additional raise of up to $17.1 million also at $.08, which if fully subscribed, would add an additional 214 million shares. We believe they have raised approximately $6 million through this vehicle, and they are actively marketing the balance. They refer to this raise as a *"2 for 5 non-renounceable entitlement offer"* which means for every 5 shares an existing shareholder owns; they are entitled to buy 2 new shares. Thus, this is available to existing shareholders, so U.S. investors might recognize this as a "rights offering". As we understand it, this offering expires 90 days from the 21st of January, so April 21, 2026. While we suspect they are hopeful regarding the use of this offering to provide critical funding, they have established an additional contingency as well.

3) Prior to the calendar (2025) year end, the Company also announced a $20 million strategic convertible note facility with New York-based Obsidian Global GP, LLC. The terms of the agreement are available on Neurizon's website, and we are happy to provide that to readers as well. In short, the capital is available in tranches, with some size and timing limitations, and they executed on the first tranche ($5 million) which was stipulated in the agreement. We believe the first installment is convertible at $.165, with subsequent installment conversion prices at a 6% discount to a Volume-Weighted Average Price ("VWAP"). The Company can choose to make additional draws on the facility, but they are not obligated to do so if other more favorable funding opportunities arise. Obviously, this facility is a favorable element with respect to the typical risks faced by small biotechs with respect to access to the capital necessary to fund ongoing trials.

Lastly, in addition to the financing they have put in place, the Company also benefits from Research & Development tax rebates afforded them by the Australian government. Succinctly, the rebate provides Neurizon with a rebate of 48.5¢ for each $1.00 they spend on research and development, and in their case, that includes R&D outside of Australia (the HEALEY Trial for instance). So then, the Australian government will essentially pay for nearly half of the HEALY trial (as well as other current/future preclinical studies), which along with the capital initiatives noted above, the Company believes will be sufficient to get them through top line data from HEALEY. We believe the Company could also potentially benefit from other ALS related funding mechanisms available from government and other private ALS stakeholders (Accelerating Access to Critical Therapies for ALS Act – ACT for ALS | FDA).

We have integrated all of the above cash requirements, capital access and resulting share count information into our model attached below. We will make appropriate changes to our model as additional information dictates.

Management Overview

Mr. Sergio Duchini, Executive Chairman

Mr. Duchini brings over a decade of board-level experience with expertise spanning professional services, life sciences, biotechnology, banking, finance, and the not-for-profit sector. His extensive background includes roles such as Chair, Executive and Non-Executive Board Director, Risk & Audit Committee Chair, and Chief Strategy Officer.

Currently, Mr. Duchini serves as a Non-Executive Director and Chair of the Audit Committee at Enlitic Inc., a US company focused on leveraging artificial intelligence to enhance work/low and patient outcomes in radiology. Additionally, he holds the position of Chair at Lymphoma Australia, a leading not-for-profit organization supporting lymphoma patients and their caregivers in Australia.

Mr. Duchini has previously sat on the AusBiotech Board of Directors for nine years, where he played a pivotal role in the development of two national life science industry strategies. He also served as a Board Director at Deloitte Australia, overseeing the governance, strategy development, and stewardship of the partnership with annual revenues of $2.2 billion.

Mr. Duchini's executive experience includes 23 years at Deloitte Australia where he held multiple senior positions as an equity partner. He advised Australian and international groups to manage their investments in R&D nationally and internationally, developed and executed the national tax strategy, and led senior cross- disciplinary teams serving prominent clients such as National Australia Bank, ANZ Bank, and Australia Post.

Sharon Tamir, ALS Program Lead

Sharon is a highly experienced drug development leader with deep expertise in ALS, spanning early discovery through late-stage clinical development. She has led ALS programs at MT-Pharma, Karyopharm Therapeutics, and United Neuroscience, advancing innovative therapies toward regulatory milestones and registration. Sharon has a strong track record in regulatory engagement, patient-centered trial design, and modern clinical execution—including digital health and real-world data integration.

Dan O'Connell, Chief Finance Officer

Dan has over 20 years of experience working in multinational companies, with extensive experience in accounting and finance, research and development, M&A, procurement, shared services, investor relations and communications, government and industry relations, and tax. Dan was CFO of Kingsgate Consolidated, Interim CFO of Newcrest Mining, and has held other senior finance and commercial positions at Newcrest Mining, BHP, and Ernst & Young.

Dr. Jeffrey M. Brown, Chief Scientific Advisor

Dr. Brown brings over two decades of drug development experience, scientific and commercial leadership, in the development of new treatments for neuropsychiatric and neurodegenerative diseases. He has overseen multiple neurology programs from early discovery through IND-enabling studies, including Huntington's disease, currently in clinical trials. He held executive roles in global biopharmaceutical companies such as Amgen, Pfizer, BMS, Alexion, Wave, Voyager, and Deep Genomics.

Mr. John Clark, Chief Operating Officer

John Clark joined Neurizon with over 20 years of pharmaceutical industry experience in phase I – IV clinical trials across numerous therapeutic areas and multiple geographical regions. Most recently, John served as Senior Project Manager at a Global CRO, leading the Clinical Operations team and providing cross-functional oversight on a national CNS trial. Before that, John held various clinical operations leadership roles responsible for implementing clinical programs.

John has a proven project management and stakeholder engagement record, with a thorough knowledge of ICH-GCP and regulatory requirements. John earned his B.Sc. in Biomedical Sciences from the University of the West of England.

Dr. Chris Freitag, Chief Medical Advisor

Dr. Freitag has 20 years of experience in the pharmaceutical industry with positions in companies including Hoffmann La Roche, Shire, and BTG, where he led global clinical development projects. He held the position of Chief Medical Officer at Dynacure and Azafaros, where he was responsible for medical and regulatory strategy, including clinical development of a rare diseases compound. Dr. Freitag was the Medical Monitor on NUZ's Phase 1 MEND study and oversaw medical and clinical activities.

Lidija Damjanovic, Head of Marketing & Corporate Affairs

Lidija Damjanovic brings over 15 years of global experience and passion for launching pharmaceutical products and leading corporate communications and affairs in the biotech and pharmaceutical industry. Her extensive leadership experience in driving commercial growth strategies includes marketing and communications roles in companies such as Merck Sharpe and Dohme, Sartorius, Patheon and other global organizations.

Dr. Herbert Brinkman, Head of Manufacturing

Dr .Herbert Brinkman, based in Denver, Colorado, has over 30 years of experience in the pharmaceutical industry. Dr. Brinkman has prepared over 25 Chemistry Manufacturing and Control (CMC) sections and updates for multiple lnvestigational New Drug (IND), New Drug Application (NDA), supplementary NDA (sNDA), lnvestigational Medicinal Product Dossier (IMPDJ, and Abbreviated NDA <ANDA) filings for United States Food and Drug Administration (FDA) and European regulatory agencies.

Dr. Brinkman has filed and commercially launched nine products encompassing oncology, metabolic, dermatology, and endocrinology therapeutic areas and contributed to filing 21ANDAs for various semisolid and parenteral products. He is also an inventor on 14 patents. His expertise includes current Good Manufacturing Practice (cGMP) systems applied to API manufacture/ Drug Product manufacture and addressing regulatory issues. Dr Brinkman's previous position was Executive Director of Product Development at NASDAQ-listed company Arcutis Biotherapeutics, Inc. (NASDAQ: AROT), where he was responsible for the successful commercial launch of ZORYVE (roflumilast).

Dr. Martin Engel, Scientific and Medical Partnerships Lead

Dr. Martin Engel is an experienced strategic lifescience partnership manager with strong expertise in neuropharmacology. In his role as Medical & Scientific Partnerships Lead at Neurizon, he drives global engagement with pharmaceutical partners, supporting scientific due diligence, shaping program

positioning, and providing input to clinical and pre-clinical development planning aligned with partner expectations. Martin brings a proven ability to work across clinical, pre-clinical, and commercial teams, with a track record of building trusted external relationships across US, EU and APAC, and representing organisations in scientific and industry forums; he holds a PhD in neuropharmacology and has published widely on treatment development across central nervous system disorders.

Risks and Caveats

Small biopharma companies are among the riskiest enterprises in the investment universe. For the thousands of compounds that start in the development stage, only 200 or 300 advance to the pre-clinical stage, of which less than 5% make it to the clinical stage and maybe a small handful of those get to an FDA approval. This industry subset is not for the risk averse.

As we have attempted to delineate Neurizon has had promising results in their animal studies as well as in initial Phase 1 human results. However, successful pre-clinical animal studies do not always translate into successful human clinical results. On the contrary, the large majority of the time, they do not.

As we have attempted to delineate throughout this report, ALS is a very challenging disease. It is, in our view, perhaps the worst diagnosis a person can receive, part of which stems from the lack of available treatments, which circles back to the challenges associated with understanding its causes and its means. Consequently, the track record of clinical success is poor, which underscores the risks of each new clinical endeavor.

Clinical trials are expensive. For instance, while industry estimates of the costs required to get a drug through FDA approval vary widely (ranging from tens of millions to billions of dollars), it is not clear to us how much Neurizon will require if they continue to achieve clinical success and are able to move through all the required phases and into an NDA new drug application ("NDA"). As we noted above, Neurizon's acceptance onto the HEALEY platform was in our view a watershed event that, among other things, should substantially derisk this particular element of the business. Moreover, as we covered in the **Operating/Clinical Overview**, the Company has completed and/or engaged a handful of funding vehicles that have (and should) provide capital to fund the HEALEY trial. However, these funding arrangements have (and will) create considerable dilution. To that end, the Company believes their current funding arrangements will get them to top line data in HEALEY. That may prove inaccurate, in which case they may need to seek *additional* funding which they may or may not be able to do. On the other hand, additional financing will also likely be dilutive, and perhaps markedly so. Our model/targets include assumptions regarding the breadth of that dilution, but that endeavor involves poor visibility so those dilution assessments could prove considerably understated and by extension, or valuation assessments prove considerably overstated.

Small companies bringing new drugs to the market, face additional challenges beyond the clinical/approval process. While achieving an FDA approval is a major milestone for any company and its respective drug, an approval does not guarantee success in the marketplace. Fortunately, ALS still impacts a relatively small portion of the population, but that naturally limits the total addressable market ("TAM") and by extension, the potential economics of any new ALS treatment. Moreover, ALS appears to be driven by multiple overlapping biological mechanisms, rather than a single identifiable cause. These mechanisms may include protein aggregation (such as TDP-43 pathology), mitochondrial dysfunction, oxidative stress, neuroinflammation, and impaired cellular cleanup pathways such as autophagy. Because these processes interact with one another, the optimal solution may involve a combination of therapies, which could in effect divide the TAM amongst those providers. While everyone is encouraged by any/all medical advances that help improve and/or prolong people's lives, there are limits to the resource available to deliver those

solutions. These realities (treatment reimbursements for instance) impact the appropriate valuation of Neurizon *even if we assume favorable clinical results*. On the back side of that notion, failure in the current HEALEY trial will likely be considerably negative and perhaps catastrophic.

In conjunction with the prior observation, there are other enterprises aggressively trying to find treatments for ALS. Clinical success by others could negatively impact Neurizon. **Table 9** below provides some color to that end.

Table 9.

Major Public Companies with ALS Programs

Company	Ticker	Key ALS Program	Notes
Biogen	BIIB	Tofersen (Qalsody)	Approved for SOD1-ALS
Amylyx Pharmaceuticals	AMLX	AMX0035	Previously approved as Relyvrio but withdrawn after failed trial
Clene Inc.	CLNN	CNM-Au8	Tested in the HEALEY platform trial
Denali Therapeutics	DNLI	DNL343	Stress-response pathway drug
Prilenia Therapeutics	(private / planning IPO)	Pridopidine	Also tested in HEALEY
Neurizon Therapeutics	ASX: NUZ / OTC: NUZTF	NUZ-001	Currently in HEALEY Regimen I

Until recently, Neurizon's shares traded exclusively on the Australian Securities Exchange ("ASX"). On October 8, 2025, the Company began trading on the U.S. OTCQB Venture Market as well, under the ticker NUZTF. That noted, the shares have traded very little on the OTCQB since the listing, and in turn on a dollar volume basis, are *relatively* thinly traded on the AUX as well. Thinly traded shares are generally illiquid and are subject to considerable technical pricing volatility that may or may not correlate with the fundamental value of the Company. That illiquidity may continue into the foreseeable future.

As with many small companies, Neurizon relies on the collective contributions of a limited number of individuals. The Company's success may depend on their ability to retain these individuals. To that end, on March 16, 2026, the Company announced the resignation of its Managing Director and Chief Executive Officer Dr. Michael Thurn. The release notes that, Dr. Thurn will "*work constructively with the Board to implement an orderly leadership transition consistent with the Company's succession planning processes and remain available to support transition activities through his contractual notice period, which will conclude in July 2026. During this period, he will also continue to serve as a Non-Executive Director of the Company until the completion of his notice period*". We have a few observations about this development, which to be clear, came about as we were concluding our work around this document.

From a high level, having a CEO resign is generally disconcerting, because it begs the question, "why". Of course, the answer to that question typically defines the impact of the event. The Company's narrative around the event, (which included comments from Dr. Thurn), remains constructive. Moreover, our own correspondence with Dr. Thurn leads us to believe that the reasons behind the decision are not related to his enthusiasm for the likelihood of the technology's potential clinical success. We assume *that is the case*, and if in fact it is, we would view that as more important than whomever is at the helm if/when they are able demonstrate that efficacy. That said, given the recent initial enrollments onto HEALEY, we think it is fair to say that the trial is underway, and again, clinical success is the most paramount data point. Moreover, as we also noted above, the Company should have access to the capital required to complete the trial as well. That brings us to another, albeit conceptual notion around Dr. Thurn's resignation.

This is not the first small biotech we have seen replace founder or early stage personnel, especially when those individuals' aptitudes were suited to addressing the challenges of getting companies/drugs into the clinical process, and thereafter through some portion of the same (into Phase II for instance). Thereafter, the focus may require a different set of aptitudes that may be better addressed by others, and that may include experiences that can provide the relationships and access to capital that can potentially take the Company through the next clinical phase and into commercialization, or perhaps via another route, to a transaction with a large partner that can execute those later stages. To be clear, we do not know if that is the case here, although we suspect it may be at least part of it, but again, the transition provides some risks that may not exist were it not the case.

These are just some of the risks we have identified. There are likely others we have missed or are otherwise unidentifiable currently.

Summary and Conclusion

We have been providing generalist research in microcaps and/or early stage companies for nearly 30 years, and over that time we have evaluated several biotech companies and provided coverage on a small portion of those. Some were successful and some were not, but ALL of them exhibited the same high risk/high reward elements that are largely indicative of the group. Moreover, not only does this group typically share that high risk high reward characteristic, but more times than not, the outcome is quite binary; either the drug works, or it does not, and the success of the company is typically bolted to that outcome. To illuminate the distinction, these characteristics are unique to nearly all clinical stage drugs, but they are not unique to all drug *companies*. Large pharmaceutical companies with commercial products and a variety of drug candidates can and do survive clinical failures, small biotech's generally do not. That said, while the most obvious risk associated with any clinical endeavor is that the drug proves to be unsafe, inefficacious or both, small companies also face an additional set of risks that many of their larger entrenched "big pharma" counterparts do not. Two of the more notable of these are:

1) Access to the considerable capital required to get through clinical trials to get to the established point of "it works or it does not".
2) Ability to enroll a sufficient patient population that fits the inclusion criteria (and avoids the *exclusion* criteria) of the trial design. To be clear, sufficient enrollment is a challenge in many diseases, but it is especially problematic in ALS for several reasons which include a limited patient population (as a result of the relative rareness of the disease), considerable lack of homogeny across the patient population, generally short patient life spans, a high likelihood that the disease is caused by multiple interacting biological processes, and others. The complexity of the disease adds to the complexity of trial design, which further inhibits enrollment.

A company cannot achieve clinical success without first addressing these two hurdles, and they are BIG hurdles. Further, one does not guarantee the other, but they can each be tied to the failure of the other. That is, having abundant capital does not ensure enrollment, but a lack of capital will likely prohibit enrollment. More practically, even for well capitalized companies, slow enrollments extend trial periods, which adds to their costs and extends the time to commercialization of the successful drugs. To reiterate, clinical trials are expensive, but clinical trials with difficult enrollment challenges are even more expensive, on multiple levels. Neurizon's unique posture mitigates each of these risks, and frankly, that posture is what attracted us to the story initially, and it remains a cornerstone of our thesis. We addressed much of that throughout this report, but we will highlight it briefly again.

The Company's inclusion in and initiation of the HEALEY ALS Platform Trial was a watershed event for the Company, in part because it allowed them to address the two risks we just addressed. In our view, without HEALEY the Company's chances of raising enough capital to get through a clinical trial were very

small, in part because the capital required to do that would have been *several multiples* of the anticipated capex required for HEALEY Regimen I as reflected in **Table 8** above. Moreover, the chances of them enrolling a trial even if they were able to raise the capital were likely *even smaller*. We do not think that statement is particularly provocative considering that HEALEY was formed from the recognition of an ALS patient that treatment options were limited because clinical trials were too expensive and too difficult to enroll. Much of the HEALEY concept is focused on mitigating these two challenges. Again, The Company's inclusion in the HEALEY ALS Platform Trial was clearly transformational and to suggest that it derisked the Company is an understatement.

While we cannot overstate the importance of HEALEY, the Company still faces that more "obvious risk" we addressed above, which is, does NUZ-001 work? To that end, we view NUZ-001's inclusion in the platform as a validation of its clinical success *to this point*. That is, we are comfortable suggesting that those in charge of allowing candidates onto the platform are among the most informed individuals in the world with respect to ALS. To that end, we assume they add candidates they believe have demonstrated the potential for further clinical success. We are certainly not suggesting the inclusion means NUZ-001 will be successful. Afterall, multiple drugs on the platform have failed. However, as we addressed throughout this report, while ALS appears to arise from a variety of genetic and environmental triggers, the abnormal aggregation of TDP-43 is present in *95%+ of all ALS patients*. Further, the abnormal aggregation of these proteins is implicated in the progression of ALS, and perhaps in multiple ways. To that end, the Company's belief is that NUZ-001 may enhance a process known as autophagy, which is the body's natural defense against the abnormal aggregation of proteins including TDP-43. Autophagy rounds up these aggregated proteins and eliminates them, but it appears ALS may overwhelm the body's normal autophagy capabilities. Thus, if NUZ-001 can stimulate or boost the autophagy process, it may be able to slow the progression of ALS. Which brings us to our final point.

As we alluded to aside from TDP-43, autophagy eliminates other abnormal protein aggregations, which means it may be helpful in fighting other neurodegenerative diseases. The Company is currently engaged in pre-clinical studies to address that potential.

To summarize, perhaps against the odds, Neurizon has managed to advance NUZ-001 to a critical Phase II stage on what may be the world's most prominent ALS trial platform, and they have begun enrolling patients in the trial. Succinctly, it is game time, and the next 18 months should provide considerable clarity around the drugs ability (or lack thereof) to slow the progression of ALS. Our thesis is that given what we know about the early success of the drug, and in the context of some of the other risks they have been able to overcome, we think the current valuation of the shares reflects a compelling risk/reward profile. As a result, we are initiating coverage of Neurizon Therapeutics Limited with 12-24 month price target of USD$.32 and our typical initiating allocation of 4. We will revisit each as new data points emerge.

Project Operating Model

Neurizon Therapeutics Limited				
Projected Operating Model (AUD$)				
Projected Cash Position (AUD$)				
Prepared by: Trickle Research				
	Actual 6 Months 12/31/25	Estimate 6 Months 06/30/26	Estimate Fiscal 2026	Estimate Fiscal 2027
REVENUE				
Sales	$ -	$ -	$ -	$ -
Other Income (R&D Rebates)	$ 5,996,886	$ 7,282,857	$ 13,279,743	$ 7,958,869
Total Revenue	**$ 5,996,886**	**$ 7,282,857**	**$ 13,279,743**	**$ 7,958,869**
EXPENSES				
Research and Development	$ 8,242,201	$ 8,500,000	$ 16,742,201	$ 18,296,250
Administration	$ 1,688,310	$ 1,700,000	$ 3,388,310	$ 3,659,250
Share Based Payments	$ 369,538	$ 350,000	$ 719,538	$ 753,375
Employee Benefits	$ 1,106,668	$ 1,200,000	$ 2,306,668	$ 2,583,000
Depreciation and Amortization	$ 717	$ 1,000	$ 1,717	$ 2,153
Finance Costs	$ 126,788	$ -	$ 126,788	$ -
Other Operating Costs	$ -	$ -	$ -	$ -
Total Operating Costs	**$ 11,534,222**	**$ 11,751,000**	**$ 23,285,222**	**$ 25,294,028**
Gain (loss) Before Income Taxes	**$ (5,537,336)**	**$ (4,468,143)**	**$ (10,005,479)**	**$ (17,335,159)**
Income Tax	$ -	$ -	**$ -**	**$ -**
Gain (loss) After Income Taxes	**$ (5,537,336)**	**$ (4,468,143)**	**$ (10,005,479)**	**$ (17,335,159)**
Other Comprehensive Gain (loss)	$ -	$ -	$ -	$ -
Foreign Currency Translation	$ 1,179	$ -	$ 1,179	$ -
Total Comprehensive Gain (Loss)	**$ (5,538,515)**	**$ (4,468,143)**	**$ (10,006,658)**	**$ (17,335,159)**
Earnings (Loss) per basic share	**$ (0.01)**	**$ (0.01)**	**$ (0.02)**	**$ (0.02)**
Earnings (Loss) per diluted share	**$ (0.01)**	**$ (0.01)**	**$ (0.02)**	**$ (0.02)**
Basic Shares Outstanding	**535,789,100**	**640,539,100**	**588,164,100**	**718,614,009**
Diluted Shares Outstanding	**535,789,100**	**640,539,100**	**588,164,100**	**718,665,676**
Estimated Cash Balance	**$ 7,926,373**	**$ 11,755,373**	**$ 11,755,373**	**$ 4,497,578**

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Trickle Research holds two microcap conferences each year. Trickle Research encourages its coverage companies to present at those conferences and Trickle charges them a fee to do so. Companies are under no obligation to present at these conferences. As of this writing, Neurizon Therapeutics Limited has not paid fees to present at Trickle co-sponsored conferences, but we will encourage them to do so in the future.

Portions of this publication excerpted from company filings or other sources are noted in *italics* and referenced throughout the report.

Rating System Overview:

There are no letters in the rating system (Buy, Sell Hold), only numbers. The numbers range from 1 to 10, with 1 representing 1 "investment unit" (for my performance purposes, 1 "investment unit" equals $250) and 10 representing 10 investment units or $2,500. Obviously, a rating of 10 would suggest that I favor the stock (at respective/current levels) more than a stock with a rating of 1. As a guideline, here is a suggestion on how to use the allocation system.

Our belief at Trickle is that the best way to participate in the micro-cap/small cap space is by employing a diversified strategy. In simple terms, that means you are generally best off owning a number of issues rather than just two or three. To that point, our goal is to have at least 20 companies under coverage at any point in time, so let's use that as a guideline. Hypothetically, if you think you would like to commit $25,000 to buying micro-cap stocks, that would assume an investment of $1000 per stock (using the diversification approach we just mentioned, and the 20-stock coverage list we suggested and leaving some room to add to positions around allocation upgrades. We generally start initial coverage stocks with an allocation of 4. Thus, at $1000 invested per stock and a typical starting allocation of 4, your "investment unit" would be the same $250 we used in the example above. Thus, if we initiate a stock at a 4, you might consider putting $1000 into the position ($250 * 4). If we later raise the allocation to 6, you might consider adding two additional units or $500 to the position. If we then reduce the allocation from 6 to 4 you might consider selling whatever number of shares you purchased with 2 of the original 4 investment units. Again, this is just a suggestion as to how you might be able to use the allocation system to manage your portfolio.

For those attached to more traditional rating systems (Buy, Sell, Hold) we would submit the following guidelines.

A Trickle rating of 1 thru 3 would best correspond to a "Hold" although we would caution that a rating in that range should not assume that the stock is necessarily riskier than a stock with a higher rating. It may carry a lower rating because the stock is trading closer to a price target we are unwilling to raise at that point. This by the way applies to all of our ratings.

A Trickle rating of 4 thru 6 might best (although not perfectly) correspond to a standard "Buy" rating.

A Trickle rating of 7 thru 10 would best correspond to a "Strong Buy" however, ratings at the higher end of that range would indicate something that we deem as quite extraordinary..... an "Extreme Buy" if you will. You will not see a lot of these.