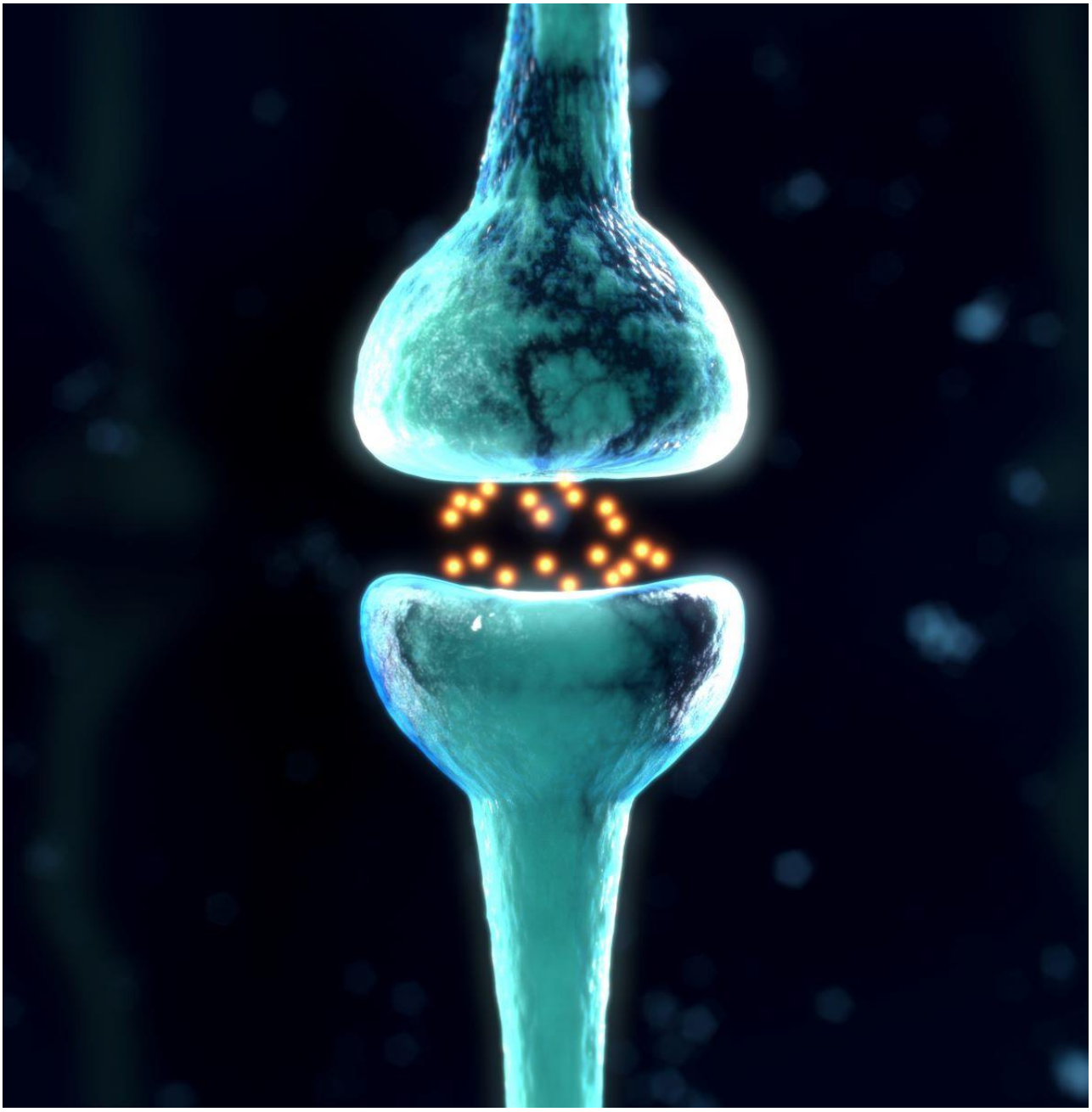

NeuroSense Therapeutics

Disclaimer

NeuroSense Therapeutics (the Company) is developing a novel drug to alter the progression of Amyotrophic Lateral Sclerosis (ALS) .

This business proposal has been prepared by NeuroSense Therapeutics solely for the consideration of eligible investors who have the knowledge and experience in financial and business matters and the expertise to conduct their own due diligence investigation in connection with the investment outlined herein.

This proposal is based on data, estimates, forecasts and other trade and statistical sources. While the Company believes that the information contained herein is accurate, it expressly disclaims any and all liability for representations or warranties expressed or implied, contained in, or for omissions from, this business plan or any other written or oral communication transmitted or made available to prospective investors. Any and all representations and warranties regarding the information described in this business plan shall be only as set forth in a definitive agreement if and when such definitive agreement is executed. Any prospective investor acknowledges his/her responsibility to perform a due diligence review prior to consummating a transaction with the Company.

Included in this proposal are certain declarations by the Company with respect to its anticipated future performance. These declarations are based on estimates and assumptions by the Company that are subject to economic and competitive uncertainties beyond the control of the Company. The Company does not provide representation or warranty withregard to the declarations and there can be no assurance that the future results will be realized or that actual results will not be significantly different from those projected. The Company is not obligated to update or revise any future declarations.

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Contents

COMPANY OVERVIEW

Our Vision

Finding a cure for Amyotrophic Lateral Sclerosis (ALS)

Our Mission

We believe in putting the patients first, and in giving back to the ALS community.
We aim to develop a breakthrough, targeted drug for ALS that will halt, or significantly delay, disease progression.
The aim of our novel fixed-dose combination platform is to treat ALS patients, with the potential to be a therapeutic option for additional neurodegenerative diseases.

Introduction

In 2016, a chance meeting between our CEO, Alon Ben-Noon, then owner of MediCan Consulting, and Shay Rishoni, an ALS patient, led to the birth of NeuroSense.

During this meeting, Alon was inspired by Shay, who served as the CEO of Prize4Life, a non-profit organization for ALS. Despite Shay not having a physical voice, therefore communicating via eye movement and computer software, the connection was instant. Shay detailed his activities in the ALS field, and described the efforts that are being made to solve the ALS puzzle, leading Alon to team up with world-renowned scientists and colleagues in order to research and develop an effective drug for ALS patients.

NeuroSense Therapeutics was established a few months later, based on research done by the team, related to several new approaches and pertinent therapeutic targets.

NeuroSense Therapeutics is spearheading a new approach for the treatment of ALS, by creating a unique formulation consisting of specific doses of two repurposed FDA-approved drugs. These compounds have been found to work synergistically on more than one pertinent target, showing an outstanding effect in two unique ALS zebrafish models.

THE DISEASE AND NEED

ALS Summary

Amyotrophic lateral sclerosis (ALS), commonly known as Lou Gehrig's disease, is a progressive and fatal motor neuron disease characterized by symptoms of degeneration of the upper motor neurons in the motor cortex as well as the lower motor neurons in the spinal cord and brain stem.
This leads to progressive paralysis of all voluntarily innervated muscles and therefore affects mobility, balance, communication, swallowing, and breathing. Death typically occurs 2–4 years after diagnosis, usually due to respiratory failure.

The exact causes of ALS are unknown but are believed to include genetic and environmental factors. Hereditary ('familial', 10% of patients) and sporadic forms (90% of patients) exist, and several gene mutations (i.e., SOD1, TDP-43, C9ORF, FUS and others) are implicated in the pathogenesis of ALS.

ALS affects adults between the ages of 30-70. 2 of 10,000 people are diagnosed with ALS annually, and around the world, at every given moment 1:10,000 has ALS. ALS is considered an orphan disease, as there are approximately 30,000 patients in the US, and half a million patients worldwide.

Current Clinical Practice and Standard of Care

Despite significant research efforts and various compounds tested, there is no cure for ALS, with only two FDA approved therapies on the market, both with limited efficacy in controlling symptoms:
- Riluzole (Rilutek®; Sanofi) - Standard of care drug administered orally and approved in 1994. It is believed to work by inhibiting glutamate release, a neurotransmitter that affects nerves sending messages from the brain to the muscles. It has only a modest effect and prolongs patient survival by an average of 2 months.

- Edaravone (Radicava™; Mitsubishi Tanabe Pharma) - Drug was approved in 2017 and is administered via infusion as an add-on to Riluzole. It is believed to work by reducing the level of free radicals and has limited efficacy, with some significant adverse events. In addition, the rigorous treatment schedule and IV administration of edaravone create additional concerns for ALS patients. Many patients need to have a port placed, patients with impaired mobility need assistance with transportation to an infusion center, creating a considerable burden to them and their care givers.

Treatment Costs

The yearly treatment costs of riluzole can reach $10k per patient, and the yearly treatment costs of edaravone can reach $150k per patient in the US.

Additional Factors Increasing the Cost of Care

The unmet need and limited efficacy of the available drugs pose a burden on health systems as all patients progress and require pharmaceutical/supportive treatments, assistance in activities of daily living (ADL), and frequent hospitalizations that may reach $180-460k per patient yearly.

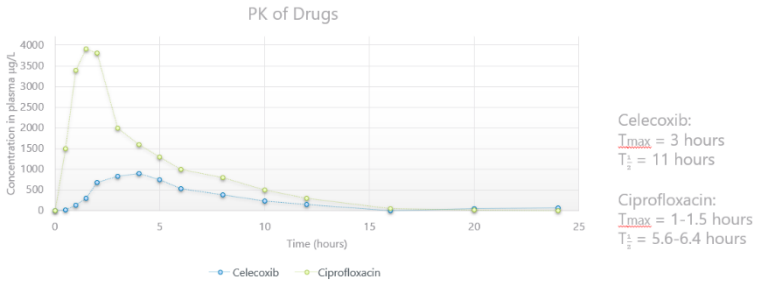
THE SOLUTION - PRIMEC

Introduction

PrimeC is an enhanced formulation of a fixed-dose combination (FDC) of two approved, generic drugs for the treatment of patients with familial or sporadic ALS.
The FDC comprises the antibacterial drug ciprofloxacin, a second-generation fluoroquinolone, together with the non-steroidal anti-inflammatory drug (NSAID) celecoxib, a selective cyclo-oxygenase-2 (COX-2) enzyme inhibitor that has anti-inflammatory, anti-glutamatergic, analgesic and antipyretic properties.

The Rationale Behind PrimeC

Neuroinflammation and dysregulation of RNA metabolism are two key pathological mechanisms in ALS. The major ALS associated genes SOD1, TARDBP, FUS, and C9orf72 are involved in aspects of RNA metabolism processes. Consequently, studies show a reduction in microRNAs expression in motor neurons of ALS patients (Parisi et al., 2013). MicroRNAs (miRNAs) are small fragments of nucleic acid that regulate gene expression. The importance of balanced miRNA expression was demonstrated in mice studies, which showed that the loss of miRNA biogenesis is sufficient to cause spinal motor neuron degeneration (Haramati et al, 2010).
Neuroinflammation is increasingly recognized as a major factor that promotes ALS progression and amplifies the motor neuron death-inducing processes. Neuroinflammation is characterized by extensive astrogliosis, microglial activation, and infiltration of peripheral immune cells at sites of neurodegeneration (Liu & Wang 2017; Morello et al., 2017).
PrimeC addresses these two important pathways: ciprofloxacin upregulates microRNA biogenesis, and celecoxib exerts an anti-inflammatory effect. These two drugs have been shown to be synergistic in regulating oxidative stress and inflammation (Kalle & Rizvi, 2011; Dey et al., 2018). PrimeC's unique formulation maximizes the synergy between the two compounds, allowing sustained release in order to utilize the PK profile of each drug, as shown below.



Zebrafish Models

Pre-clinical studies were conducted in two known ALS zebrafish models carrying the common SOD1 G93R mutation, or TDP-43 G348C mutation in order to demonstrate the synergistic effect of ciprofloxacin and celecoxib. The SOD1 G93R zebrafish model is known to show convincing aspects of adult-onset, has a slow progressing motor degenerative phenotype, and mimics many aspects of the disease in patients. The second zebrafish ALS model, TDP-43 G348C, is known to show several key phenotypes such as reduced motor function, reduced motor axons length and aberrant branching.
These models have several advantages over mouse models. When injected with the wildtype (WT) SOD1 gene, the mouse model develops axonopathy, as opposed to the fish, who do not show any ALS symptoms, as in humans with WT SOD1. In fish, only slight overexpression of mutant SOD1 is required to cause ALS symptoms, as opposed to mice, in which the mutated SOD1 must be abundant. Lastly, many drugs have shown extremely promising results in mice ALS models, but when tested on ALS patients in the clinic, they did not have any effect at all. In contrast, when these drugs were tested in zebrafish they showed no effect, therefore indicating a more predictive model.

The zebrafish is increasingly used as a powerful model for pharmaceutical and toxicological studies over the past decade. There are several reported zebrafish studies in CNS diseases which showed efficacy, and then continued directly into clinical trials, such as Clemizole. Clemizole, a novel drug for Dravet Syndrome, a catastrophic form of childhood epilepsy, was discovered in a zebrafish model and continued directly to clinical trials. In this study, Clemizole, a first-generation antihistamine, was shown to have antiepileptic activity in the zebrafish model for Dravet Syndrome (Griffin et al, 2017). The antiepileptic effect of clemizole cannot be tested in mouse models since the drug is rapidly metabolized in mice with a plasma half-life of <10 min (compared to 3.4 h in humans).

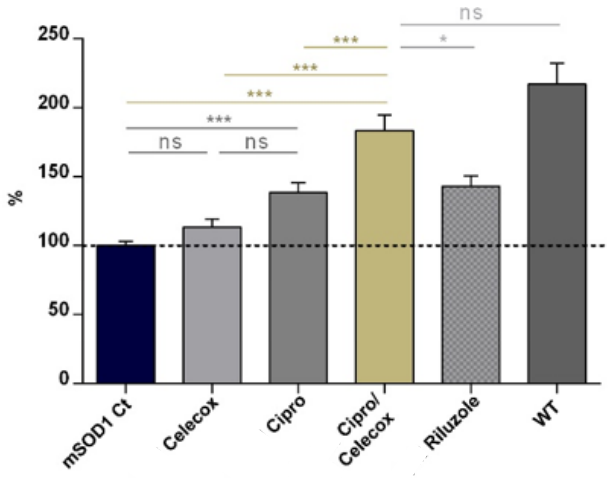
The mounting evidence in neuropharmacology, coupled with the large sample sizes, which contribute to the statistical strength of the study, as well as the large similarity between human and zebrafish CNS, convinced us to utilize this model for ALS drug discovery. Additionally, zebrafish larvae are transparent, contributing to the ease of the analysis of the drug's effects on different cell types, such as motor neurons, neuromuscular junctions, and microglial cells.

The study in the SOD1 ALS zebrafish model demonstrated a substantial improvement in swimming activity of the fish when treated with ciprofloxacin alone. This effect was significantly enhanced by 83% (P<.001) when PrimeC was tested, and considerable preservation of motor neuron axonal morphology and neuromuscular junctions was noted. Moreover, treatment with PrimeC was able to preserve microglial morphology in the mutated fish similar to the WT control fish. When the fish were treated with riluzole, the standard of care, PrimeC showed substantially better results (see graph).

The study findings in the TDP-43 model substantiated these results and demonstrated that PrimeC significantly improved the maximal swimming velocity and distance by 44% and 111% respectively. Moreover, treatment with PrimeC was able to preserve the movement pattern of mutant larvae similar to the WT control fish.

These results, that were replicated in two separate zebrafish models of ALS, show the great potential of PrimeC as a treatment for ALS patients.

*Full results will be published later this year



Comparison of increase in distance moved of WT, mutants and riluzole/PrimeC treated SOD1 mutants

REGULATORY STRATEGY

Introduction

Ciprofloxacin and celecoxib have a great deal of safety and tolerability data, due to their extensive long-time use in the clinic. Therefore, the regulatory pathway suitable for PrimeC is the 505(b)(2) new drug application (NDA).
Additionally, as ALS is an orphan disease with less than 200,000 patients in the US or EU, an Orphan Drug Designation for PrimeC has been granted by the FDA in the US, and will be requested in the EU as well.

Clinical Pathways

PrimeC is currently being evaluated clinically, with two parallel studies running in the US and in Israel in a total of ~45 ALS patients, with treatment duration of 1 year. Outcome measures include ALSFRS-R, Vital Capacity (VC), safety and biomarkers of axonal damage and motor neuron degeneration (i.e., miRNAs levels and neurofilaments) to assess the impact of treatment on disease progression rate.

During 2020, a Pharmacokinetics (PK) study in healthy volunteers will be initiated in Canada.

Upon completion of the PK study and interim analysis of the current studies in patients, and contingent on FDA approval, NeuroSense intends to initiate a seamless Phase II/III study, placebo-controlled (standard of care) in familial and sporadic ALS patients. The trial will begin with a 6-month dose range finding period, and will then continue for an additional 6 months, with one chosen dose of the treatment arm. The number of patients is estimated at ~150-200. Outcome measures will include ALSFRS-R, VC, several advanced functional outcomes (i.e., HHD, EIM, PET imaging), and two biomarkers collection (i.e, miRNA levels and neurofilaments) to evaluate whether PrimeC can slow, or even halt the disease.
In addition, safety measures will be taken to verify PrimeC is safe and tolerable.

Once the phase II/III trial is complete, and based on a previous case study in ALS (Mitsubishi Tanabe's Edaravone), the drug may achieve conditional approval from the FDA.

THE MARKET

Market Insights

Until 2017, riluzole (rilutek) was the only approved drug for ALS. It is considered the standard of care and is therefore taken by more than half of ALS patients. As mentioned, riluzole is sold at $10,000 per patient, per year.

In May of 2017, Mitsubishi Tanabe's edaravone (Radicava) became the second FDA-approved ALS drug. Despite high hopes, sales have recently decreased since many patients have trouble with its administration, price and efficacy. The cost of treatment reaches $150,000 per patients, per year.

It is observed that in the base year 2016, North America dominated the ALS treatment market in terms of revenue due to factors such as upsurge in funding by government agencies and private organizations in the healthcare system, increase in prevalence of neurological diseases, rise in geriatric population, favorable reimbursement policies and higher treatment awareness.

The ALS market is currently valued at a few hundred million USD, consisting of two low efficacy drugs. Once an effective drug has been found, the market is estimated to reach $2-5 billion USD (several tens of thousands of USD per patient per year, with approximate 100K patients in the western world on the drug).

COMPETITIVE LANDSCAPE

Market Competition Assessment

The ALS market is observed to be relatively competitive and is comprised of several players. The market is currently dominated by few companies such as BrainStorm Cell Therapeutics, Orion Pharmaceutical, Orphazyme, Mitsubishi Tanabe Pharma Corp., and others. There are multiple different strategies that are being tested in clinical trials for ALS treatment. PrimeC holds several advantages over the competitors: PrimeC targets multiple pathways in parallel compared to other treatments which target only one pathway (this strategy may lead to more significant results since this is a complex disease). Additionally, PrimeC is a non-invasive treatment, compared to other treatments (e.g Brainstorm Therapeutics-NurOwn). Finally, PrimeC is a disease-modifying treatment compared to some treatments that are being tested for the relief of ALS symptoms only (e.g Levosimendan).

Phase III Studies

Company	Product	MoA
Brainstorm Therapeutics	NurOwn	Mesenchymal stem cells
Orion Pharma	Levosimendan (OMD-109)	Help muscles contract more easily
Orphazyme	Arimoclomol	Stimulate a normal cellular protein repair pathway

Phase II Studies

Company	Product	MoA
Grifols Therapeutics	Albutein® 5%	Increase albumin levels in the plasma
Gilead Sciences	Ranolazine (an FDA-approved drug for angina pectoris)	Potential neuroprotection via inhibition of sodium current and intracellular calcium accumulation
Amylyx Pharmaceuticals	AMX0035 (a fixed dose combination of phenylbutyrate and taurourosodeoxycholic acid, both have been used in the clinic, with proof of safety and tolerability)	The drug combination was shown to inhibit nerve cell death and inflammation of nerve cells in animal models
Mallinckrodt Pharmaceuticals	Achtar gel	Prevention of neuroinflammation through stimulation of the immune system by induction of hormones

IP Status & Position

IP Status

PrimeC has a combination use patent, PCT/IL2018/050684, which is currently under evaluation. The pre-clinical studies were conducted in cooperation with Dead Sea and Arava Science Center (DSASC), Ben-Gurion University and McGill University in a service agreement only, therefore these two institutes do not hold rights of the patent.

The patent includes claims related to: doses, ratios, formulation, and families of compounds in combination.

Partial freedom to operate (FTO) analysis has been conducted, and according to Dr. Eyal Bressler & Co, a synergistic combination of an anti-inflammatory drug and a Dicer activator can be considered to have FTO for ALS indication.

We intend to submit additional patent applications for the unique FDC formulation. Additional patents will be submitted following clinical trial findings.

In addition to the patent, there is extended market exclusivity (7 years in the US and 10 years in Europe) for PrimeC due to an Orphan Drug Designation.

BUSINESS MODEL & STRATEGY

Business Model

NeuroSense Therapeutics will take advantage of the accelerated regulatory pathway for its initial target application (orphan drug designation for ALS) in order to bring PrimeC to the market on a tight budget and an aggressive timeline.

Significant value-building milestones for NeuroSense's unique combination will be achieved as the clinical trials progress. Refer to the Go-to-Market Strategy below for more information about the commercialization pathway for NeuroSense's initial target application.

NeuroSense may utilize the Right-to-Try legislation, in order to sell PrimeC to patients in the US for compassionate use, hereby generating a small revenue, whilst awaiting clinical trial results.

NeuroSense may also consider an IPO on NASDAQ, based on cash flow and needs. This may be pursued in late 2020 or 2021 and will require sufficient clinical data and a broader pipeline, which are being addressed.

NeuroSense is currently exploring additional indications for treatment with PrimeC. Pre-clinical studies have commenced in Parkinson's disease (PD), based on the shared pathological mechanisms of PD and ALS.

Revenues for sale of PrimeC are expected to begin in 2023 following an NDA approval. Sales for ALS will be focused primarily on the US and later in the Western European markets (following submission to EMA), through a network of leading distributors with proven channels into the target markets.

Having clearly established the scientific, clinical and business value propositions, NeuroSense will be well-positioned to collaborate with global pharma companies such as Mitsubishi, Pfizer, and others in order to bring additional CNS applications to market. Licensing agreements and joint ventures will mitigate NeuroSense's risks as it fully leverages its fixed-dose combination platform, with royalties and shared revenues further enhancing the Company's profitability.

Pricing Model

As previously mentioned, treatment with riluzole costs $10k per patient, whereas edaravone costs $150k.

Based on NeuroSense's current understanding of production costs, the company believes PrimeC can be made available to patients at prices that are competitive to other innovative therapies with substantial profit margins. PrimeC will cost ~$25k per year, and will most probably be covered by insurance, similarly to edaravone, which is covered for the relevant patients. NeuroSense assumes that by 3 years from reaching the market, most patients will take PrimeC. See Revenue Forecast on page 20 for additional information.

Go to Market Strategy

A phase II clinical trial began in Israel in November 2019, in parallel to a clinical study in the US in two leading medical centers. Following successful results in these studies, a second phase II/III trial will be initiated, bringing PrimeC to the market in H1 2023.

Reimbursement

PrimeC's pricing model is feasible, due to the assumption that the costs of the drug will be covered by insurance companies. The new drug radicava, is reimbursed despite its high price, for the sub-population of patients who fit the inclusion criteria of Mitsubishi Tanabe's pivotal clinical trial. This example, therefore, illustrates that reimbursement is most likely.

PARTNERS & COLLABORATIONS

Barrow Neurological Institute (BNI)

The world's largest neurological disease treatment and research institution, located in Pheonix, Arizona. BNI has a large ALS clinic that treats patients, as well as providing them with news and data of clinical trials. NeuroSense is collaborating with BNI on sequencing of miRNA samples, and additional programs, including preparations for a clinical trial.

Niva Russek-Blum's Neurobiology lab, Dead Sea and Arava Science Center (DSASC), Ben-Gurion University

The lab studies the connection between the immune system and the CNS, as well as screening for potential drugs for ALS using zebrafish models of the disease. NeuroSense's collaboration with this lab includes testing of PrimeC on a SOD1 zebrafish model of ALS.

Gary Armstrong's lab, McGill University, Canada

The lab studies synaptic defects in ALS, using zebrafish models of ALS. Additionally, the lab has developed a zebrafish drug screening platform for the disease. NeuroSense's collaboration with this lab includes testing of PrimeC on a TDP-43 zebrafish model of ALS.

Tel-Aviv Sourasky Medical Center (TASMC), Israel

The neuromuscular disorders clinic in TASMC, headed by Professor Vivian Drory, has been treating ALS patients for many years. TASMC is running a phase II clinical study sponsored by NeuroSense, in order to evaluate the safety and efficacy of PrimeC in patients.

BUDGET OVERVIEW: 2019-2023

Expense Budget in USD

	2019	2020	2021	2022	2023
R&D Expenses	0.35M	1M	4.3M	5M	2M
Operating Expenses	0.25M	0.4M	0.7M	1M	5M
Total Expenses	0.6M	1.4M	5M	6M	7M

The clinical trial phase will require the company to recruit 1-2 more employees and the company will mainly rely on outsourcing services. In addition, NeuroSense is using a global CMO, Recipharm, to manufacture the final PrimeC formulation, according to current good manufacturing practice (cGMP) FDA requirements.

Funding by Round Period

NeuroSense has raised ~$1M from angel investors (pre-seed and seed rounds). In July 2019, NeuroSense raised an additional $1M through PipelBiz, a crowd-funding platform (round A).

NeuroSense is currently running a second crowd-funding campaign to raise $0.5M, in order to accelerate the clinical development program.

During 2020, following successful interim results from the current clinical studies, NeuroSense plans to commence an additional fund-raising round (round B), to raise $15-$20M from VCs or through an IPO, in order to prepare and execute a pivotal phase II/III study in ALS patients, leading to FDA submission.

FINANCIALS: 2023-2030

Sales Forecast

The sales forecast is based on the following assumptions:

- The main geographic markets are North America, Europe, Australia and Japan.
- The relevant patient population in these geographies for ALS is approximately 150,000.
- It is assumed that the patient population will increase annually by ~1% in these regions
- The drug price to distributors is estimated at ~$25k per patient per year.

Potential Revenues Forecast

Best case scenario, in which drug price is $25k a year, with a growth of 20-30k consumer patients per year:

	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032	Total
Number of Patients	30K	50K	80K	110K	130K	130K	150K	150K	150K	150K	
Cost per Year	25K	25K	25K	25K	25K	25K	25K	25K	25K	25K	
Total	750M	1.25B	2B	2.75B	3.25B	3.25B	3.75B	3.75B	3.75B	3.75B	28.25B

Worst case scenario, in which drug price is $7.2k a year, with a growth of 3-5k consumer patients per year, and assuming a more effective drug enters the market in 2032:

	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032	Total
Number of Patients	8K	12K	15K	20K	25K	30K	30K	30K	30K	18K	
Cost per Year	7.2K	7.2K	7.2K	7.2K	7.2K	7.2K	7.2K	7.2K	7.2K	7.2K	
Total	57.6M	86.4M	108M	144M	180M	216M	216M	216M	216M	129.6M	1.57B

THE TEAM

Core Team

Alon Ben-Noon, CEO& Co-Founder

- Former owner of Medican Consulting which worked with Teva, NeuroDerm, Chiasma, Mediwound, FutuRx portfolio firms, and many more.
- Former Project Manager for Innovative R&D Programs at Teva Pharmaceuticals
- MBA and BSc in Industrial Engineering, both from Ben-Gurion University

Dr. Oron Yacobi-Zeevi, CSO

- Former CSO at NeuroDerm (sold for $1.1B), responsible for the development of their unique formulation
- Twenty years of experience in leading R&D programs for numerous small molecule drugs and biologicals in various indications, including CNS diseases
- DVM: The Hebrew University of Jerusalem; PhD in Microbiology and Immunology: Ben-Gurion University

Dr. Jeff Sterling, Head of R&D and CMC

- Over 38 years R&D experience at Teva Pharmaceuticals
- One of the inventors of Azilect® (Parkinson's)
- Rothschild Prize for Innovation; Horev Prize for Excellence in Medicinal Chemistry
- PhD in Chemistry: Johns Hopkins University

Avital Pushett, Project Manager

- Has a personal interest in ALS drug development (lost her father to ALS).
- BSc-Biology and Business Management; MSc – Biomedical Sciences. Both from the Hebrew University of Jerusalem
- Certified CRA
- Has experience in stem cell research, including molecular biology and tissue culture.

Scientific Advisory Board & Supportive Team

Prof. Jeremy Shefner

- A senior VP at the Barrow Neurological Institute; Chair of the Department of Neurology
- Member of the Editorial Boards of the ALS Journal and Neurotherapeutics
- Co-founded the Northeast ALS Clinical Trials Consortium (NEALS; 1996)
- MD: Northwestern University Medical School; PhD (sensory physiology): The University of Illinois

Dr. Ben-Zion Weiner

- A senior VP of Global Research and Development at Teva (37 years total at Teva)
- Twice received the Rothschild Prize for industrial innovation for the development of Copaxone for the treatment of multiple sclerosis, and alpha D3 for kidney and bone disorders
- PhD in Organic Chemistry. Postdoctoral Fellowship at Schering Plough

Dr. Shoshi Tessler

- VP R&D Biosight Pharma
- Former VP R&D at Enzymotecand Project Champion at Teva Pharmaceutical Innovative R&D
- PhD in Biology: the Technion-Israel; Postdoctoral Fellowship: Babraham Inst, Cambridge UK (in the field of glutamate transporters and neurological diseases)

Dr. Nili Shultz

- CEO of a portfolio company at FutuRx accelerator
- Over 10 years of leadership experience in pharma discovery & development (EPIX, Dynamix)
- PhD from USC (LA, USA) under the supervision of Arieh Warshel (Nobel Prize in Computational Chemistry)

Dr. Yossi Gilgun-Sherki

- A clinical, regulatory and scientific Consultant and Lecturer
- 20 years in the pharma industry in various companies and key R&D leadership roles
- Former head of Specialty Products portfolio at Dexcel Pharma and Global Clinical Leader & Senior Scientist at Global IR&D, Teva Pharmaceuticals
- PhD in neuroscience from Tel-Aviv University and MBA from the Open University, Israel

Dr. Revital Mandil-Levin

- Former Chief Business Development Officer at CollPlant
- Served as Vice President Corporate Development at NeuroDerm
- 9 years as Vice President Business Development at HealOr
- PhD in Biochemistry from Bar-Ilan University, and MBA from Israeli College of Management School of Business

Board of Directors

Gil Hakim

- Chairman and CEO of Armenta
- Over 20 years of experience in the biotech industry, including; President of Israeli Operations at Urogen Pharma, CEO of Theracoat (Urogen Pharma), Director of New Product Development at Medispec, etc.
- B.Sc. in Life Sciences from Ben-Gurion University

Amir Gross

- Co-founder & Managing partner of CardioValve
- Founder & former CEO of Valtech Cardio (sold for $1B)
- Over 15 years of leadership experience in the medical device industry
- Amir currently holds more than 40 patents and patent applications

Prof. Itzhak Krinsky

- 12 years at Teva, including; Executive Vice President, Corporate Business Development, member of the Teva Executive Committee, Chairman of Teva Japan, Chairman of Teva South Korea, and Head of Business Development Asia Pacific.
- Named by SCRIP as one of the top 100 Global Leaders in the Pharmaceutical Industry
- Emeritus Professor of Finance & Business Economics at the Michael G. DeGroote School of Business, McMaster University, Ontario, Canada

In addition - **Dr. Oron Yacobi-Zeevi & Alon Ben-Noon**

Discussion & Conclusions

PrimeC has shown its efficacy in two different ALS mutations in zebrafish models, thus supporting its ability to improve locomotion activity and preserve the morphology of WT motor neurons and microglia.

PrimeC showed no safety concerns, including cardiovascular abnormalities (heart rate, morphology, hemorrhage and edema) or mortality.

The results clearly support the synergistic activity of PrimeC, as previously demonstrated in in-vitro studies, and show that a combination of ciprofloxacin and celecoxib can effectively target various pathological mechanisms involved in ALS, including neuroinflammation and miRNA dysregulation.

These overall findings suggest that PrimeC will have clinically meaningful effects in slowing or even halting ALS progression in humans, thus giving hope to treat this devastating disease. Therefore, NeuroSense Therapeutics is spearheading the clinical phase of PrimeC's development.

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