NeuroVive

Sector: Biotech

On the right track

Redeye initiates coverage of NeuroVive, a high-risk case with a promising early-stage portfolio in primary mitochondrial diseases. While previous high-profile failures and dilutive rights issues have hurt the company's standing, the share's huge decline has taken valuation to undemanding levels – though nearer-term catalysts are few and the current capital raising may weigh on performance.

World-class science

Although it has only one asset in active clinical development, NeuroVive is on the right track, in our view. It is focusing its world-class science and highly competent core team on bringing two compounds to market. Moreover, non-core assets could be out-licensed.

Orphan potential

KL1333, in-licensed in 2017 and already in Phase Ib, has orphan drug designation, and the internally developed NV354 – has orphan drug potential. The designation enables cost-efficient development and an exclusivity period after approval.

Undervalued, but high risk

Our base case scenario, which puts both KL1333 and NV354's likelihood of approval (LOA) at around 10% and factors in the new issue, values NeuroVive at SEK 1.2 per share. Our bull case, which assumes KL1333 demonstrates efficacy and LOA rises above 50%, is as high as SEK 6 – but our bear case, which sees KL1333 fail Phase II endpoints, is below SEK 0.30.

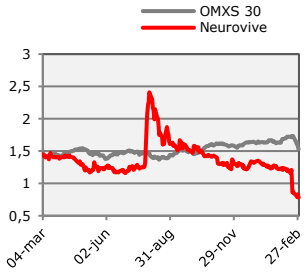
Limited catalysts

While efficacy data is unlikely until 2022, minor catalysts in the meantime could be KL1333 completing phase I and NV354 starting phase I, as well as receiving an Investigational New Drug (IND) designation by the FDA.

FAIR VALUE RANGE

BEAR	BASE	BULL
0.3	1.2	6.4

NVP.st VERSUS OMXS30



REDEYE RATING

KEY STATS

Ticker	NVP.st
Market	Small cap
Share Price (SEK)	0.8
Market Cap (MSEK)	220
Net Debt 20E (MSEK)	-67
Free Float	100 %
Avg. daily volume ('000)	86

KEY FINANCIALS (SEKm)	2018	2019	2020E	2021E	2022E	2023E
Net sales	0	0	0	0	0	2
EBITDA	-69	-79	-53	-92	-82	-102
EBIT	-73	-77	-56	-94	-84	-104
EPS (adj.)	-0.8	-0.4	-0.2	-0.2	-0.2	-0.2
EV/Sales	N/A	N/A	N/A	N/A	N/A	N/A
EV/EBITDA	-1.6	-2.4	-2.9	-1.0	-2.1	-2.7
EV/EBIT	-1.5	-2.5	-2.7	-1.0	-2.0	-2.6
P/E	-1.9	-3.3	-3.9	-3.6	-4.0	-3.3

ANALYSTS

Gergana Almquist

gergana.almquist@redeye.se

Niklas Elmhammer

niklas.elmhammer@redeye.se

Investment Case

Clinical progress

NeuroVive has world-class science and a very competent core team in the mitochondrial medicine field, as well as a large network of collaborators and partners. We are optimistic about its ability to execute on the strategy to focus on compounds with orphan drug potential in the primary mitochondrial disorders space.

Since 2017 NeuroVive has strengthened its project portfolio in primary mitochondrial diseases, notably through the in-licensing of the clinical asset KL-1333. A phase I study is currently well underway. In addition, the internally developed drug candidate NV354 is making good progress towards the clinical stage. We see a good prospect of these projects advancing to the Phase II and I stage, respectively, in the next 12-24 months.

Financing pressure

At the current market level of around SEK 149m, we view the project portfolio as undervalued. A key reason for the recent share price slump has been the need for additional financing to advance clinical development. This is being addressed currently through a rights issue of SEK 74m. Provided existing shareholders participate strongly in the issue, we see potential for the shares to recover towards SEK 1.2 (our base case scenario).

Out-licensing upside

Possible out-licensing of the non-core assets NeuroSTAT and NV556 could provide significant upside to our base case. We do not include these opportunities in our current valuation as interest from potential partners is difficult to assess due to limited clinical data.

Risks

Development risk

Developing drugs in the CNS/mitochondrial space is challenging and the historical likelihood of reaching approval is low. NeuroVive is highly dependent on its clinical project KL1333 for the treatment of Melas-MIDD spectrum disorders.

Further financing

The company has limited funding. This might delay the project portfolio's advancement beyond Phase I.

Guarantor selling

As the shares are trading just above the upcoming rights issue's subscription price, there is a risk that guarantors will have to take up a significant proportion of the issue. This could lead to subsequent selling pressure.

Company overview

NeuroVive is a Lund-based, Swedish biotech company founded in 2000 by Dr Eskil Elmér. It focuses on developing treatments for primary mitochondrial diseases. The company was listed on the unregulated trading platform Aktietorget in 2008 and on NASDAQ OMX Small Cap in 2010.

After initially focusing on the organ-protective properties of cyclosporine A and twice failing in clinical trials, the company shifted focus towards primary mitochondrial disorders. The compounds that failed at efficacy were CicloMulsion (discontinued in 2015) for injury protection during heart surgery, and NVP019, a cyclophilin inhibitor intended for intravenous treatment to protect organs from injury owing to oxygen deprivation.

NeuroVive's new business strategy is to develop or in-license niche compounds that can acquire orphan drug status. This makes them economical to develop and more efficient to sell. Its main drug candidates advancing towards efficacy testing are KL1333, for the treatment of MELAS-MIDD spectrum, and NV354, a succinate prodrug designed to bypass the mitochondrial complex I deficiency.

Primary mitochondrial disorders affect the ability of cells to convert nutrition and oxygen into energy. The precise incidence of mitochondrial diseases is unknown as many are undiagnosed. As a group, mitochondrial diseases occur in about 1 in 4,000 people[1].

People and partnerships

NeuroVive depends on the competence and quality of its core team. We believe the leadership team has the profound expertise in mitochondrial medicine needed to implement the strategy to develop effective treatments for rare mitochondrial diseases.

Right team to implement the strategy

Founder Eskil Elmér is still the company's Chief Scientific Officer and has the overall research responsibility for NeuroVive. He also remains a significant owner, active in research, and a practicing physician in neurophysiology.

Pre-clinical and clinical development is the responsibility of Magnus Hansson, who has a PhD in experimental brain research and extensive research and clinical experience. He has been with the company since 2008 and has served as Chief Medical Officer since 2016.

Mark Farmery, responsible for business development, has experience in this role from AstraZeneca and Karo Bio AB, and has a PhD in biochemistry and molecular biology.

CEO Erik Kinnman has experience from both the pharmaceutical and financial industries.

In all, more than 10 employees are active in pre-clinical and clinical development, and all employees are science graduates, with five holding PhDs in medical sciences. Most of the company's researchers are qualified physicians engaged in identifying segments with unmet

[1] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5967365/

medical needs and in conducting early basic research and clinical research towards approval of drug candidates.

Partnerships with world-class research organisations

NeuroVive has established good relationships with the academic and business communities active in mitochondrial research in Europe, Asia, and the US.

Isomerase is a British company active in discovery, development, and small- and large-scale manufacturing. It is one of NeuroVive's key partners. This relationship is strengthened by a partial ownership stake and the collaboration focuses primarily on development of NeuroVive's chemistry platforms, i.e. producing active compounds for treating ischemic stroke and mitochondrial disorders, and compounds for organ protection. Isomerase also helps identify new development platforms in indications with a pressing medical need, and its drug development expertise provides valuable backing for NeuroVive's projects.

Furthermore, NeuroVive collaborates with Korean pharmaceutical company Yungjin Pharm on the clinical development of the KL1333 project for the treatment of primary mitochondrial disorders, which it has in-licensed from Yungjin.

It also has a partnership with PENN (the University of Pennsylvania), CHOP (Children's Hospital of Philadelphia), Karolinska Institute and a range of contract research organizations.

Ownership and the share

No institutional ownership

NeuroVive is owned mainly by small private investors and almost all institutional investors have left the stock. There are no strong main owners. The share has lost 99% of its value since it peaked at more than SEK 70 in August 2014, due to the discontinuation of advanced projects and dilution from new share issues.

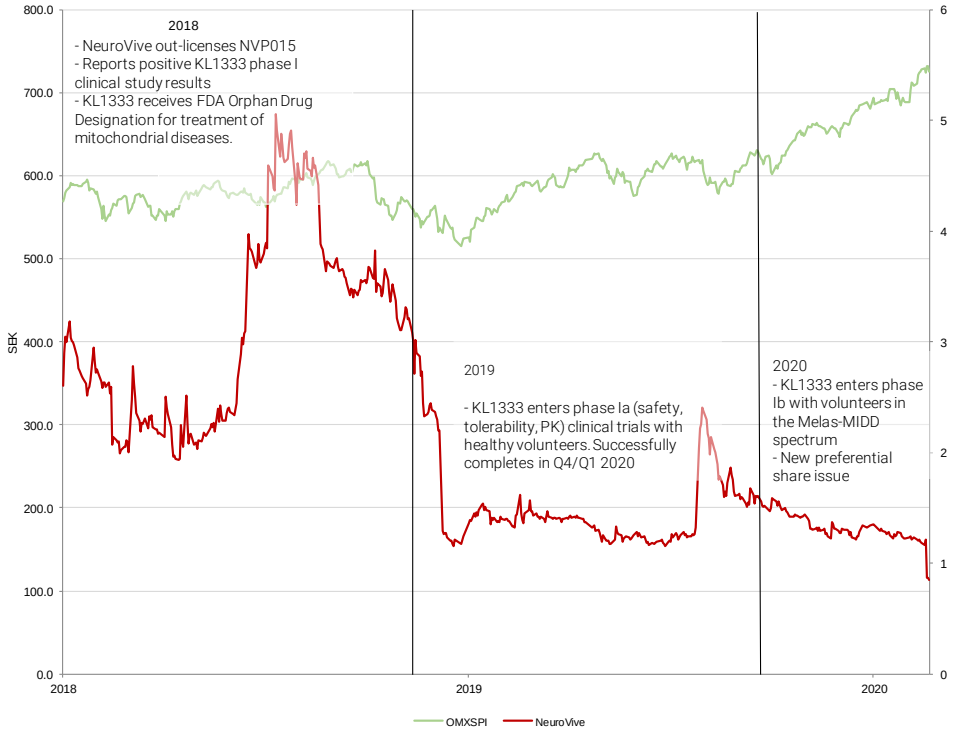
New share issue might trigger guarantor sales

At the time of writing, the total number of shares is 185,952,591 and the market cap is around SEK 150m. The free float is 100%, and the management and board own less than 0.87% of the capital.

On 19 February 2020, the company proposed a new share issue at a price of SEK 0.80, which will add 93m new shares and bring the total number of shares to 278.9m. For existing shareholders who choose not to participate in the rights issue, this implies a dilution of approximately 33% of shares and votes given full subscription.

Continued share price slide

The total turnover of the stock since the beginning of 2020 is SEK 36m for an average price of SEK 1.2 per share. Since the spike in July 2019 (thanks to reported progress in KL1333's development and the fast track designation for NeuroSTAT), the share is down approximately 66%, and since the beginning of 2020 it has lost around 40%, mainly due to the proposed new share issue.



Source: Redeye Research

We see no significant catalysts for the stock in the near future. A possible uplift could be the successful completion of phase Ib for KL1333 later in H2 2020, but we believe this will have a limited impact on the stock price. In our view, the first real stock price catalyst is the results of the efficacy tests in 2022.

Rights issue to fund development towards phase II

To fund the completion of the ongoing clinical phase Ib trial for KL1333, and the preparations for further clinical development, the board has recently proposed a right issue of up to **93m** new share. The new funds will be used to also obtain IND for KL1333 and NV354, to prepare for phase II for KL1333 and clinical phase I for NV354), The **subscription price is SEK 0.80 per share**, which implies the company will raise approximately **SEK 74m** before costs. The board's resolution is subject to approval at the EGM on 17 March 2020.

Each existing share in NeuroVive entitles the holder to one subscription right. Two subscription rights entitle the holder to subscription of one new share. The record date for participation is 1 April 2020 and the subscription period is 6-24 April 2020. The last day for trading in NeuroVive's share including the right to participate in the rights issue is 30 March 2020.

The company has entered into subscription commitments with existing owners totalling SEK 6.1m, corresponding to approximately 8.2% of the rights issue. A number of investors have committed to guaranteeing up to SEK 60.9m, corresponding to approximately 82% of the rights issue. In total, the rights issue is covered by subscription and **guarantee commitments amounting to SEK 67m, corresponding to approximately 90%**.

Upon full subscription in the rights issue, the number of shares will increase from 185.9m shares to 278.9m shares. For existing shareholders who choose not to participate in the issue, this implies a dilution of approximately 33% of shares and votes given full subscription.

The proceeds will increase NeuroVive's cash position to SEK 123m after costs. We believe this should provide the company with sufficient funding to complete the phase I clinical trial of KL1333 and the initial stages of phase II, as well as phase I for NV354. We expect that NeuroVive will need to raise new capital in mid-2021 or into H2 that year.

NeuroVive's main assets

As a niche biotech company, NeuroVive focuses its resources on developing an orphan drugs portfolio for primary mitochondrial diseases, an area of high unmet medical need. It currently focuses on two projects:

- KL1333, an NAD+ modulator for the treatment of primary mitochondrial diseases such as MELAS-MIDD spectrum disorders. It is currently in phase Ib tests in the UK and has been granted orphan drug designation.
- NV354, an energy replacement succinate prodrug (an alternative energy source) for the treatment of Leigh syndrome. It is currently in preliminary pre-clinical safety studies in experimental models and scaling up drug production, in preparation for clinical trials to start in 2021.

NeuroVive is actively looking for a partner or to out-license the two other projects in its pipeline: NeuroSTAT, with active substance cyclosporine for the treatment of traumatic brain injury; and NV556, for the treatment of NASH.

KL1333

KL1333 is a small molecule and NAD+ modulator

NeuroVive in-licensed the global rights (except in South Korea and Japan) for KL1333 in 2017 from Yungjin Pharm Corporation Ltd. Its goal is to develop KL1333 for the treatment of MELAS, KSS, Pearson syndrome, PEO and MERRF – primary mitochondrial diseases. KL1333 is a small molecule and an NAD+ modulator, stimulating the production of new mitochondria within an organ's cells to increase energy. It has been tested on material containing the A3243G genetic mutation and is especially relevant for mitochondrial diseases caused by this mutation. KL1333 has orphan drug designation in both the US and Europe.

Terms of the licensing agreement

Since KL1333 is NeuroVive's most important asset, we explain the details of the licensing deal. The licensing deal agreement states NeuroVive will pay Yungjin Pharm an upfront payment of USD 1 million at signing, USD 1 million one year after signing (this combined USD 2m has been paid), and an additional USD 1 million after the completion of a successful phase I clinical trial.

Further payments will be made:

- upon successful achievement of clinical development (USD 12m in total)
- market and reimbursement approval milestones (USD 42m in total).

In addition, Yungjin Pharm will also receive **tiered single- to low double-digit royalties on future net sales** and both companies will develop KL1333 in their respective territories, primarily for the treatment of genetic mitochondrial disease. KL1333 is patented until 2034.

Mechanism of action and initial proof of concept

KL1333 modulates cellular levels of NAD+, a central coenzyme in the cell's energy metabolism, and is intended for oral treatment. KL1333 has been shown to increase mitochondrial energy output, reduce lactate accumulation, diminish the formation of free radicals, and have long-term beneficial effects on energy metabolism.

In its discovery phase, KL1333 was tested for efficacy in vitro, in a cell model, and in animal cells. It was also tested in healthy human cells and in the cells of MELAS patients with the m.3243A>G mutation. No results have been published other than press releases and public summaries of the trial. No biomarkers were analysed[2]. These tests showed that KL1333 improves "energy balance, decreases oxidative stress, and restores mitochondrial functions and could be used to relieve the deleterious effects of mitochondrial diseases" [3]

The compound is manufactured in Europe, a process that is currently being refined. A detailed description of the clinical trial design can be found in Appendix 3.

Milestones for KL1333

NeuroVive completed the phase Ia clinical trial in healthy volunteers in Q4 2019/Q1 2020 and phase Ib is about to start and is due to be completed in Q3 2020. The company expects to initiate a clinical phase II efficacy study in H1 2021.

NV354 – an alternative energy source

A prodrug for energy replacement in Leigh syndrome

Of the in-house developed NVP015 programme, NV354 has been selected for further development. It is a succinate prodrug for energy replacement, targeting the treatment of paediatric conditions such as Leigh syndrome. It is patented until 2034. It is a chronic, oral treatment that prevents further deterioration of symptoms and signs. It is currently in pre-clinical toxicity studies, due to be completed in 2020.

The problem with succinate is that it does not enter the cells efficiently in its natural state. To deliver it to the cells, NeuroVive has in-licensed a prodrug technology. A prodrug is an inactive drug that is first activated on entering the body through the transformation of its chemical structure. This technology allows it to enter the cells, enabling electron transport from complex II, by-passing the mitochondrial complex I deficiency in the electron transport chain. NV354 has been selected for further development in the programme based on its tolerability, oral bioavailability, plasma stability, and organ delivery, specifically to the brain (high brain distribution).

NV354 has shown good tolerability in initial toxicology studies. It has a preclinical proof-of-concept in experimental models of complex I dysfunction and bypasses the most common mutations in Leigh syndrome and LHON (complex I).

[2] https://clinicaltrials.gov/ct2/show/NCT03056209?term=KL1333&draw=2&rank=2

[3] Min, Ki-Nam et all (2018) Frontiers in Neurology 9:552: KL1333, a Novel NAD+ Modulator, Improves Energy Metabolism and Mitochondrial Dysfunction in MELAS Fibroblasts.

Moreover, NeuroVive has established academic collaborations with two labs (Children's Hospital of Philadelphia and UAB in Barcelona) and in-house/contract lab studies. Early data has been presented at conferences, but the studies are not yet completed or published. The general concept has been published using the first generation of compounds (rather than NV354).[4]

Further development steps

Mitochondrial complex I (CI) deficiency is the most prevalent defect in the respiratory chain (the electron transport chain) in paediatric mitochondrial disease. This heterogeneous group of diseases includes serious or fatal neurological presentations such as Leigh syndrome. Only very limited evidence-based treatment options are available. NeuroVive plans to apply for Investigational New Drug (IND) designation to be able to do clinical testing in the US. Phase I (SAD or SAD/MAD) is projected to start in 2021, and the compound is expected to receive orphan drug and rare paediatric diseases designation the same year. One challenge with the clinical tests could be the access to patients with Leigh, and the company is considering testing the drug also for MELAS-patients.

Mitochondrial diseases

A **mitochondrion** is a membrane-bound organelle found in the cytoplasm of almost all eukaryotic cells (cells with clearly defined nuclei). Its primary function is to generate large quantities of energy in the form of adenosine triphosphate (ATP). Mitochondria are typically round to oval and range in size from 0.5 to 10 μm. In addition to producing energy, mitochondria store calcium for cell-signalling activities, generate heat, and mediate cell growth and death. The mitochondria have their own separate genetic code (mtDNA), which is inherited from the mother and is **highly susceptible to mutations,** because it does not possess the robust DNA repair mechanisms common to nuclear DNA.

The processes that convert food into energy occur primarily on the inner membrane, through the main energy-generating system of cells, the electron transport chain (ETC). The ETC uses a series of oxidation-reduction reactions to move electrons from one protein component to the next, ultimately producing free energy that is harnessed to drive the phosphorylation of ADP (adenosine diphosphate) to ATP. When this chain is disrupted, the mitochondria cannot produce a sufficient amount of energy.

Primary mitochondrial diseases

Typically, there are about **1,000 copies** of mitochondrial DNA per cell, and the percentage of these that are damaged or mutated determines whether a person will suffer from mitochondrial disease or not. Usually, more than 60% of the mitochondrial DNA molecules in a cell need to be mutated for the disease to appear, and the more mutated mitochondrial DNA a person has, the more severe their disease will be. Conversely, if the percentage of mutated DNA can be reduced, the disease could potentially be treated.

The mitochondrial diseases that appear to cause the most damage are those affecting cells of the brain, heart, liver, skeletal muscles, kidney, and the endocrine and respiratory systems. On the other hand, many people affected can have normal life spans, although they typically suffer from debilitating symptoms such as severe fatigue.

[4] https://www.nature.com/articles/ncomms12317

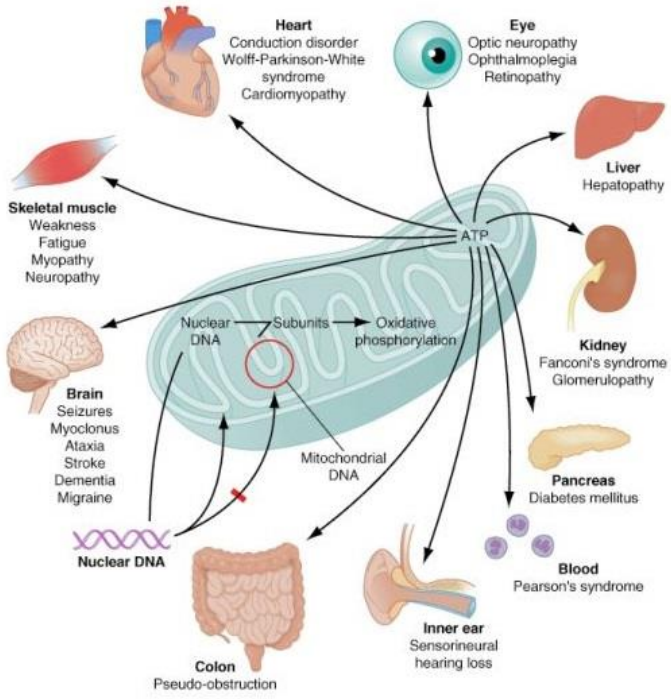


Image source: Fauchi AS, Kasper DL, et al (2008): Harrison's Principles of Internal Medicine, 17th Edition.

Mitochondria dysfunction implicated in CNS diseases and cancer

There are many inherited and acquired mitochondrial diseases. Inherited diseases may arise from mutations transmitted in maternal or paternal nuclear DNA or in maternal mtDNA. Both inherited and acquired mitochondrial dysfunction is associated with several diseases, including Alzheimer's, Parkinson's, and cancer.

The most affected are the organs requiring most energy

When the mitochondria fail, less and less energy is generated within the cell, and the cells cease to function properly. Cell injury and even cell death can follow. If this process is repeated throughout the body, whole organ systems begin to fail. The parts of the body requiring the greatest amounts of energy, such as the heart, brain, muscles, and lungs, are the most affected.

Depending on the location of the affected cells, certain parts and/or functions of the body may no longer behave properly, leading to health problems and symptoms of varying severity. Because of the complex interaction between the hundreds of genes and cells that must co-operate to keep the body's metabolic machinery running smoothly, identical mtDNA mutations may not produce identical diseases.

High unmet medical need

It is estimated that at least one in 6,000 people and as many as one in 4,000 have a mitochondrial disease, with approximately 20,000 people in the US believed to have a form of mitochondrial myopathy, for which there is currently no cure. Approximately 1,000 to 4,000 American children are born with this disorder each year[5]. Currently, there is no treatment for primary mitochondrial disorders.

[5] www.mitochondrialdiseasenews.com

Mitochondrial diseases are **difficult to diagnose** because they affect everyone differently. Symptoms can include seizures, strokes, severe developmental delays, as well as an inability to walk, talk, see, or digest food, combined with a host of other complications. If three or more organ systems are involved, a mitochondrial disease should be suspected. As these diseases are rare and difficult to diagnose, their exact prevalence is not known.

MELAS

MELAS, or mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes, is a progressive, multisystem genetic disease.

Symptoms and natural history

MELAS symptoms can present at any age, although most often in childhood, with 65-76% of sufferers developing symptoms before the age of 20. Few cases present before the age of 2 (5-8%) or after 40 (1-6%). Individuals with MELAS typically experience disease progression that results in death. Median survival time from point of diagnosis is about 16.9 years, with a subgroup of 20.8% who are more severely affected and die within 7.3 years of diagnosis. Overall, children and young adults diagnosed with MELAS and displaying classic symptoms have a shorter lifespan than older adults with milder symptoms.

The natural history of MELAS involves gradual impairment of motor abilities, vision, and cognitive ability by the time of adolescence or young adulthood due to recurring stroke-like episodes. Almost all individuals with MELAS (94%) have lactic acidosis[6]. Individuals may also have recurrent headaches, anorexia, recurrent vomiting, possible exercise intolerance or proximal limb weakness, Wolff-Parkinson-White syndrome, or diabetes mellitus.

Determining the prevalence

The exact prevalence is unknown and has been estimated anywhere between 9 in a million and 16-18 per 100,000 individuals.[7] A study in northern Finland found the prevalence of MELAS mutation was 16.3 per 100,000 (95% confidence interval 11.3-21.4 in 100,000)[8]. The gene relevant for NeuroVive's KL1333 – m.3243A>G – had a prevalence of 10.2 in 100,000 (95% confidence interval 6.2-14.2 per 100,000).

One of the most detailed studies[9] of primary mitochondrial disease prevalence found that m.3243A>G (A3243G) causes disease in 3.5 per 100,000 adults in north-east England. A proportion of these patients will receive a MELAS diagnosis, but most have a combination of debilitating disease expressions, such as hearing loss, metabolic dysfunction, myopathy, and fatigue.

Caused by mutations in mitochondrial DNA

MELAS is caused by mutations in the mitochondrial DNA (mtDNA) that are always maternally inherited. This means that a female who carries the mtDNA mutation will pass it on to all her children, but a male who carries it will not pass it on to his children. Mutations in the mtDNA

[6] *Lactic acidosis* is a medical condition characterized by the buildup of *lactate* (especially L-*lactate*) in the body, with formation of an excessively low pH in the bloodstream. It is a form of metabolic *acidosis*, in which excessive *acid* accumulates due to a problem with the body's oxidative metabolism.
[7] National Center for Biotechnology Information https://www.ncbi.nlm.nih.gov/books/NBK1233/
[8] https://www.sciencedirect.com/science/article/pii/S0002929707614888
[9] Gorman et al (ANN NEUROL 2015;77:753–759)

gene, MT-TL1, cause MELAS. The majority of affected individuals with classic symptoms (about 80%) have a specific mutation, **A3243G** (relevant for NeuroVive). Other rare mtDNA mutations in the MT-TL1 gene, T3271C and A3252G, and in 9 other mtDNA genes, are also associated with MELAS.

Currently there is **no treatment** for MELAS spectrum disorders.

Kearns-Sayre Syndrome, or KSS

Kearns-Sayre Syndrome is a rare inborn error of metabolism characterized by progressive external ophthalmoplegia (PEO) or weakness of the eye muscles, a slowly progressive inability to move the eyes and the eyebrows. Onset is before the age of 20.

Prevalence is estimated at 1-3 per 100,000[10]. The disease progresses slowly, over decades, with symptoms slowly worsening and new symptoms appearing. A very few cases of Pearson syndrome have progressed into KSS.

The disease often **presents in childhood** and is caused by deletions of a large portion of mtDNA, resulting in the loss of genes involved in the oxidative phosphorylation pathway. Deletions are heteroplasmic (i.e. a single cell can harbour both deleted and normal DNA molecules). Symptoms only appear if the proportion of abnormal DNA is high. The abnormal DNA threshold depends on the organ (for example, about 60% for the skeletal striated muscle).

The treatment of KSS is supportive. In those with high-grade heart block, a permanent pacemaker/implantable cardioverter-defibrillator device may improve the prognosis. Hearing aids may be given to those with sensorineural deafness. Supplementation with coenzyme Q10 has been beneficial in some cases. Ophthalmologic manifestations may be treated with surgery but there is a high risk of recurrence and possible ocular complications.

The prognosis essentially depends on the number of organs involved and on the proportion of the abnormal mtDNA in each organ. In many cases, life expectancy can be normal with the appropriate support.

Pearson syndrome

Deletions identical to those in KSS may be found in Pearson syndrome. This is characterized by sideroblastic anaemia, often accompanied by a deficiency of neutrophils and platelets, pancreatic dysfunction, and abnormal liver function, but no neurological symptoms (Pearson et al., 1979). The onset of Pearson syndrome often comes during **early childhood**. This syndrome is multisystemic, with reports of patients presenting with short stature, liver failure, proximal tubulopathy, skin rash, and chronic diarrhoea.

Detections occurring as early as embryogenesis

The widespread finding of the deletion in multiple tissues suggests the deletion event occurs early during embryogenesis. The clinical course of this syndrome is severe, with most children with it suffering an early death. However, some patients do survive and subsequently develop Kearns-Sayre syndrome at a later age. The course of the disease follows the

[10] U.S. National Library of Medicine https://ghr.nlm.nih.gov/condition/kearns-sayre-syndrome#statistics

correlation of the size of deletion, the heteroplasmy level in muscle, and the location of the deletion.

Low prevalence, no treatment

Prevalence is less than one in a million and there is **no specific treatment** of Pearson syndrome. Management is symptomatic and includes treatment of infectious episodes and of metabolic accidents, transfusion in the case of deep anaemia (sometimes associated with erythropoietin therapy), uptake of pancreatic extracts, and management of endocrine disorders. Death often occurs before the age of 3.

CPEO

Chronic Progressive External Ophthalmoplegia (CPEO) is a condition characterized by a loss of the muscle functions involved in eye and eyelid movement. Signs and symptoms tend to begin between the ages of 18 and 40 and commonly include weakness or paralysis of the muscles that move the eye (ophthalmoplegia) and drooping of the eyelids (ptosis).

General loss of muscle function and nerve damage

Sometimes, CPEO may be associated with other signs and symptoms. Some affected individuals also have general weakness of the skeletal muscles (myopathy), which may be especially noticeable during exercise. Muscle weakness may also cause difficulty swallowing. In these cases, the condition is referred to as "progressive external ophthalmoplegia plus" (PEO+). Additional signs and symptoms can include hearing loss caused by nerve damage in the inner ear (sensorineural hearing loss), weakness and loss of sensation in the limbs due to nerve damage (neuropathy), impaired muscle co-ordination (ataxia), a pattern of movement abnormalities known as Parkinsonism, or depression.[11]

Mutations of mtDNA cause

The most commonly identified genetic defect is a pathogenic mitochondrial DNA. CPEO can be caused by mutations in any of several genes, which may be in mtDNA or nuclear DNA. It has different inheritance patterns depending on the gene involved in the affected individual.

Low prevalence and supportive treatments

Prevalence[12] has been estimated at 3.39 per 100,000, or about 1 in 30,000 of the general population, and the **treatment** of ptosis includes surgical correction or using glasses with a "ptosis crutch" to lift the upper eyelids. Strabismus surgery can be helpful in carefully selected patients if diplopia (double vision) occurs.

Some individuals with a deficiency of coenzyme Q10 have CPEO as an associated abnormality. **Coenzyme Q10** is important for normal mitochondrial function. In individuals with this deficiency, supplemental coenzyme Q10 has been found to improve general neurologic function and exercise tolerance. However, coenzyme Q10 has not been shown to improve the ophthalmoplegia or ptosis in people who have isolated CPEO.

MERRF

MERRF (Myoclonic Epilepsy with Ragged Red Fibres) syndrome is mitochondrial encephalomyopathy characterized by myoclonic seizures.

[11] Genetic and Rare Diseases Information Center, US.
[12] https://iovs.arvojournals.org/article.aspx?articleid=2270685

MERRF syndrome is caused by **mutations in mtDNA**. More than 80% of individuals with MERRF syndrome carry the 8344A>G mutation in the lysine transfer RNA (*tRNA Lys*) gene (*MTTK*). Other mutations have been found in other transfer RNA genes or in the *MTND5* gene. They may be associated with MERRF/MELAS overlap syndrome, in which affected individuals also suffer from stroke-like episodes.

Patients usually present during adolescence or early adulthood with myoclonic epilepsy, sometimes with neurosensory deafness, optic atrophy, short stature, or peripheral neuropathy.

Prevalence and treatment

MERRF is a rare condition and its prevalence is unknown[13]. As with other mitochondrial encephalomyopathies, there is **no specific treatment** for MERRF syndrome. Seizures can be treated with conventional anticonvulsant therapies. In the absence of proper clinical trials, it is difficult to evaluate the effect of a proposed supportive treatment such as coenzyme Q10.

Prognosis

The prognosis for patients with MERRF syndrome is globally poor because of the progressive nature of the disease. However, the severity varies greatly. Some patients, mainly those with non-cerebral presenting symptoms, may have prolonged survival with relatively little handicap.

Leigh syndrome

Typical onset of the symptoms of Leigh syndrome occurs before the age of 12 months but, in rare cases, the disease may manifest during adolescence or even early adulthood. Loss of motor milestones, hypotonia with poor head control, recurrent vomiting, and a movement disorder are common initial symptoms. Pyramidal and extrapyramidal signs, nystagmus, breathing disorders, ophthalmoplegia, and peripheral neuropathy are often noted later. Epilepsy is relatively rare.

Leigh syndrome has multiple causes, all of which imply a defect in aerobic energy production. Most mutations are in the nuclear genome. The genes identified to-date encode either one of the sub-units of the pyruvate dehydrogenase (PDH) complex, one of the sub-units of respiratory complexes I or II, or a protein involved in the assembly of respiratory complex IV.

Rare disease, early onset, no treatment

Its prevalence at birth has been estimated at approximately 1 in 36,000 (Orpha.net). Diagnosis of the syndrome relies on biochemical investigations looking for the underlying defect in energy production.

There is **no treatment** for Leigh syndrome. Several different vitamins or cofactors, including vitamin B1 (thiamine), vitamin B2 (riboflavin) and coenzyme Q10, have been proposed and may be tried systematically. Their efficacy depends upon the underlying defect. The prognosis of Leigh syndrome is poor, with life expectancy reduced to only a few years for most patients.

[13] https://ghr.nlm.nih.gov/condition/myoclonic-epilepsy-with-ragged-red-fibers#statistics

Most patients with Leigh syndrome have mutations in nuclear DNA and about 20% of patients have mutations in mtDNA. Most genes associated with Leigh syndrome are involved in the process of energy production in mitochondria (oxidative phosphorylation). Five protein complexes are involved. Many of the gene mutations associated with Leigh syndrome disrupt the function of proteins in these complexes, how the complexes form, or additional steps related to energy production. Researchers believe that impaired oxidative phosphorylation may cause cells to die because they do not have enough energy. The death of brain cells likely contributes to the neurologic features of the condition, while the death of cells in other tissues may lead to additional symptoms in other parts of the body.[2]

No existing treatment and high unmet medical need

There are no existing treatments. The general symptom management consists of increasing energy levels within the cells, through lifestyle changes or supplements: some combination of vitamins and minerals are helpful, termed "mito-cocktails." Aerobic exercise has shown benefits in improving strength and lessening fatigue. Exercise programmes have been shown to improve the quality of life in many affected individuals. In rare instances, affected individuals have coenzyme Q10 deficiency and some of these individuals may respond to therapy with high doses of coenzyme Q10 supplementation.

Advantages of orphan drug designation

Briefly, orphan drug designation means that development costs are lower compared with non-orphan drugs, the development work can be carried out faster, and orphan drug candidates have a high chance of reaching the market. The pricing of orphan drugs is also highly attractive, and marketing is focused on a limited number of specialist centres. The longer exclusivity period means that a company will have longer to recuperate the development costs.

Government incentives

- Financial incentives. Tax credits and R&D grants have been made available and there are waivers for regulatory fees.
- A reduction in the number of patients needed for trials. In the US, orphan drugs require a median of 538 participants in phase III trials compared with a median of 1,491 participants for non-orphan drug trials.
- Accelerated development and market access. On average, clinical review times have been shortened by 18 months in the US and regulatory review periods by eight months.
- Extended exclusivity periods. Orphan drugs are allowed seven years of market exclusivity in the US, compared with five years for non-orphan drugs. In Europe and Japan, exclusivity is granted for 10 years.

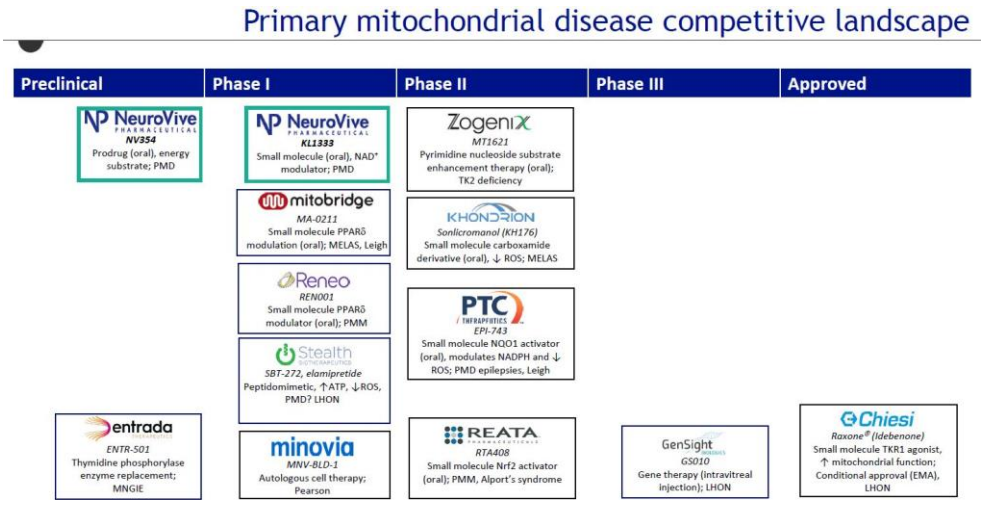
Mitochondrial disease pipeline: mainly early-stage projects

There are very few treatments in development for primary mitochondrial diseases. No FDA-approved drug is currently available for the treatment or cure of mitochondrial diseases, and all currently recommended non-FDA-approved treatment options are only aimed at alleviating symptoms. Although all new experimental drugs for the therapy of mitochondrial diseases

have the potential to substantially alleviate clinical symptoms, none has the potential to actually cure a particular mitochondrial disease permanently.

We have taken a closer look at NeuroVive's competitors in the primary mitochondrial disorders space.

Currently, there is only one small molecule approved in the mitochondrial space: Raxone (idebenone), a TKR1 agonist, which increases mitochondrial function and has been developed for the treatment of LHON (Leber's hereditary optic neuropathy). It was approved in 2015 and Chiesi Group in-licensed it from Santera with the rights to commercialise it worldwide except in the US and Canada.



Source: NeuroVive presentation

Entrada Therapeutics is a biotechnology company dedicated to transforming the treatment of devastating diseases through the intracellular delivery of biologics. It is developing ENTR-501, a novel thymidine phosphorylase enzyme replacement therapy for the treatment of mitochondrial neurogastrointestinal encephalomyopathy (MNGIE). The drug is engineered using Entrada's proprietary Endosomal Escape Vehicle (EEV^{TM}) technology and is in a preclinical stage.

Of special interest to our valuation is Khondrion's wholly owned lead asset, Sonlicromanol (previously known as KH176). The oral small molecule is a novel redox (ROS species) modulator with anti-inflammatory properties and belongs to a new class of drugs used to control oxidative and redox pathologies. Sonlicromanol safeguards lipid peroxidation-dependent cell death and counteracts inflammation. It has been granted orphan drug designation for MELAS-MIDD spectrum disorders and Leigh syndrome in Europe and for all inherited mitochondrial respiratory chain disorders in the US.

It is currently in phase II:

- The first IIa KHENERGY study, published in January 2019, showed consistent pharmacokinetic, safety, and tolerability data for Sonlicromanol, but the study failed to meet efficacy endpoints. Some positive effects in some exploratory outcomes supported further development.
- In January 2020, Khondrion commenced its phase IIb KHENERGYZE study of Sonlicromanol across Europe. The double-blind, randomised, placebo-controlled, 3-way cross-over study is recruiting adult patients with a specific genetically confirmed DNA mutation in the mitochondrial transfer RNALeu(UUR) (MT-TL1m.3243A>G). This

mutation is responsible for MELAS spectrum disorders, including MELAS and MIDD syndromes, and mixed phenotypes. Planning is also underway to investigate the compound's potential in children, with a paediatric study expected to start later in 2020.

While addressing the same genetic mutation as NeuroVive's compound, Sonlicromanol has a different mechanism of action.

Stealth Biotherapeutics saw its share fall 63% after its late-stage trial for elamipretide, a compound for the treatment of primary mitochondrial myopathy (a muscular disease caused by genetic mutations), failed to meet the study's primary efficacy endpoint. Prior to the top-line results, orphan drug bellwether Alexion had acquired an option to in-license elamipretide for primary mitochondrial disease indications for an up-front payment of USD 30m. Stealth is still developing the compound for LHON (currently in phase I), the most common mitochondrial optic neuropathy.

Another compound that Stealth is developing, SBT-272, is a novel peptidomimetic in pre-clinical stage for the treatment of neurodegenerative diseases involving mitochondrial dysfunction. SBT-272 has been shown to increase adenosine triphosphate (ATP) production and decrease levels of reactive oxygen species (ROS) in dysfunctional mitochondria in preclinical studies. SBT-272 demonstrates higher mitochondrial uptake, greater concentrations in the brain, and improved oral bioavailability relative to elamipretide, Stealth's first-in-class lead compound.

Minovia Therapeutics has designed and developed a personalised cell therapy platform to be applied to patients with KSS, LHON, MELAS, and IND, which is anticipated by the end of 2020. Mitochondria Augmentation Therapy (MAT) is a proprietary method of isolating mitochondria from white blood cells or placentas. Minovia describes a step-by-step process for replacing damaged with healthy mitochondria in the patient's hematopoietic stem cells. Healthy mitochondria introduced into the stem cells circulate around the body and then return to the bone marrow where they proliferate, for the organs that need them. The therapy is currently in phase I clinical trials.

Mitobridge is currently developing two compounds from its programme for modulation of PPARδ, a key regulator involved in improving muscle endurance and increasing mitochondrial fatty acid utilization. While it targets some of the same symptoms (mitochondrial myopathy), its mechanism of action is different from the NeuroVive compounds.

ASP1128 is a potentially first-in-class approach to treating acute kidney injury (AKI). The compound is believed to have protective effects on kidney cells that are under cellular stress in patients at increased risk of AKI following coronary artery bypass and/or valve (CABG/V) surgery. It does so by promoting fatty acid oxidation in the mitochondria. A proof of concept phase II study with ASP1128 is ongoing.

ASP0367 offers a mitochondrial-directed approach for the treatment of Duchenne Muscular Dystrophy (DMD) by increasing fatty acid oxidation and mitochondrial biogenesis in muscle cells. ASP0367 has successfully completed phase I clinical trials and is scheduled to enter a safety and efficacy study in DMD patients in early 2020.

Reneo is a clinical-stage pharmaceutical company focused on the development of therapies for patients with genetic mitochondrial diseases. Its lead compound is REN001, a small molecule and oral PPARgamma modulator with estimated clinical phase I completion in April 2020. The goal of the study is to determine the safety and tolerability of REN001 in patients with primary mitochondrial diseases. REN001 is an investigational clinical-stage compound

known to control a number of genes involved in mitochondrial activity, to improve cellular energy metabolism by enhancing mitochondrial function, and potentially to increase the number of mitochondria.

RTA 408 (omaveloxolone) is a small molecule Nrf2 activator that could potentially be beneficial for patients with Friedreich's ataxia. The treatment was being developed by **Reata Pharmaceuticals** in collaboration with AbbVie, but Reata reacquired global rights to develop and commercialize RTA 408 in October 2019.

The Paris-listed **GenSight Biologics** is developing a mitochondrial targeting sequence (MTS) to restore the function of NADH dehydrogenase resulting from a mutation of the ND4 gene in LHON. GenSight has exclusive access to MTS technology that permits the synthesis, translocation, internalization, and proper localization of the missing mitochondrial protein into the matrix of the mitochondrion. The **GS010** product candidate, using the MTS tech platform, is now in **late phase III trials**. The company plans to use its proprietary MTS technology platform to address other LHON mutations. Its next potential product candidate, GS011, targets the *ND1* gene mutation. GenSight also believes that the MTS technology platform could benefit non-ophthalmologic diseases that involve defects of the mitochondrion, such as neurodegenerative disorders.

Zogenix is investigating **MT1621** for the treatment of thymidine kinase 2 (TK2) deficiency, currently in trial registration.

PTC Therapeutics is currently developing **EPI-743 (Vincerenone)** for the treatment of mitochondrial disorders, Friedreich's ataxia, and Leigh syndrome. It is an antioxidant and NQ01 modulator. It is currently in **phase III for Leigh** and in phase II for Friedreich's ataxia, methylmalonic acidaemia, and mitochondrial disorders. PTC Therapeutics plans registrational trials for mitochondrial epilepsy and Friedreich's ataxia. EPI-743 is an orally administered drug that can cross the blood-brain barrier. It does not target or up- or down-regulate a particular enzyme, but instead is a co-factor for key enzymes involved in cellular metabolic control. It exerts its therapeutic effect by optimizing a series of electrochemical reactions in the mitochondria.

So far, with the exception of Raxone, none of these compounds have successfully passed efficacy tests.

Valuation

Our **valuation includes two assets** that NeuroVive plans to bring through clinical trials: the in-licensed **KL1333** and the internally developed **NV354**. We do not include NeuroSTAT and NV556 as out-licensing opportunities could prove elusive due to limited clinical data, challenging indications and, in the case of NV556, competition from more advanced projects. We also exclude the NVP015 project (out-licensed to BridgeBio) as it is still in very early development.

In our calculations, we assume that NeuroVive will have to raise capital in 2021 before efficacy tests for KL1333, and after obtaining IND from the FDA US.

KL1333

The exact **prevalence** of the disorders KL1333 targets is unknown. We base our patient population calculations on Gorman et al (Annals of Neurology 2015;77:753–759), a study that puts the prevalence of primary mitochondrial diseases of the Melas-MIDD spectrum at

around 3.5/100,000. Other studies suggest the prevalence is much higher 16.3/100,000. We have worked with a prevalence of 4.5/100,000.

We calculate the patient population to be around 14,805 in the US and 15,311 in Europe's 6 largest countries (EU6). We let this grow at the general population growth rate and assume that around **60%** of cases with the disease will be **diagnosed**. Of these, we assume that **80%** will **receive treatment**. We arrive at around 7,106 annually treated patients in the US and 7,349 in Europe6 - around 14,455 in total.

These patients must be treated daily with an oral dose of KL1333. We assume that, once approved, this chronic treatment will have an **annual price of USD 100,000** in the **US** and **USD 60,000 in EU6**.

We assume royalty payments of between 8% and 11% to Yungjin Pharm until patent protection runs out in 2034. Our calculations include milestone payments to Yungjin Pharm of USD 1m (end phase I), USD 12m (upon successful clinical development potentially in 2024) and USD 42m upon market and reimbursement approval in 2025.

NV354

NeuroVive's second asset is NV354 for the treatment of Leigh Syndrome, typically a pediatric disorder. The compound is expected to enter clinical development in 2021. If approved, it should reach the market in 2027.

The **prevalence** of Leigh Syndrome is about 27/1,000,000, or about 8,883 in the US and 9,187 in EU6.

In our calculations we assume that about 70% of children born with this disorder will be diagnosed and 80% of these will receive this oral treatment daily. Furthermore, we conservatively assume annual treatment prices of around USD 90,000 in the US and USD 50,000 in Europe.

Base Case

Including the new number of shares after the issue proposed on February 19 (278.9m in total), our risk-adjusted net present value (rNPV) sum-of-the-parts (SOTP) analysis puts NeuroVive's base case intrinsic value per share at **SEK 1.2**.

The assumptions underlying our base case are:

- KL1333 succeeds in efficacy and is approved in 2025
- NV354 successfully passes Phase I clinical trials in 2021 and is approved in 2027
- Sales and marketing through NeuroVive's own specialised salesforce
- We have not included future share issues in our calculation

Likelihood of Approval (LOA) for KL1333 and NV354 of only 10% and 8.6%, respectively. Both compounds are early stage and historically, the average LOA for drugs targeting CNS or primary mitochondrial disorders is below 10%, while LOA of orphan drugs is typically between 6.2% and 13.6%[14].

With the assumption that NeuroVive sells the medicines through its own specialised sales organization and not out-license them, we factor in an EBIT margin of 50% for both assets.

[14] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6409418/

We risk-adjust forecasted cashflow with the probability of passing each clinical phase (90% for Phase I, 20% phase II, 70% phase III and 80% probability for NDA), as well as a likelihood of succeeding in efficacy tests of around 20% and a weighted average cost of capital (WACC) of 16%.

Base Case

Project	Indication	Peak sales	Launch	NPV	NPV per share
KL1333	Mitochondrial diseases	180	2025	210	0.75
NV354	Leigh	368	2027	81	0.29
Technology value (EV)				292	1.05
Net cash				123	0.44
General and admin costs				-78	-0.28
NPV				**336**	**1.21**
Number of shares					278.9

Source: Redeye Research

In the base case, the rNPV of KL1333 is SEK 210m and for NV354 at SEK 81m. Total NPV of SEK 336m results in a fair value of SEK 1.2 per share.

As noted, the stock offers upside to our base case value. But we emphasize at the same time that it is a very high-risk case and we anticipate no strong catalysts in the near term.

Bull Case

Our bull case factors in a 56% LOA for KL1333, assuming successful development through Phase II in 2021.

In this scenario we assume NeuroVive raises capital for further development prior to read-out of a Phase II trial for KL1333. This means on the one hand a lower subscription price per share and higher dilution, but on the other secured funding for future projects - even if KL1333 fails.

In this calculation, we also raise the probability of NV354 in phase I to 100%, ie assume that the compound will successfully pass safety, tolerability and pharmacokinetics.

We assume NeuroVive will need to raise SEK 150m before efficacy test results are clear, probably at two separate capital raisings. We calculate the new number of shares at 428.9m. SEK 150m cash is added before expenses (150m new shares, on an average SEK 1 per share, NeuroVive spends around SEK 70m until then), resulting in a new cash position of SEK 200m. The price of SEK 1 per share will be motivated by successful Phase Ib (KL1333) and receiving IND from the FDA US.

We base our calculations on the new number of shares and the new capital added:

Bull Case Valuation

Project	Indication	Peak sales	Launch	NPV	NPV per share
KL1333	Mitochondrial diseases	180	2025	2481	5.78
NV354	Leigh	430	2027	141	0.33
Technology value (EV)				2622	6.11
Net cash				200	0.47
General and admin costs				-78	-0.18
NPV				**2743**	**6.40**
Number of shares					428.9

Source: Redeye Research

In our bull case KL1333's rNPV increases to SEK 2,475m. The total rNPV of NeuroVive is SEK 2,736m or more than SEK 6 per share, after dilution.

Bear Case

In our bear case KL1333 fails to meet efficacy endpoints in Phase II and its rNPV becomes 0. For continued development of NV354 we model a rights issue of 500m new shares at a price of SEK 0.30 per share.

In this scenario we calculate a value of SEK 194m based on NV354 advancing into clinical stage This corresponds to an NPV per share of SEK 0.3 after assumed dilution.

Bear Case

Project	Indication	Peak sales	Launch	NPV	NPV per share
KL1333	Mitochondrial diseases	140	2025	0	0.00
NV354	Leigh	430	2027	194	0.25
Technology value (EV)				194	0.25
Net cash				123	0.16
General and admin costs				-78	-0.10
NPV				**239**	**0.31**
Number of shares					778.9

Source: Redeye Research

In conclusion, the value of NeuroVive depends on the main compound KL1333. If the compound succeeds, the company can be worth quite a lot, if it fails, it will be very difficult to raise more capital and the value could potentially gravitate to zero.

Appendix 1

NeuroVive's non-core projects

NeuroSTAT is a molecule that NeuroVive is looking to partner and fund with non-dilutive soft money. NeuroSTAT has received orphan drug designation in the US and Europe as well as IND approval and fast track designation from the US's FDA for clinical development in the US and EU alone. It is developed for the management of moderate to severe traumatic brain injury (TBI), which affects three million people each year in the US alone. The clinical studies have so far demonstrated that NeuroSTAT is safe and well-tolerated. The studies also confirmed that the drug passes the blood-brain barrier in patients with TBI and showed signs of clinical effectiveness, as measured by 4 biomarkers (GFAP, NF-L, Tau, and UCH-L)[15].

NeuroVive has a ready design for the planned phase II efficacy trials, an open IND, a patent, and orphan drug and fast track designations. Efficacy trials will begin when the company has found a suitable partner.

The FDA has approved the following phase II efficacy design: a placebo-controlled, randomised study with novel biomarker/imaging design in collaboration with top KOL networks in the US, including 75-105 patients, 17 high-end clinical sites, 5 countries, and 6 months' follow-up. This will be followed by 2-3 years in a phase II study in a large patient population.

NeuroSTAT is consequently 6-7 years away from the market (possible approval by 2026) and NeuroVive is looking for a partner to invest in the phase II study and share the risks and rewards.

Mechanism of action

NeuroSTAT's formulation is made using a patented non-allergenic lipid emulsion to keep lipophilic drug CsA in solution. In 2010, NeuroSTAT received orphan drug status from the FDA and European Commission for the treatment of moderate and severe TBI. Cyclosporine inhibits activation of the mitochondrial permeability transition pore, a key event in TBI pathology. This way the mitochondria can withstand more stress and it is possible to prevent secondary neuronal injury and cell death. NeuroSTAT is a new intravenous cyclosporine formulation for the treatment of TBI, a novel and proprietary lipid emulsion that prevents chromophore-associated anaphylaxis. All its ingredients are registered for human use and manufactured to GMP standards by Fresenius-Kabi in a ready-to-use and easy-to-use solution.

Clinical development status

The compound has orphan drug designation in the US and Europe, and fast track designation in the US. Clinical phase Ib/IIa pharmacokinetics and a safety study were conducted in May 2017 in the Copenhagen Head Injury Cyclosporin (CHIC) Study[16], which measured microdialysis in the brain parenchyma and protein biomarkers of brain injury in the cerebrospinal fluid (CSF).

The study included 16 adult patients with severe TBI (Glasgow Coma Scale 4–8), all of whom received an initial loading dose of 2.5 mg/kg followed by a continuous infusion for 5 days.

[15] https://www.liebertpub.com/doi/10.1089/neu.2018.6369
[16] https://www.liebertpub.com/doi/10.1089/neu.2018.6369

The first 10 patients received an infusion dosage of 5 mg/kg/day, whereas the subsequent 6 patients received 10 mg/kg/day.

No mortality was registered within the study duration, and the distribution of adverse events was similar between the two treatment groups. Pharmacokinetic analysis of CSF confirmed dose-dependent brain exposure.

Between- and within-patient variability in blood concentrations was limited, whereas CSF concentrations were more variable. The four biomarkers – glial fibrillary acidic protein, neurofilament light, Tau, and ubiquitin carboxy-terminal hydrolase L1 – showed consistent trends of decreasing during the 5-day treatment period, whereas the samples taken on the days after the treatment period showed higher values in the majority of patients.

The study concluded that cyclosporin is safe and well-tolerated, confirmed that it can pass the blood-brain barrier in a TBI population, and provided an initial biomarker-based signal of efficacy.

Another study with NeuroSTAT at the University of Pennsylvania (PENN) was aimed at "evaluating the efficacy of a novel and cremophor/kolliphor EL–free lipid emulsion formulation of cyclosporine in a translational large animal model of TBI"[17].

Traumatic brain injury

Disrupted brain function

A TBI is defined as a blow to the head or a penetrating head injury that disrupts the normal function of the brain. TBI can occur when the head suddenly and violently hits an object or when an object pierces the skull and enters the brain tissue. Most often, TBI is caused by a traffic accident or a fall. This is the first stage.

Symptoms of TBI can be mild, moderate, or severe, depending on the extent of damage to the brain. Mild cases may result in a brief change in mental state or consciousness, while severe cases may result in extended periods of unconsciousness, coma, or even death.

TBI is the cause of about 2.5 million emergency department visits, 282,000 hospitalizations, and 56,000 deaths in the US, most often on account of mild TBI. TBI contributes to 30% of all injury-related deaths in the US. These numbers are thought to significantly underestimate the burden of TBI, since they do not include patients who did not seek medical attention, received ambulatory care, were seen at Veterans Affairs centres, or are in the military.

Cascading biochemical reactions cause further damage

In the immediate aftermath of the initial shock, the trauma triggers a series of cascading intra-cellular biochemical reactions that can continue for days, weeks, or even months. The most severe damage occurs during this period, and the outcome is unpredictable. NeuroSTAT has been developed to address this second wave of brain damage when the injury continues to evolve and worsen.

Major problem for the US military

The incidence of TBI varies greatly across countries and regions, from 811 per 100,000 each year in New Zealand to 7.3 per 100,000 a year in Western Europe. The highest rates of TBI were observed in older adults (≥75 years; 2,232 per 100,000 population), very young (0 to 4 years; 1,591 per 100,000), and young adults (15 to 24 years; 1,081 per 100,000). TBI is a

[17] https://www.liebertpub.com/doi/10.1089/neu.2018.5706

major problem for the US military; the Department of Defense reports that in 2000-2017, 375,230 military personnel suffered TBI.

A study in Norway found that the overall incidence of mild TBI in people between the ages of 16 and 59 was 302 per 100,000 person-years (95% confidence interval 281-324). The incidence rate was highest in the 16-20 years age group, where rates were 835 per 100,000 person-years in males and 726 in females.[18] Annual direct and indirect TBI costs are estimated at USD 48-56 billion.

Other non-core projects

NV556 is being developed for the treatment of fibrosis in patients with non-alcoholic steatohepatitis (NASH). This disease is estimated to affect 3-5% of the global population, implying a large amount of documentation in all clinical development phases and meaning that moving through clinical trials to registration will require significant resources. NeuroVive is now looking for a partner to progress this project through clinical trials and to market.

The NVP015 project is in the pre-clinical stage. It is based on a NeuroVive innovation in which the body's own energy substrate, succinate, is made available in a cell via a prodrug technology. A prodrug is an inactive drug that is first activated on entering the body through the transformation of its chemical structure. NeuroVive has selected a lead candidate from the programme for development in Leigh syndrome, NV354, and it has out-licensed other parts of the chemistry programme to BridgeBio/Fortify Therapeutics for the development of local treatment of LHON (Leber's hereditary optic neuropathy).

NVP025 for the treatment of muscle myopathy (muscle weakness) is at the early pre-clinical stage. In early 2018, a joint study with Karolinska Institute demonstrated that the project's model compound has favourable effects in countering disease progression of mitochondrial myopathy. It is currently in ongoing dose-response studies. The goal for 2020 is to continue pharmacology studies and prepare for the formal preclinical development of drug candidates.

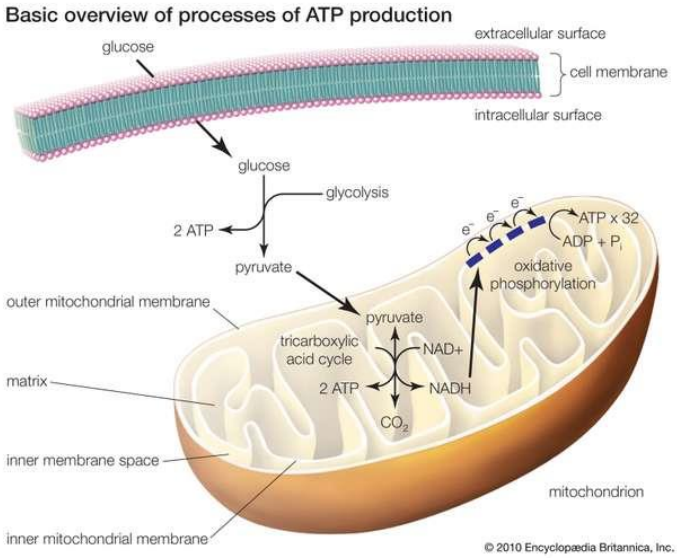
[18] https://www.frontiersin.org/articles/10.3389/fneur.2019.00638/full

Appendix 2

The outer mitochondrial membrane is freely permeable to small molecules and contains special channels capable of transporting large molecules. In contrast, the inner membrane is far less permeable, allowing only very small molecules to cross into the gel-like matrix that makes up the organelle's central mass. The matrix contains the DNA of the mitochondrial genome and the enzymes of the tricarboxylic acid (TCA) cycle (also known as the citric acid cycle, or Krebs cycle), which metabolizes nutrients into by-products that the mitochondrion can use for energy production.

Mitochondria contain the rate-limiting enzymes for pyrimidine biosynthesis (dihydroorotate dehydrogenase) and heme synthesis (d-aminolevulinic acid synthase) required to make haemoglobin. In the liver, mitochondria detoxify ammonia in the urea cycle. Mitochondria are also required for cholesterol metabolism, for oestrogen and testosterone synthesis, for neurotransmitter metabolism, and for free radical production and detoxification.

A person inherits mitochondria from their mother, because the mother's egg cell donates the majority of cytoplasm to the embryo. The mitochondria inherited from the father's sperm are usually destroyed. Inherited mitochondrial DNA is based on the mitochondria that happen to be in the egg.



Mitochondrial DNA

Although most DNA is packaged in chromosomes within the nucleus, mitochondria also have a small amount of their own DNA. This genetic material is known as mitochondrial DNA or mtDNA.

In humans, mtDNA spans about 16,500 DNA building blocks (base pairs), representing a small fraction of the total DNA in cells.

There are 3,000 genes involved in the production of mitochondria, yet mitochondrial DNA encodes just 37 of these genes; the remaining genes are encoded in the cell nucleus and the resultant proteins are transported to the mitochondria. Only about 3% of the genes necessary to make a mitochondrion (100 of the 3,000) are allocated for making ATP. More than 95% (2,900 of 3,000) are involved with other functions tied to the specialized duties of the

differentiated cell in which it resides. These duties change as a person develops from embryo to adult, and as tissues grow, mature, and adapt to the postnatal environment.

For a more detailed explanation on how the mitochondria works, see attachment 3.

Mitochondrial DNA is highly susceptible to mutations, largely because it does not possess the robust DNA repair mechanisms common to nuclear DNA. In addition, the mitochondrion is a major production site for reactive oxygen species (ROS; or free radicals) due to the high propensity for aberrant release of free electrons.

While several different antioxidant proteins within the mitochondria scavenge and neutralize these molecules, some ROS may inflict damage on mtDNA. In addition, certain chemicals and infectious agents, as well as alcohol abuse, can damage mtDNA. In the latter instance, excessive ethanol intake saturates detoxification enzymes, causing highly reactive electrons to leak from the inner membrane into the cytoplasm or into the mitochondrial matrix, where they combine with other molecules to form numerous radicals.

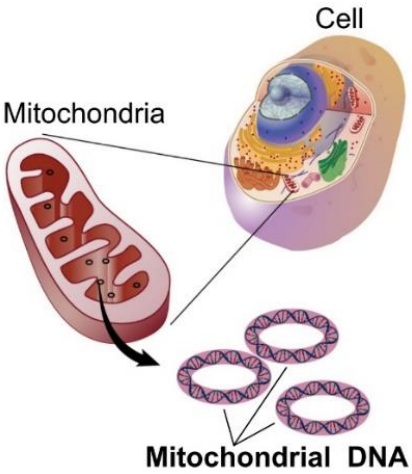


Image by National Human Genome Research Institute

Appendix 3

Phase Ia/b study design

The goal of the clinical development stage of KL1333 intends to record the use of chronic oral treatment in genetic primary mitochondrial disorders, in particular the MELAS-MIDD and PEO-KSS spectrums.

KL1333 is in clinical trials in the UK for safety, tolerability, and pharmacokinetics. NeuroVive has completed the first stage of the phase Ia/b trial in healthy volunteers. The second phase of this study is with patients with genetically confirmed primary mitochondrial disease.[19]

The primary goal of phase Ia is to evaluate the safety, tolerability, pharmacokinetics, and pharmacodynamics of KL1333. This is a double-blind, randomised, placebo-controlled, single and multiple oral dose studies conducted in 3 parts: A, B, and C.

Parts A and B include healthy volunteers only and will be completed before part C, when the study with patients with the primary mitochondrial disease will be initiated. The starting dose in the first cohort of part A will be 25 mg. The dose level in the additional cohorts will be decided following a review of the data of the previous cohorts.

Part A has been completed: 8 healthy subjects have been studied in a single cohort (Group A1). Potential subjects will be screened to assess their eligibility to enter the study within the 28 days prior to the first dose administration. Subjects will participate in 2 treatment periods, fasting or after consuming a standard high-fat breakfast. For each treatment period, subjects will reside at the phase I clinical site from days 1 to 3 (48 hours post-dose). Subjects will return to the clinical site for outpatient visits on days 4 and 5. There will be at least a 10-day washout between doses. Additional single-dose cohorts may be enrolled based on data obtained from either parts A or B.

Part B has been completed: 16 healthy subjects have been studied in 2 cohorts (Groups B1 and B2), with each cohort consisting of 8 subjects. Following a review of safety, tolerability, and PK data, up to 3 additional dose cohorts of healthy subjects may be added to further explore the PK, safety, and tolerability of KL1333. Potential subjects will be screened to assess their eligibility to enter the study within the 28 days prior to the first dose administration. All subjects will participate in 1 treatment period and will reside at the phase I clinical site from days -1 to 12 (48 hours after final dose). Subjects will return to the clinical site for outpatient visits on days 13 and 14. On day 1, 6 subjects will be randomised to receive KL1333 and 2 subjects will be randomised to receive placebo. Subjects will return for a follow-up visit on day 15, 5 days after their final dose.

Part C is also completed: A total of 8 patients diagnosed with any mitochondrial disease will be enrolled in this part of the study. Part C may start after the dose selection conference has been completed for the final cohort of part B, at a daily dose no higher than the highest well-tolerated dose in part B. Potential study patients will be screened to assess their eligibility to enter the study within the 35 days prior to the first dose administration. Patients will reside at the clinical site from days -1 to 2 and days 10 to 11 and return to the clinical site for outpatient visits on days 4 and 8. It is planned for patients to receive the study drug once daily on days 1 to 10. Patients will return for a follow-up visit on day 15, 5 days after their final dose. (www.clinicaltrials.gov)

[19] https://clinicaltrials.gov/ct2/show/NCT03888716

Exploratory biomarkers to be used in efficacy tests

This study includes exploratory biomarkers. The phase I study is relatively short at 10 days, too short to expect to see a clear pharmacological effect, but the biomarker information from the ongoing study will be helpful for successful implementation in the upcoming phase II efficacy study. Two of the assessed biomarkers, GDF15 and FGF21, are currently receiving a lot of attention in the mitochondrial scientific community for use in primary mitochondrial disease, both for aiding in diagnosis and following disease progression. In pre-clinical studies, KL1333 has demonstrated a reduction of these biomarkers.

Appendix 4

Management team

Erik Kinnman, CEO

Born in 1958, Erik Kinnman has held a number of senior leadership positions in biopharmaceutical companies such as AstraZeneca and SOBI. His expertise and experience include clinical development, business strategy, business development, and investor relations. Erik also has experience in the financial sector.

Education: Executive MBA from the Stockholm School of Economics, PhD and Associate Professorship at Karolinska Institutet. He is an MD, board-certified in Neurology and Pain Management. Chairman of the Board in Kinnman Solutions AB.

Employed at NeuroVive since 2016.

No of shares: 400,298

Catharina Jz Johansson, CFO and Vice President of Investor Relations

Born in 1967, Catharina Jz Johansson's experience includes serving as interim CFO for medical device company Cellavision (listed on Nasdaq Stockholm) and as Accounting Manager for Bong and Alfa Laval Europe. She holds an MSc in Business and Economics.

Employed at NeuroVive since 2013.

No of shares: 60,000

Eskil Elmér, CSO and Vice President of Discovery

Born in 1970, Eskil Elmér is an associate professor of experimental neurology at Lund University (Sweden) and group leader of the Mitochondrial Medicine lab at the Department of Clinical Neurophysiology. Dr Elmér is patentee and co-founder of both Maas Biolab, LLC and NeuroVive Pharmaceutical AB, as well as CSO of NeuroVive, with overall responsibility for pre-clinical research. In addition, Eskil Elmér is a practicing physician in the department of clinical neurophysiology at Skåne University Hospital in Lund, Sweden.

Employed at NeuroVive since 2000.

No shares: 577,487 privately owned (including family) and 17.09% of Maas Biolab, which owns 4.2% of NeuroVive.

Magnus Hansson, CMO and Vice President of Preclinical and Clinical Development

Born in 1976, Magnus Hansson has extensive experience in mitochondrial medicine. He has been Senior Scientist at NeuroVive since 2008 and serves as a consultant physician and associate professor in medical imaging and physiology at Skåne University Hospital. Dr Hansson has overall responsibility for the company's pre-clinical and clinical development programmes. He holds a PhD in experimental brain research from Lund University and has authored more than 30 scientific publications and 10 patent applications.

Employed at NeuroVive since 2008.

No of shares: 414,339 (including family)

Mark Farmery, Vice President of Business Development

Born in 1969, Mark Farmery is a senior executive with more than 15 years' experience in biopharma business development from Karolinska Institutet Innovations AB, AstraZeneca, and Karo Bio AB. He has also managed research teams and led specific projects in the fields of Alzheimer's disease modification and protein modification and misfolding at Karolinska Institutet and the Universities of Gothenburg and Manchester. Dr Farmery received his BSc in

Biomedical Sciences (Microbiology) from the University of Bradford and his PhD in Biochemistry and Molecular Microbiology from the University of Leeds.

Employed at NeuroVive since 2017.

No of shares: 13,225

INCOME STATEMENT	2018	2019	2020E	2021E	2022E
Net sales	0	0	0	0	0
Total operating costs	-69	-80	-53	-92	-82
EBITDA	-69	-79	-53	-92	-82
Depreciation	0	0	-3	0	0
Amortization	-5	2	0	-2	-1
Impairment charges	0	0	0	0	0
EBIT	-73	-77	-56	-94	-84
Share in profits	0	0	0	0	0
Net financial items	0	0	0	0	0
Exchange rate dif.	0	0	0	0	0
Pre-tax profit	-73	-77	-56	-94	-84
Tax	0	0	0	0	0
Net earnings	-73	-77	-56	-94	-84

BALANCE SHEET	2018	2019	2020E	2021E	2022E
Assets					
Current assets					
Cash in banks	26	58	67	131	50
Receivables	0	0	0	0	0
Inventories	0	0	0	0	0
Other current assets	3	1	1	1	1
Current assets	29	60	69	132	51
Fixed assets					
Tangible assets	0	1	1	2	2
Associated comp.	13	13	13	13	13
Investments	0	0	0	0	0
Goodwill	0	0	0	0	0
Cap. exp. for dev.	0	0	0	0	0
O intangible rights	73	75	77	73	72
O non-current assets	24	0	0	0	0
Total fixed assets	110	89	91	88	87
Deferred tax assets	0	0	0	0	0
Total (assets)	139	148	159	220	138
Liabilities					
Current liabilities					
Short-term debt	0	0	0	0	0
Accounts payable	17	20	0	0	0
O current liabilities	1	1	20	25	27
Current liabilities	18	20	20	25	27
Long-term debt	0	0	0	0	0
O long-term liabilities	0	0	0	0	0
Convertibles	0	0	0	0	0
Total Liabilities	18	21	20	25	27
Deferred tax liab	0	0	0	0	0
Provisions	0	0	0	0	0
Shareholders' equity	121	128	139	195	111
Minority interest (BS)	0	0	0	0	0
Minority & equity	121	128	139	195	111
Total liab & SE	139	148	159	220	138

FREE CASH FLOW	2018	2019	2020E	2021E	2022E
Net sales	0	0	0	0	0
Total operating costs	-69	-80	-53	-92	-82
Depreciations total	-5	2	-3	-2	-2
EBIT	-73	-77	-56	-94	-84
Taxes on EBIT	0	0	0	0	0
NOPLAT	-73	-77	-56	-94	-84
Depreciation	5	-2	3	2	2
Gross cash flow	-69	-79	-53	-92	-82
Change in WC	5	3	0	5	2
Gross CAPEX	-4	24	-5	1	-1
Free cash flow	-68	-52	-58	-86	-81

CAPITAL STRUCTURE	2018	2019	2020E	2021E	2022E
Equity ratio	87%	86%	87%	88%	80%
Debt/equity ratio	0%	0%	0%	0%	0%
Net debt	-26	-58	-67	-131	-50
Capital employed	82	56	58	51	48
Capital turnover rate	0.0	0.0	0.0	0.0	0.0

GROWTH	2018	2019	2020E	2021E	2022E
Sales growth	-81%	2,580%	-97%	0%	0%
EPS growth (adj)	-20%	-48%	-52%	9%	-11%

DCF VALUATION	
WACC (%)	16.0 %
Fair value e. per share, SEK	1.2
Share price, SEK	0.8

PROFITABILITY	2018	2019	2020E	2021E	2022E
ROE	-59%	-62%	-42%	-56%	-55%
ROCE	-59%	-62%	-42%	-56%	-55%
ROIC	-84%	-94%	-99%	-161%	-166%

DATA PER SHARE	2018	2019	2020E	2021E	2022E
EPS	-0.80	-0.41	-0.20	-0.22	-0.20
EPS adj	-0.80	-0.41	-0.20	-0.22	-0.20
Dividend	0.00	0.00	0.00	0.00	0.00
Net debt	-0.28	-0.31	-0.24	-0.31	-0.12
Total shares	91.70	186.00	279.00	429.00	429.00

VALUATION	2018	2019	2020E	2021E	2022E
EV	112.6	193.2	153.0	89.3	170.5
P/E	-1.9	-3.3	-3.9	-3.6	-4.0
P/E diluted	-1.9	-3.3	-3.9	-3.6	-4.0
P/Sales	Na	Na	Na	Na	Na
EV/Sales	Na	Na	Na	Na	Na
EV/EBITDA	-1.6	-2.4	-2.9	-1.0	-2.1
EV/EBIT	-1.5	-2.5	-2.7	-1.0	-2.0
P/BV	1.1	2.0	1.6	1.1	2.0

SHARE PERFORMANCE		GROWTH/YEAR	18/20E
1 month	-35.8 %	Net sales	-10.6 %
3 month	-37.8 %	Operating profit adj	-12.5 %
12 month	-	EPS, just	-49.9 %
Since start of the year	-41.0 %	Equity	7.3 %

SHAREHOLDER STRUCTURE %	CAPITAL	VOTES
Avanza Pension	9.5 %	9.5 %
John Fällström	3.9 %	3.9 %
Rothesay Ltd	3.6 %	3.6 %
Danske Bank International S.A.	3.6 %	3.6 %
Nordnet Pensionsförsäkring	2.5 %	2.5 %
Euroclear Bank S.A/N.V	2.4 %	2.4 %
Aras Atroshi	2.2 %	2.2 %
Maas BioLab LLC	2.1 %	2.1 %
Handelsbanken Liv Försäkring AB	1.6 %	1.6 %
Swedbank Försäkring	1.3 %	1.3 %

SHARE INFORMATION	
Reuters code	NVP.st
List	small cap
Share price	0.8
Total shares, million	279.0
Market Cap, MSEK	220.4

MANAGEMENT & BOARD	
CEO	Erik Kinnman
CFO	Catharina Johansson
Chairman	David Laskow-Pooley

FINANCIAL INFORMATION	
Q1 report	May 20, 2020
Q2 report	August 21, 2020
Q3 report	November 20, 2020
FY 2020 Results	February 19, 2021

ANALYSTS	Redeye AB
Gergana Almquist	Mäster Samuelsgatan 42, 10tr
gergana.almquist@redeye.se	111 57 Stockholm
Niklas Elmhammer	
niklas.elmhammer@redeye.se	

Redeye Rating and Background Definitions

Company Quality

Company Quality is based on a set of quality checks across three categories; PEOPLE, BUSINESS, FINANCE. These are the building blocks that enable a company to deliver sustained operational outperformance and attractive long-term earnings growth.

Each category is grouped into multiple sub-categories assessed by five checks. These are based on widely accepted and tested investment criteria and used by demonstrably successful investors and investment firms. Each sub-category may also include a complementary check that provides additional information to assist with investment decision-making.

If a check is successful, it is assigned a score of one point; the total successful checks are added to give a score for each sub-category. The overall score for a category is the average of all sub-category scores, based on a scale that ranges from 0 to 5 rounded up to the nearest whole number. The overall score for each category is then used to generate the size of the bar in the Company Quality graphic.

People

At the end of the day, people drive profits. Not numbers. Understanding the motivations of people behind a business is a significant part of understanding the long-term drive of the company. It all comes down to doing business with people you trust, or at least avoiding dealing with people of questionable character.

The People rating is based on quantitative scores in seven categories:

- Passion, Execution, Capital Allocation, Communication, Compensation, Ownership, and Board.

Business

If you don't understand the competitive environment and don't have a clear sense of how the business will engage customers, create value and consistently deliver that value at a profit, you won't succeed as an investor. Knowing the business model inside out will provide you some level of certainty and reduce the risk when you buy a stock.

The Business rating is based on quantitative scores grouped into five sub-categories:

- Business Scalability, Market Structure, Value Proposition, Economic Moat, and Operational Risks.

Financials

Investing is part art, part science. Financial ratios make up most of the science. Ratios are used to evaluate the financial soundness of a business. Also, these ratios are key factors that will impact a company's financial performance and valuation. However, you only need a few to determine whether a company is financially strong or weak.

The Financial rating is based on quantitative scores that are grouped into five separate categories:

- Earnings Power, Profit Margin, Growth Rate, Financial Health, and Earnings Quality.

Redeye Equity Research team

Management

Björn Fahlén
bjorn.fahlen@redeye.se

Håkan Östling
hakan.ostling@redeye.se

Technology Team

Jonas Amnesten
jonas.amnesten@redeye.se

Henrik Alveskog
henrik.alveskog@redeye.se

Havan Hanna
havan.hanna@redeye.se

Kristoffer Lindström
kristoffer.lindstrom@redeye.se

Erika Madebrink
erika.madebrink@redeye.se

Fredrik Nilsson
fredrik.nilsson@redeye.se

Tomas Otterbeck
tomas.otterbeck@redeye.se

Eddie Palmgren
eddie.palmgren@redeye.se

Magnus Skog
magnus.skog@redeye.se

Oskar Vilhelmsson
oskar.vilhelmsson@redeye.se

Viktor Westman
viktor.westman@redeye.se

Linus Sigurdsson (Trainee)
linus.sigurdsson@redeye.se

Editorial

Eddie Palmgren
eddie.palmgren@redeye.se

Mark Siöstedt
mark.siostedt@redeye.se

John Hintze
john.hintze@redeye.se

Johan Kårestedt (Trainee)
johan.karestedt@redeye.se

Life Science Team

Gergana Almquist
gergana.almquist@redeye.se

Oscar Bergman
oscar.bergman@redeye.se

Anders Hedlund
anders.hedlund@redeye.se

Arvid Necander
arvid.necander@redeye.se

Erik Nordström
erik.nordstrom@redeye.se

Klas Palin
klas.palin@redeye.se

Jakob Svensson
jakob.svensson@redeye.se

Ludvig Svensson
ludvig.svensson@redeye.se

Disclaimer

Important information
Redeye AB ("Redeye" or "the Company") is a specialist financial advisory boutique that focuses on small and mid-cap growth companies in the Nordic region. We focus on the technology and life science sectors. We provide services within Corporate Broking, Corporate Finance, equity research and investor relations. Our strengths are our award-winning research department, experienced advisers, a unique investor network, and the powerful distribution channel redeye.se. Redeye was founded in 1999 and since 2007 has been subject to the supervision of the Swedish Financial Supervisory Authority.
Redeye is licensed to; receive and transmit orders in financial instruments, provide investment advice to clients regarding financial instruments, prepare and disseminate financial analyses/recommendations for trading in financial instruments, execute orders in financial instruments on behalf of clients, place financial instruments without position taking, provide corporate advice and services within mergers and acquisition, provide services in conjunction with the provision of guarantees regarding financial instruments and to operate as a Certified Advisory business (ancillary authorization).

Limitation of liability
This document was prepared for information purposes for general distribution and is not intended to be advisory. The information contained in this analysis is based on sources deemed reliable by Redeye. However, Redeye cannot guarantee the accuracy of the information. The forward-looking information in the analysis is based on subjective assessments about the future, which constitutes a factor of uncertainty. Redeye cannot guarantee that forecasts and forward-looking statements will materialize. Investors shall conduct all investment decisions independently. This analysis is intended to be one of a number of tools that can be used in making an investment decision. All investors are therefore encouraged to supplement this information with additional relevant data and to consult a financial advisor prior to an investment decision. Accordingly, Redeye accepts no liability for any loss or damage resulting from the use of this analysis.

Potential conflict of interest
Redeye's research department is regulated by operational and administrative rules established to avoid conflicts of interest and to ensure the objectivity and independence of its analysts. The following applies:

- For companies that are the subject of Redeye's research analysis, the applicable rules include those established by the Swedish Financial Supervisory Authority pertaining to investment recommendations and the handling of conflicts of interest. Furthermore, Redeye employees are not allowed to trade in financial instruments of the company in question, from the date Redeye publishes its analysis plus one trading day after this date.
- An analyst may not engage in corporate finance transactions without the express approval of management and may not receive any remuneration directly linked to such transactions.
- Redeye may carry out an analysis upon commission or in exchange for payment from the company that is the subject of the analysis, or from an underwriting institution in conjunction with a merger and acquisition (M&A) deal, new share issue or a public listing. Readers of these reports should assume that Redeye may have received or will receive remuneration from the company/companies cited in the report for the performance of financial advisory services. Such remuneration is of a predetermined amount and is not dependent on the content of the analysis.

Redeye's research coverage
Redeye's research analyses consist of case-based analyses, which imply that the frequency of the analytical reports may vary over time. Unless otherwise expressly stated in the report, the analysis is updated when considered necessary by the research department, for example in the event of significant changes in market conditions or events related to the issuer/the financial instrument.

Recommendation structure
Redeye does not issue any investment recommendations for fundamental analysis. However, Redeye has developed a proprietary analysis and rating model, Redeye Rating, in which each company is analyzed and evaluated. This analysis aims to provide an independent assessment of the company in question, its opportunities, risks, etc. The purpose is to provide an objective and professional set of data for owners and investors to use in their decision-making.

Redeye Rating (2020-03-03)

Rating	People	Business	Financials
5p	11	12	4
3p - 4p	94	71	30
0p - 2p	10	32	81
Company N	115	115	115

Duplication and distribution

CONFLICT OF INTERESTS

Gergana Almquist owns shares in the company : No

Niklas Elmhammer owns shares in the company : No