Intron Health

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08/01/2021

Abliva

INITIATION – Creating Energy to Save Lives

Abliva

Creating Energy to Save Lives

Abliva is an early-stage Swedish biotech with the potential to transform the treatment of primary mitochondrial diseases (PMD). The lead candidate, KL1333, has swathes of exciting pre-clinical data which demonstrates its effectiveness at treating cells with impaired mitochondria and compares favourably to the only licensed drug in the EU. The first-in-human phase I trial is ongoing with a readout expected in H121; if positive, Abliva intend to launch pivotal trials by year-end. Our valuation of SEK2.25/share only values the two lead assets and assumes sufficient equity is raised in 2021 to enable the company to remain cash positive to 2025. Two recent deals in this arena highlight Abliva's low valuation.

>$1bn Opportunity with Fast-to-Market Development Strategy

There are c.42k pts in the EU & US with PMDs that KL1333 can target. It has been granted orphan disease status and based on data seen thus far, Abliva has been able to agree a fast-to-market development strategy with the FDA that enables pivotal trials to start in H221.

H121 Catalyst Could Lead to Major Value Inflection

To date, the pre-clinical data has been excellent and gives us confidence that Abliva's upcoming P1 readout for KL1333 (H121) could provide a significant valuation uplift. This would allow them to raise money for the pivotal trial at a higher valuation than we assume in our NPV.

Precedent Transactions Highlight Abliva's Mis-pricing

In January 2018, Astellas acquired Mitobridge for $225m plus another potential $225m. Mitobridge is at a similar stage to Abliva. In addition, just last month, Reneo Pharma raised $95m in a series B and is in phase II for PMM. At just ~$30m mkt cap, Abliva looks clearly mis-priced to us.

We see ~3x Upside to the Valuation Despite High Risk-Adjustment

Our NPV of the 2 lead assets is heavily risk-adjusted to just 20% of the potential sales we forecast. If we de-risk our sales, the upside would be 17x the current valuation, including the dilutive effects of a ~$50m raise.

Sponsored Research

Price: SEK0.76
Target Price: SEK2.25

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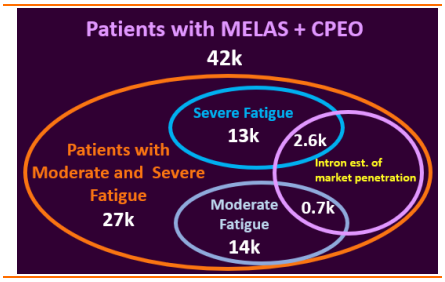
Rich catalyst timeline

Timing	Event
H121	KL1333 phase Ib readout
H121	KL1333 IND filing
H121	NV345 preclinical safety studies end
H221	KL1333 pivotal study start
H221	NV354 phase I start

Source: Company reports

Target Population for KL1333 (Intron)



Source: Intron Health estimates

Summary Financials

	20E	21E	22E	23E
Sales (SEKm)	0.3	0.3	0.3	0.3
EPS (SEK)	-0.32	-0.17	-0.12	-0.13
Net cash (SEKm)	35.2	331.4	247.8	147.7
Market cap ($m)	27			

Source: Intron Health estimates

Contents

Investment Summary

Abliva is an early-stage Swedish biotech valued at c. $30m by the market but with the potential to revolutionise the treatment of primary mitochondrial diseases. Their lead candidate, KL1333, has generated swathes of preclinical data which demonstrate its effectiveness at treating cells with impaired mitochondria and compares favourably to the only licensed drug in the EU. The first-in-human phase I trial is ongoing with a readout expected in H121; if positive, Abliva intend to push the drug into pivotal trials before the end of the year. Our company valuation of SEK2.25/share includes corporate and R&D costs to 2027 but only values the two lead assets, KL1333 and NV354, excluding multiple other sources of potential value. Our valuation assumes sufficient equity is raised in 2021 to enable the company to remain cash positive to 2025.

KL1333: SEK1.90/share

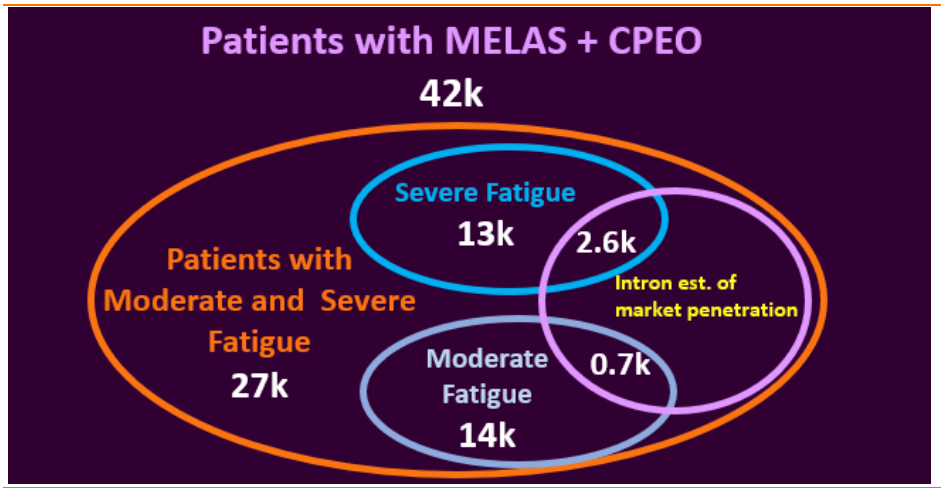
MELAS syndrome (Mitochondrial encephalopathy with lactic acidosis and stroke-like episodes) and MIDD (maternally inherited diabetes mellitus and deafness) belong to a clinical spectrum of mitochondrial diseases most frequently caused by the m.3243A>G mutation of the MT-TL1 gene in mitochondrial DNA.

CPEO (chronic progressive external ophthalmoplegia) and KSS (Kearns-Sayre) Syndromes refer to a clinical spectrum of eye disorders caused by a diverse set of mutations in highly conserved areas of mitochondrial DNA.

Abliva in-licensed the global rights (ex-Korea/Japan) for KL1333 from Yungjin in May 2017. It is being developed for the treatment of primary mitochondrial diseases (PMD), specifically targeting MELAS-MIDD and KSS-CPEO spectrum disease, which together account for ~40% of PMD. Unlike its two closest competitor drugs, which are both PPARδ agonists, KL1333 works by normalising NAD+ levels in patients by targeting an upstream receptor which also has downstream effects on PPARδ.

We expect to see first clinical data from the phase Ib trial in adult PMD patients in H121, with a pivotal trial initiating in H221. Using low penetration estimates to account for the majority of patients that are not on hospital rosters or are undiagnosed, we forecast highly risk-adjusted peak sales of $150m/year ($750m unadjusted). We conservatively value the drug at SEK1.90/share, using a 2022 share count (following assumed equity dilution to keep Abliva cash positive until 2025).

Chart 1: Target Population for KL1333 and Intron Health penetration assumptions



Source: Company reports

NV354: SEK0.47/share

The preclinical asset NV354 is an oral succinate prodrug for the treatment of Leigh Syndrome and we expect it to enter first-in-human trials this year. Unlike the drug succinate, which is not cell-permeable but known to help with Leigh Syndrome, NV354 is able to enter cells before being converted into succinate. We currently anticipate a 2026 launch and forecast highly risk-adjusted sales of up to $38m/year ($190m unadjusted), valuing it at SEK0.47/share.

Rest of Pipeline Could Provide Free Upside

Abliva is seeking to out-license the two other pipeline assets: NeuroSTAT for the treatment of traumatic brain injury and NV556 for the treatment of non-alcoholic steatohepatitis (NASH).

Table 1: Abliva's pipeline assets

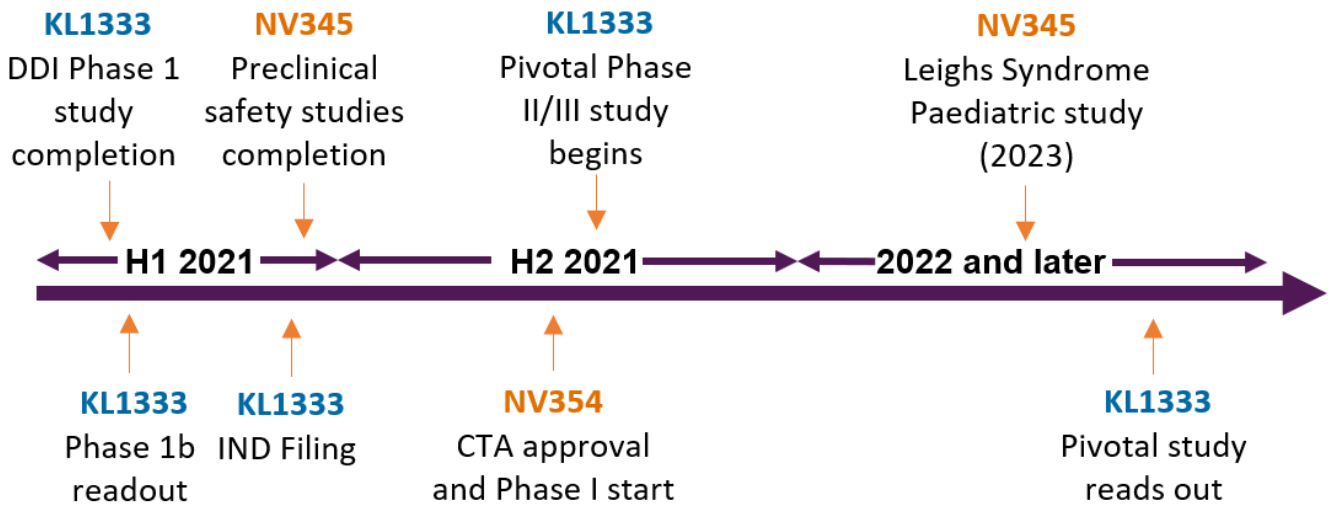
Project (partner)	Indication	Phase
KL1333 (Yungjin)	PMD	Phase I
NV354	Leigh Syndrome	Pre-clinical
NeuroSTAT	Traumatic Brain Injury	Phase I
NV556	NASH	Pre-clinical
NVP025	Mitochondrial Myopathy	Discovery

Source: Company reports

Multiple Visible Catalysts

Abliva has a glut of catalysts expected in the first 6 months of 2021, including the first clinical readout (phase I) for their lead asset KL1333. If the exploratory efficacy endpoints are positive, this would be likely to result in an uplift to our valuation of the asset, which we currently risk adjust down to 20%. We also expect NV345 to finish its preclinical safety studies in H121, after which it can begin a phase I study in H221.

Chart 2: Abliva catalyst timeline



Source: Company reports

What Are Mitochondria?

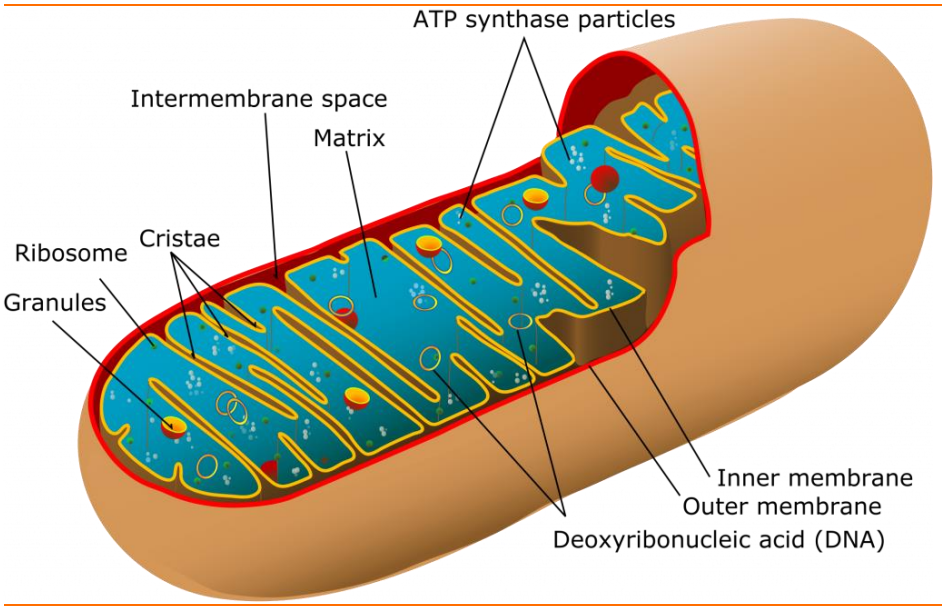
Mitochondria are membrane bound organelles (a structure that has a specific job to do inside a cell – much like an organ in the body) and are present in the cytoplasm of every cell in the human body. As the site of cellular respiration, they are responsible for generating cellular energy in the form of adenosine triphosphate (ATP), which is an essential molecule for the cell to function. Additionally, mitochondria store calcium for cell-signalling activities and are involved in mediating cellular growth and death.

Structurally Designed For Energy Release

Mitochondria possess two membranes: an inner and outer. The outer membrane is porous to permit ions and other small uncharged molecules to pass freely through it. However, the inner membrane is highly impermeable to all molecules, only permitting molecules to pass through specific membrane transporters. This means the intermembrane space has the same concentrations of small molecules as is found in cellular cytoplasm. The inner membrane is also highly folded (forming Cristae), which increases their surface area, increasing the efficiency of their function, which is to be the site of **oxidative phosphorylation**.

- Mitochondrial **matrix** is the fluid filled "cytoplasm" of the organelle and is the site of mitochondrial DNA replication, transcription, protein synthesis and numerous enzymatic reactions.
- The Inner Membrane contains vital components for oxidative phosphorylation, such as the electron transport chain proteins and the membrane localised enzyme, ATP synthase.

Chart 3: Structure of a mitochondrion



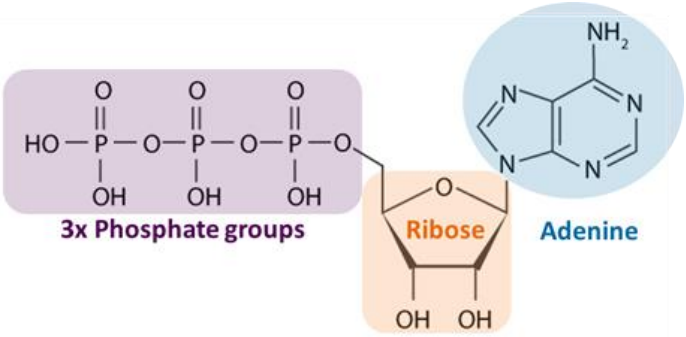
Source: TeachMe Physiology

The ATP Molecule is Life's Energy Storage Solution

All cells require a constant supply of energy simply to maintain their state, without which, under the second law of thermodynamics, they would progressively revert into disorder and die. However, the storage of large amounts of free energy would mean a temperature higher than cellular components such as proteins can tolerate. Biology has found the solution in the constant manufacture and storage of the molecule ATP, which stores chemical energy and can rapidly be employed to do "work" and keep the cell in a healthy and ordered state. Analogously, one could think of ATP as batteries and mitochondria as battery rechargers.

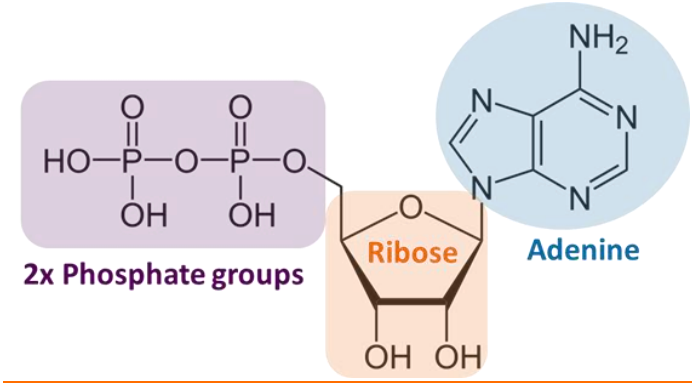
ATP is composed of a nitrogenous base adenine, a ribose sugar and three serially bonded phosphate groups. These phosphate groups are bonded together with very high energy, reactive phosphodiester bonds. The energy in the outermost bond can easily be released during a reaction, which can be used to catalyse a process which contributes to cellular functioning. During such a process, the phosphodiester bond will be broken and ATP is hydrolysed to form ADP. ADP must be converted back to ATP before it can be used again; this is called oxidative phosphorylation and is the job of mitochondria.

Chart 4: Structure of ATP



Source: Intron Health

Chart 5: Structure of ADP



Source: Intron Health

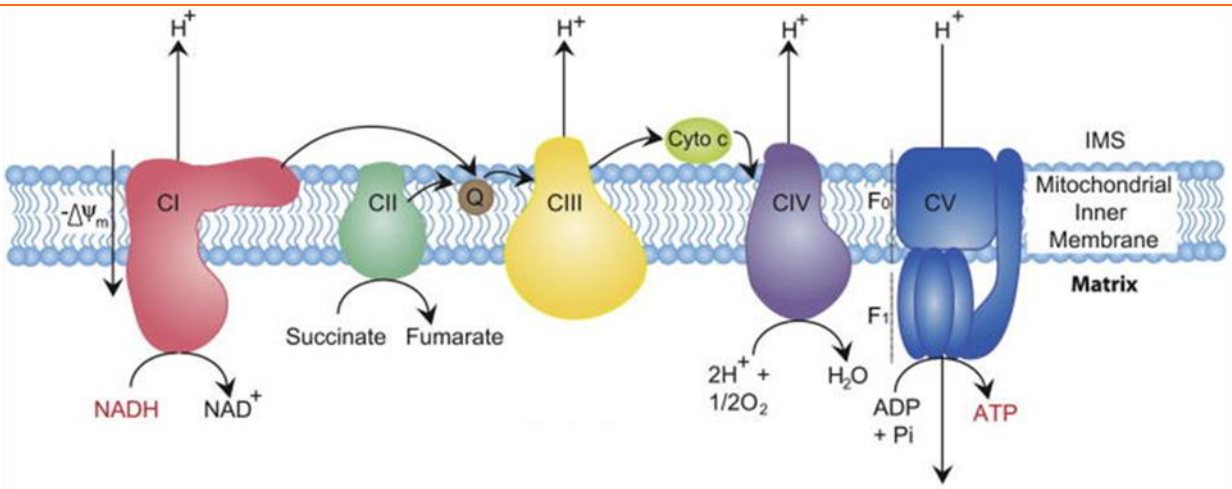
Generation of ATP from Cellular Respiration

Cellular respiration is essentially a series of redox reactions that transfers the energy in the form of electrons from ingested glucose to protein complexes in the mitochondria in order to form ATP.

ATP is generated by three essential cellular mechanisms:

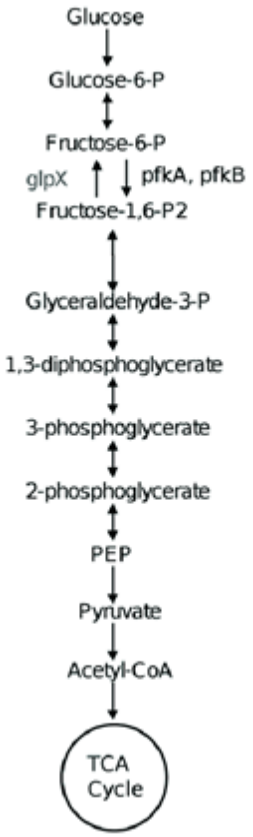
- Glycolysis
- Tricarboxylic acid (TCA) Cycle
- Oxidative Phosphorylation

Chart 6: Overview of ATP Synthesis



Source: Osellame et al, 2012

Chart 7: Glycolysis steps



Source: Phong et al, 2013

Step 1: Glycolysis

This is the first step in the breakdown of glucose to extract energy. It occurs in cellular cytoplasm and is anaerobic (does not require oxygen). In this process, glucose is converted to pyruvate via several intermediates. Although a small amount of ATP is produced, its main purpose is to manufacture pyruvate in order to fuel larger ATP-generating processes. Each pyruvate generated subsequently enters the mitochondria from the cytoplasm. Here, it is converted to acetyl-CoA in the matrix, which acts as fuel for the TCA cycle.

Step 2: The TCA Cycle

The TCA cycle takes place in the mitochondrial matrix and is a **series of eight enzymatic reactions where acetyl-CoA is oxidised (has electrons removed) to release energy.** The electron carrier molecules nicotinamide adenine dinucleotide (NAD+) and flavin adenine dinucleotide (FAD) receive these electrons (are themselves “reduced”) and become NADH and FADH2. The result of this is that the carrier molecules ferry electrons to the electron transport chain of the inner mitochondrial membrane.

Step 3: Oxidative Phosphorylation

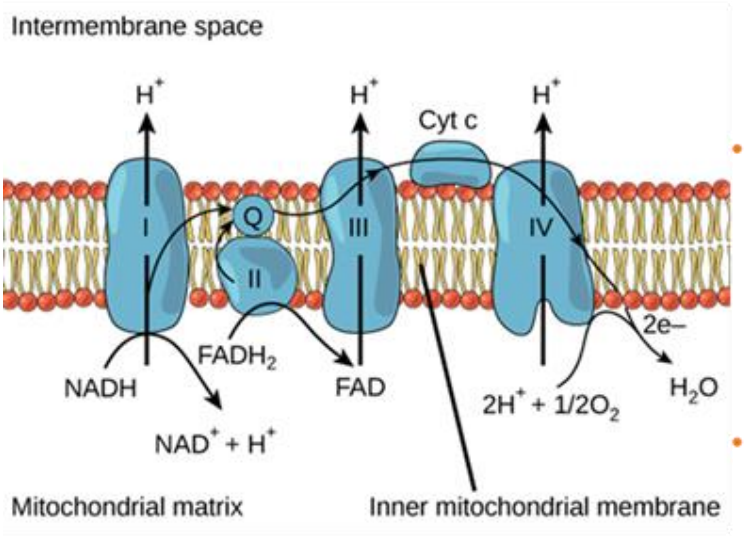
Oxidative phosphorylation is the final stage of cellular respiration and is where most ATP is generated. It is composed of two linked processes: 1) The Electron Transport Chain and 2) Chemiosmosis.

The Electron Transport Chain

This consists of a series of multi-subunit protein complexes embedded in the inner mitochondrial membrane, where electrons are passed four times from one complex to another. The energy released during these electron transfers is harnessed by the mitochondria, which use it to pump H+ ions across the inner membrane from the mitochondrial matrix to the

intermembrane space. This creates an “electrochemical gradient”, essentially storing a form of energy which can be exploited to make ATP by chemiomosis.

Chart 8: Electron Transport Chain explained



- **Complex I- NADH Dehydrogenase**

  NADH is oxidised by Complex I, releasing a hydrogen ion into the intermembrane space. Simultaneously, the two electrons from this redox reaction are transferred to Ubiquinone (Q)

- **Complex II- Succinate dehydrogenase and Ubiquinone (Q)**

  Electrons are directly donated from FADH2 to Complex II (do not pass through Complex I). The electrons received at Complex I and II from NADH and FADH2 are received by Ubiquinone, which delivers these electrons to Complex III.

- **Complex III – Cytochrome C Reductase**

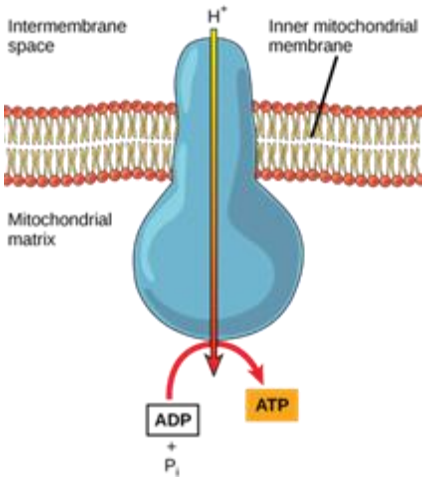
  Complex III pumps H+ ions to the intermembrane space and passes its electrons to Complex IV.

- **Complex IV- Cytochrome C Oxidase**

  The electrons from the ETC reach the last complex, where they are combined with oxygen to form water. Alongside this reaction, H+ ions are pumped from the mitochondrial matrix into the intermembrane space.

Source: Lumen Learning

Chart 9: Activity of ATP Synthase

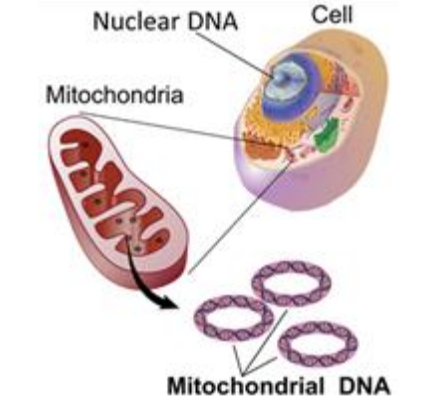


Source: Lumen Learning

Chemiosmosis

This is where the energy stored in the electrochemical gradient is used to synthesise ATP. The membrane bound enzyme ATP synthase creates a channel through the inner mitochondrial membrane. This permits the passage of H+ from inside the inner mitochondrial membrane to the mitochondrial matrix. As these H+ ions flow down their electrochemical gradient, they are used by ATP synthase to drive the energetically unfavourable addition reaction of a phosphate group to ADP to form ATP.

Chart 10: Mitochondrial DNA



Source: National Human Genome Research Inst.

PMD is An Unsolved Problem

In some people this process of mitochondrial energy creation is impaired due to mutations in the mitochondrial DNA and leads to Primary Mitochondrial Diseases (PMD). Abliva aims to treat two such disease spectrums.

Mitochondria replicate themselves separately to cellular division and for evolutionary reasons even contain their own DNA. This DNA is known as mitochondrial DNA and is always passed down the maternal line. The primary mitochondrial diseases (PMDs) which Abliva aims to treat arise from mutations or deletions in this mitochondrial DNA (or mutations in mitochondrial-associated genes in the nucleus). These diseases can be severe and/or incurable, often affecting vital organs. MELAS-MIDD and Leigh Syndrome are two such PMDs and constitute a major unmet medical need.

Disease Results From Disruption to ATP Synthesis

Mutations in mitochondrial DNA (mtDNA) are likely to result in an impaired ability to synthesise ATP. This is usually because the mutation is in a mitochondrial gene that is needed for the production of a protein that is integral to the functioning of the electron transport chain. Thus, if the mutation leads to such a protein being defective, this process is disrupted and will result in less ATP being produced which will have a cascade of negative effects downstream.

PMDs are the most common hereditary mitochondrial diseases, which are estimated to affect at least 12.5 per 100,000 of the general population (1 in 8,000). MtDNA mutations that cause these may either be inherited through the maternal line or acquired in an individual via a spontaneous mutation. Several distinct PMDs include MELAS-MIDD, Leigh Syndrome, CPEO, Alpers Syndrome, LHONs and Kearns-Sayres Syndrome.

Table 2: Distinct Primary Mitochondrial Disorders

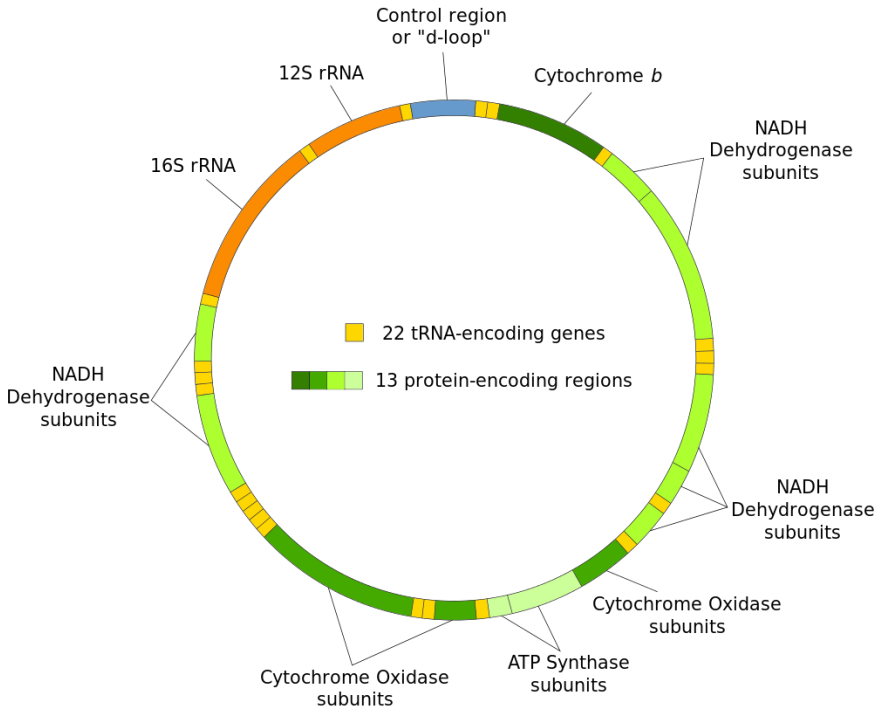
Disease	Prevalence estimate	Onset	Symptoms	Mutation Location
MELAS-MIDD spectrum	1 per 20,000	Older childhood-adult	Fatigue, myopathy, mitochondrial diabetes, progressive neurodegeneration (in 5-10%)	mtDNA
CPEO	1 per 45,000	Childhood	Impaired movement of extrinsic eye muscles, epilepsy, nervous impairment, myopathy.	mtDNA / nuclear DNA
Alpers Syndrome	1 per 100,000	Childhood	Epilepsy, brain damage, dementia, paralysis, liver, GI and nervous complications.	Nuclear DNA
Kearns-Sayres Syndrome	1 per 125,000	Early childhood	Neuromuscular abnormalities, CNS complications, dementia and electrical conduction disturbances of heart.	mtDNA
Leigh Syndrome	1 per 500,000*	Early childhood	CNS degeneration, loss of motor ability, weakness, seizures.	mtDNA / nuclear DNA

*Source: NORD, National Institute of Neurological Disorders and Stroke, Yu-Wai-Man et al 2014, NCBI * 1/34k births but is a childhood disease with high mortality*

Mitochondria Are Under Dual Genetic Control

Mitochondria functioning is controlled by both nuclear and mtDNA, though it is mutations in the latter which are the predominant cause of PMD in adults. The mitochondrial genome consists of 37 genes that code for essential polypeptides and RNA machinery needed to synthesise the important molecules required for oxidative phosphorylation.

Chart 11: Human Mitochondrial Genome



Source: Medicalxpress

Mitochondrial DNA is highly susceptible to mutation:

Mitochondrial DNA mutates at roughly 10-20x the rate of nuclear DNA, for three main reasons:

- Lacks protective histones (proteins that wrap around the DNA)
- Greater exposure to mutagenic reactive oxygen species (ROS)
- Mitochondria do not possess effective DNA repair mechanisms

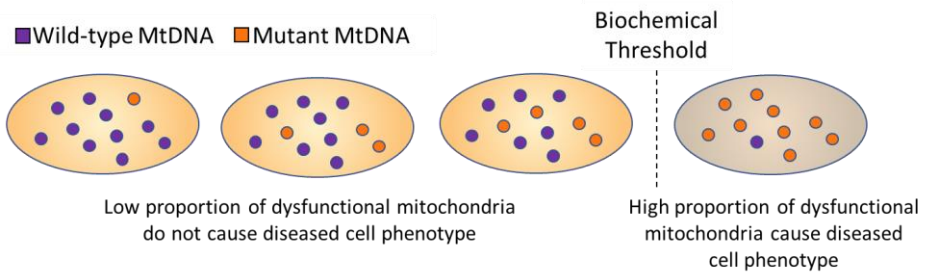
Given the vital importance of mitochondria to the body and the high mutation rate of mtDNA, it is hardly surprising that PMDs are so severe. However, as we go on to show, the precise manifestations of PMD can vary greatly from patient to patient.

PMD is a Highly Heterogeneous Group of Diseases

MtDNA exists as circles of DNA within mitochondria. These circles collectively contain hundreds to thousands of copies of the same genetic material in a single mitochondrion. Under normal conditions, all of these copies are genetically identical (termed homoplasmic). When an

unfavourable mutation occurs, only one of the gene copies is usually affected, which means that mutated and normal mtDNA may co-exist in a single cell (termed heteroplasmic). Over time, as the mutated DNA is copied, the proportion of gene copies with the mutation may rise. However, as long as the proportion of mutated genes remains low, an individual is likely to remain healthy and may not display any symptoms. If, on the other hand, more than around 60% of the copies are mutated, then disease is likely to appear. This degree of mitochondrial mutational burden is one of the key factors giving rise to the heterogeneity of disease.

Chart 12: A high proportion of dysfunctional mitochondria cause disease



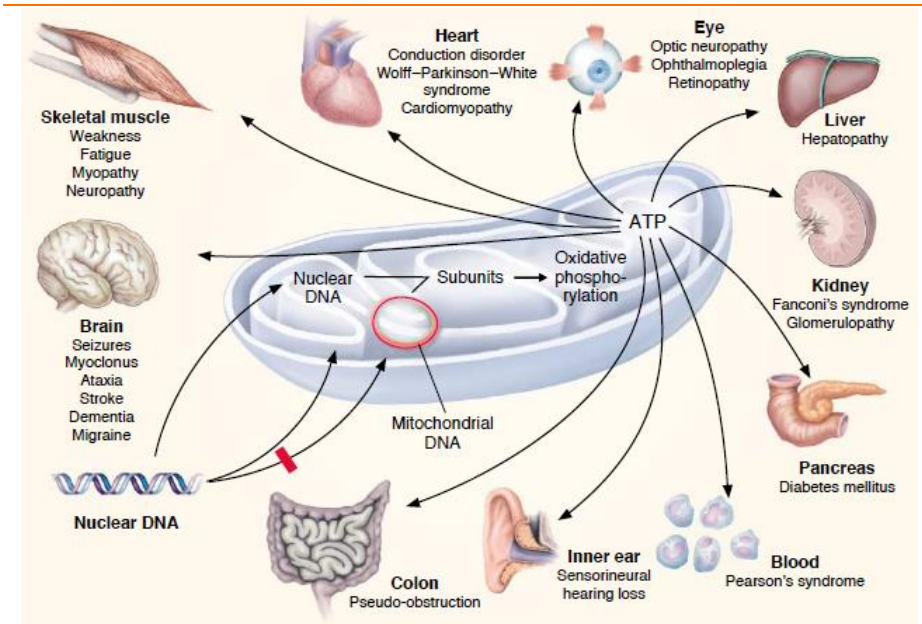
Source: Intron Health

High Energy Requiring Organs are Most Affected

We have already explained how mitochondria may harbour negative mutations without causing symptoms, but the distribution of diseased mitochondria throughout the body also affects disease phenotype. Some tissues may have large numbers of mitochondria with a high proportion of negative mutations and other tissues may be unaffected. This makes PMD vary in symptoms and severity from patient to patient.

Mitochondria occur in different concentrations in different tissues according to function. Organs that require a lot of energy to function like the brain, heart, liver, kidneys, eyes and skeletal muscle are particularly afflicted by PMD. Patients commonly experience severe symptoms that include learning difficulties, fatigue, myopathy, heart failure, diabetes, movement disorders, blindness, seizures and limited eye mobility.

Chart 13: PMD affects many vital organs



Source: Kimberly Jimenez

MELAS-MIDD Spectrum Disorders

The m.3243A>G mutation in mtDNA is responsible in c. 80% of patients for a diverse spectrum disorder known as MELAS-MIDD. This consists of two separate syndromes caused by the same mtDNA mutation that prevents the assembly of essential mitochondrial proteins. MELAS is at the more severe end of the disease spectrum and is a potentially fatal syndrome. With KL1333, Abliva look to treat the most common manifestation of MELAS-MIDD, in which fatigue, myopathy and metabolic dysfunction are the major symptoms.

This fatal disease normally presents early in life

MELAS-MIDD spectrum disorders mainly cause symptoms in adolescents and adults, but 75% of patients develop symptoms before the age of 20 including in childhood. Unfortunately, the median survival time for MELAS is just 17 years post-diagnosis and only ~7 years for the fifth of patients who present with the severe form of the disease.

Major symptoms are fatigue, myopathy and metabolic disfunction

The broadest group of patients within the MELAS-MIDD spectrum typically experience symptoms including fatigue, myopathy and metabolic dysfunction. This fairly homogenous set of patients forms the main target population for Abliva's drug KL1333. Patients on the more severe end of the spectrum with a confirmed MELAS diagnosis must have had a stroke-like episode, but most patients never experience this.

At the less severe end of the spectrum, MIDD (maternally inherited diabetes and deafness) is a separate syndrome to MELAS but is caused

by the same mutation; why some patients develop this rather than MELAS or *vice versa* is unclear.

At the more severe end of the spectrum, MELAS (mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes) is caused by an accumulation of lactic acid in the blood (lactic acidosis) that leads to muscle weakness, abdominal pain, vomiting, fatigue and breathing difficulty. Patients also suffer from cognitive deterioration, seizures, diminished communication ability and the recurrence of stroke-like episodes which is the hallmark of the disease.

MELAS-MIDD is caused by a mutation in the MT-TL1 gene

In 90% of patients, MELAS-MIDD is attributed to a mutation in the Mitochondrially Encoded TRNA-Leu (UUA/G) 1 (MT-TL1) gene of mtDNA. A single base substitution from A to G at the nucleotide pair 3243 (m.3243A>G) is found in 80% patients, and a second common mutation, m.3271T>C, is found in another 10% of patients.

The mutation prevents construction of essential proteins

The MT-TL1 gene provides instructions for making the transfer RNA (tRNA) molecules which help to build amino acids into functional proteins. When this gene is mutated, the mutant protein:

- Impairs the synthesis of essential proteins needed for the mitochondrial respiratory chain and oxidative phosphorylation
- This causes the increased generation of reactive oxygen species (ROS) which further damages the mitochondria
- As the cell tries to compensate by generating more ATP via glycolysis (an anaerobic process), lactate is produced which further damages tissue and accelerates organ failure

The systemic dysfunction caused by insufficient ATP, increased lactic acid production and ROS manifests as MELAS-MIDD. MELAS-induced cell death leads to severe disability and eventual death as whole organ systems fail.

Leigh Syndrome is a Very Severe PMD

This PMD is a severe, early-onset progressive neurodegenerative disorder caused by failure of the mitochondrial respiratory chain. With insufficient amounts of ATP being produced, overuse of glycolysis results in tissue damage from lactic acid build up. Sadly, this disease has a median age of death of <5 years.

Early onset and accelerated mortality

Disease onset occurs within the first year of life and causes rapid regression of cognitive ability and motor skills. This typically manifests as loss of movement control, seizures, stunted growth and decreased ability to feed. Median patient age is just 2.4 years, with patients rarely surviving childhood.

Caused by both mitochondrial and nuclear DNA mutations

Leigh syndrome occurs due to mutations in any of over 75 different mitochondrial and nuclear genes which are responsible for components of the mitochondrial respiratory chain. Around 75% of Leigh syndrome patients have a mutation in nuclear DNA, with 25% presenting mutated mtDNA. These mutations can be inherited via the maternal line or as an autosomal recessive trait from mutated genes of the nucleus.

These mutations impair the Electron Transport Chain

PDH (pyruvate dehydrogenase) is a mitochondrial matrix multienzyme complex that provides the link between glycolysis and the TCA cycle by catalysing the conversion of pyruvate into acetyl-CoA.

Leigh syndrome is frequently caused by mutations that affect the PDH complex, Complex I (in 50% of cases), Complex II, Complex IV and ATP synthase. These mutations impair the activity of the ETC complexes, which leads to the generation of reactive oxygen species (ROS) and causes insufficient ATP production. As the cell tries to compensate by generating more ATP via glycolysis (an anaerobic process), lactate is produced which leads to further tissue damage and accelerates organ failure.

Few Treatments For MELAS & Leigh

Currently, there is no effective treatment for either MELAS or Leigh Syndrome, with drugs instead focusing on managing the symptoms of the conditions rather than slowing progression. Symptomatic treatment includes the administration of anti-convulsants to treat seizures and vitamin supplementation of CoQ10 and L-carnitine to attempt to increase the energy output of mitochondria. Idebenone, a synthetic CoQ10 has displayed limited positive efficacy in MELAS clinical trials but has nevertheless been approved in the EU for LHON after showing a positive effect on patient fatigue.

KL1333 - $150m Risk-Adj. Peak Sales

Abliva in-licensed KL1333 from Yungjin Pharm and have exclusive global rights for the drug, excluding Korea and Japan. It is in development for the treatment of primary mitochondrial diseases and holds Orphan Drug Designation in both the US and Europe. Based on our assumptions, we show how the drug could generate heavily risk-adjusted peak sales of up to $150m/year ($750m/year when fully de-risked) and we value it at SEK1.90/share based on a 2024 launch. We expect to see first clinical data from the phase Ib trial in adult PMD patients in H1 2021. A pivotal phase II/III trial could initiate as soon as H2 2021.

KL1333 Treats PMD By Normalising NAD+ Levels

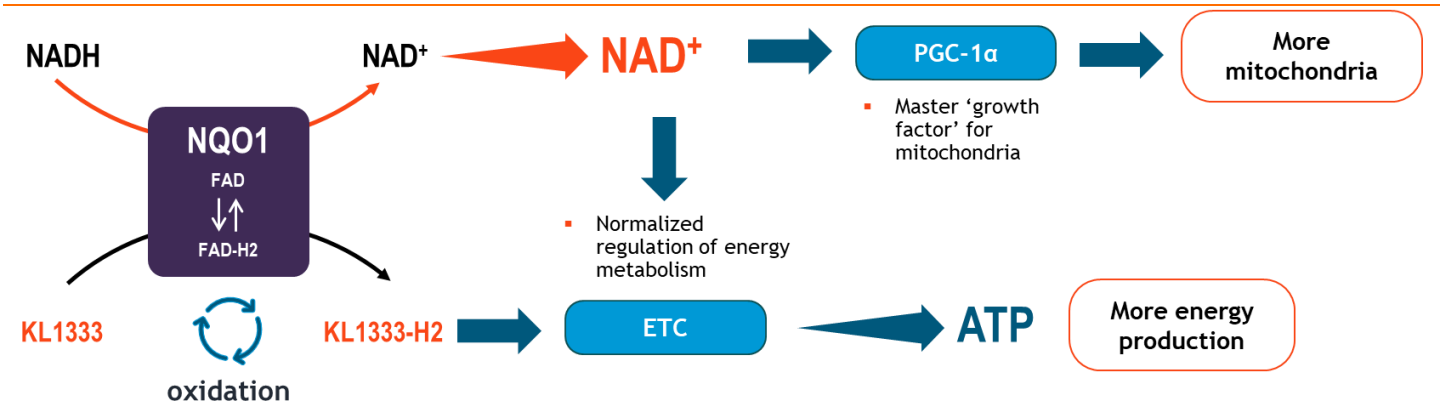
KL1333 is an orally administered small organic molecule derived from β-lapachone, a quinone-containing compound obtained from the Lapacho tree. It treats mitochondrial disease by increasing the concentration of NAD+ to normal levels. This increased NAD+/NADH ratio has a two-pronged effect which helps treat the underlying condition by:

- Increasing ATP production
- Increasing mitochondrial biogenesis

NAD+ is a Crucial Molecule in Respiration

The ratio of intracellular NAD+ to NADH is decreased in diseases associated with mitochondrial dysfunction. As NAD+ is required for numerous steps in cellular respiration, this limits the rate at which it can occur, causing symptoms. KL1333 helps to alleviate this by acting as a substrate for the enzyme NQO1. When KL1333 is metabolised by the enzyme, NQO1 simultaneously catalyses the oxidation of NADH to NAD+, increasing the concentration of intracellular NAD+, which helps to increase ATP production. KL133 is then recycled via direct donation into the electron transport chain and this fresh KL1333 is then able to re-stimulate another cycle of NADH oxidation to NAD+.

Chart 14: KL333 acts by increasing NAD+ formation



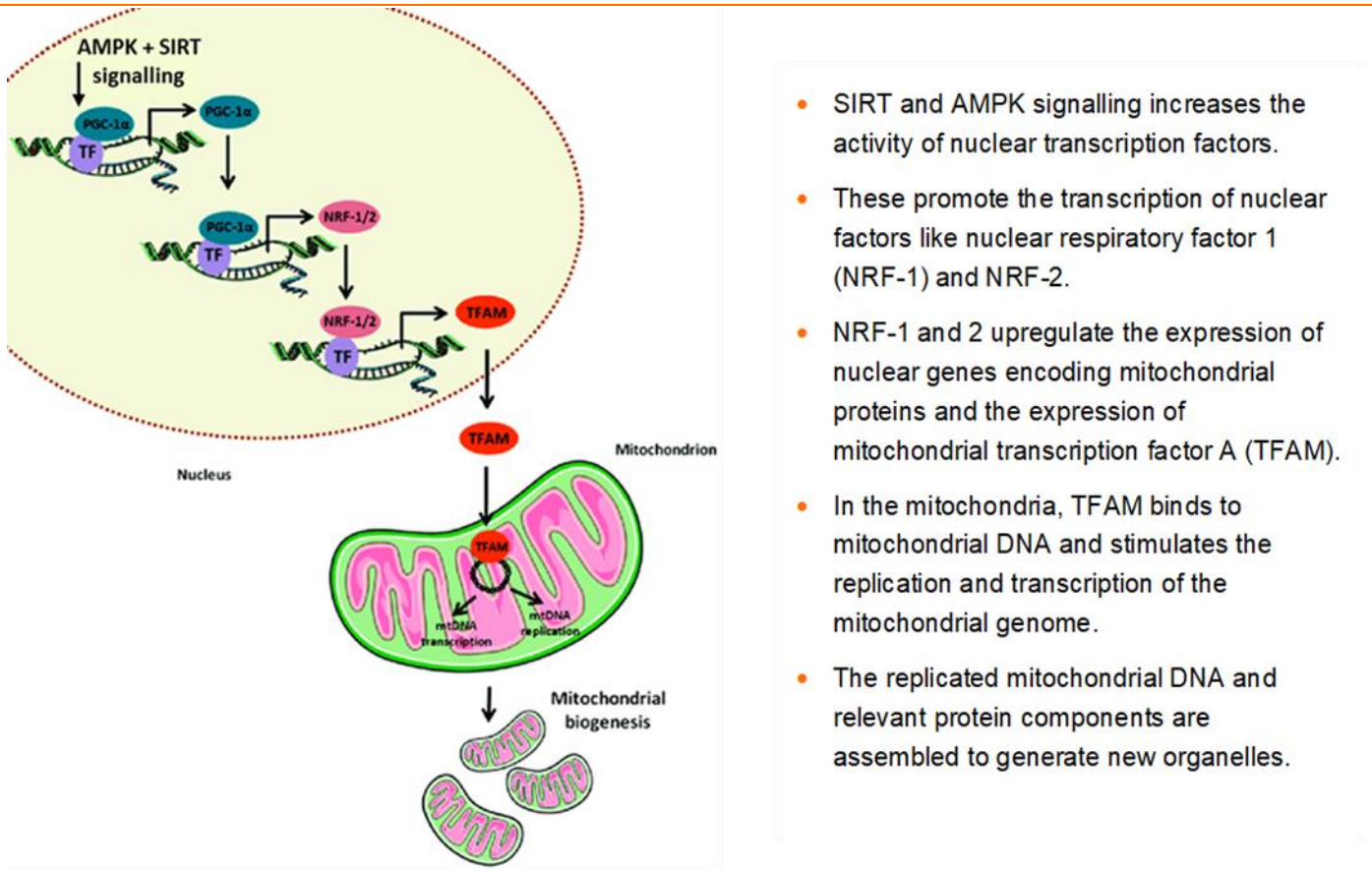
Source: Company reports

NAD+ Increases Mitochondria Biogenesis

SIRT proteins activate PGC-1α, which kick starts mitochondria biogenesis. They also enhance mitochondrial functioning by increasing the activity of antioxidant enzymes and others that repair mtDNA. Thus, KL1333, through increasing NAD+ concentrations, both increases the number of new mitochondria and optimises their functioning.

As well as helping to increase ATP production within mitochondria, NAD+ also acts to increase mitochondria biogenesis (proliferation), which is regulated by complex signalling pathways in response to various stimuli. PGC-1α is a master regulator which activates several transcription factors required for mitochondrial DNA replication. NAD+ activates this regulator by functioning as a co-substrate for the activation of SIRT proteins (see margin) and the activation of the AMP-activated protein kinase pathways. These proteins activate PGC-1α and so increase the synthesis of new mitochondria. Full details of this biochemical pathway are given in the diagram below.

Chart 15: KL1333 causes increased mitochondria biogenesis



Source: Picca et al, 2017

Very Strong Pre-Clinical Data

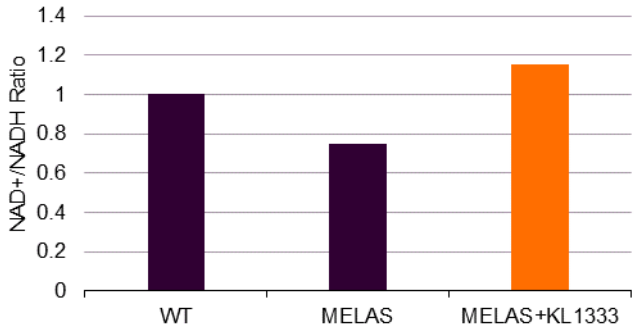
Abliva and academic researchers have shown KL1333 to exhibit broad and strong efficacy in fibroblasts derived from human MELAS patients and in an *in vivo* mouse model. Given that it displayed stronger potency against existing therapies which are known to have a clinical benefit, we are hopeful that KL1333 will also be able to demonstrate a clinical benefit in future trials. In its preclinical studies, KL1333 has demonstrated increased NAD+ and ATP

production, improved mitochondria biogenesis and higher mass and reduced oxidative stress. In the *in vivo* mouse model, it also showed improved muscle function and improvement across three biomarkers of mitochondrial damage and muscle injury.

Increased NAD+/NADH Ratio and ATP Levels

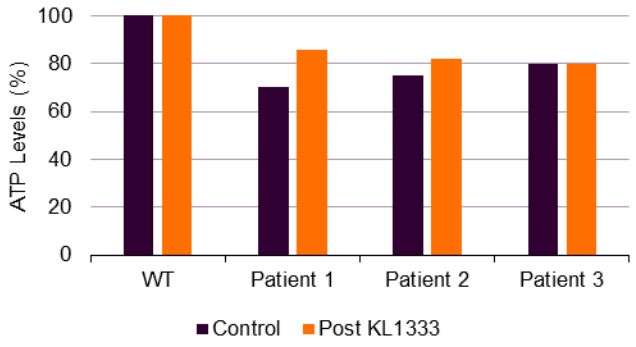
KL1333 induced the NQO1-mediated oxidation of NADH to NAD+ in patient fibroblasts. As shown below, MELAS fibroblasts have a lower NAD+/NADH ratio than wild type (normal) cells (WT), but KL1333 significantly increased this ratio beyond that of healthy cells (below left). This increase in NAD+ translated to a significant increase in ATP levels (in two out of three patient samples, below, right). Patients 1 and 2 saw a 15% and 8% increase in ATP respectively. We believe this is likely to be clinically meaningful and may result in markedly improved symptoms.

Chart 16: Improved NAD+/NADH ratio



Source: Seo et al, 2018

Chart 17: KL1333 increases patient ATP levels

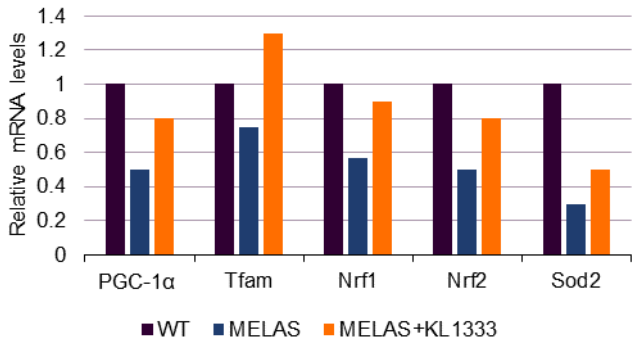


Source: Source:Seo et al, 2018

Increased Mitochondrial Biogenesis

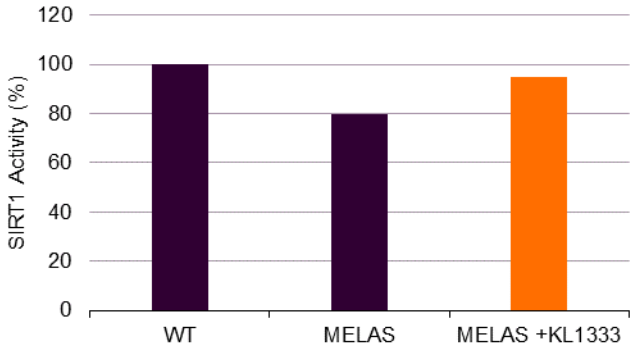
KL1333 significantly increased the activation of PGC-1α in MELAS fibroblasts, as shown by the increased expression of PGC-1α and its target genes (bottom, left). Greater SIRT1 and AMPK activity was also exhibited in these KL1333-administered cells (bottom, right). These findings are important as the activity of these molecules are required to manufacture new mitochondria.

Chart 18: KL13333 increases gene transcription



Source: Seo et al, 2018

Chart 19: KL1333 normalises SIRT1 activity

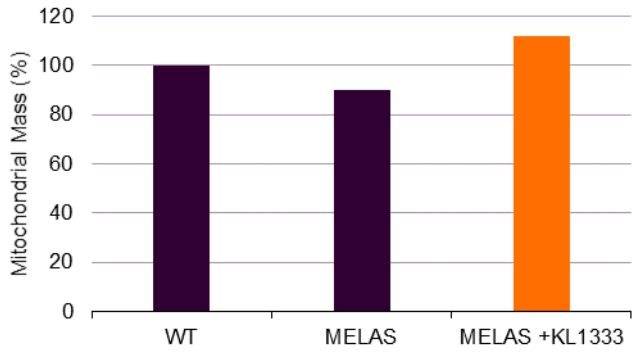


Source: Source: Seo et al, 2018

Increased Mitochondrial Mass

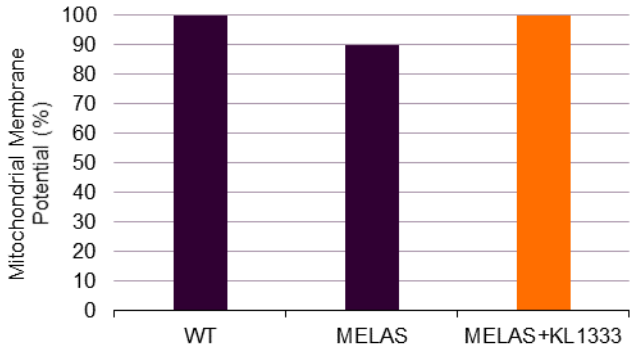
KL1333 significantly increased the mitochondrial mass of MELAS fibroblasts, surpassing that of healthy cells by 10%. Mitochondrial functioning was also improved, with the mitochondrial membrane potential increased to match that of healthy cells.

Chart 20: KL1333 increases mitochondrial mass



Source: Seo et al, 2018

Chart 21: Mitochondrial membrane potential is normalised

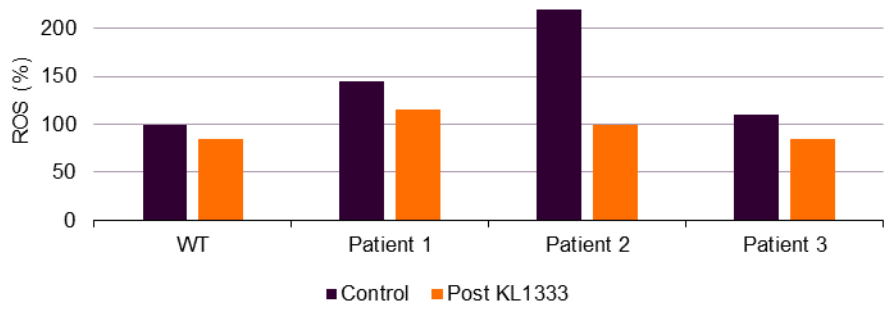


Source: Seo et al, 2018

Reduced Oxidative Stress

MELAS fibroblast lines were observed to have significantly higher levels of reactive oxygen species (ROS) compared to healthy cells. KL1333 significantly reduced ROS in all three patient fibroblasts samples, with all three patients showing similar ROS levels to normal (WT) cells post-administration. These findings are significant as this reduction in ROS could reduce cellular damage and prolong organ functioning.

Chart 22: KL1333 normalises patient ROS levels



Source: Seo et al, 2018

KL1333 is More Potent Than Existing Therapeutics

Idebenone is a synthetic analogue of CoQ10, a vital cell antioxidant and component of the Electron Transport Chain. Although it is a NQO1 substrate, it lacks the recycling "ping pong" activity of KL3333 which may explain its lower effectiveness.

KL1333 has shown greater potency than existing NAD+ stimulators idebenone and CoQ10 (see margin). The former has previously demonstrated a clinical benefit and is approved in the EU (for LHON).

In a phase II trial sponsored by Santhera, two cohorts of MELAS patients received daily idebenone for one month and reported significantly improved fatigue as assessed by the Fatigue Severity Scale (FSS). Patients that received either dose of idebenone saw a decrease in FSS score, indicating clinical improvement, but those on placebo had a higher FSS score at the end of the trial (suggesting a worsening of fatigue).

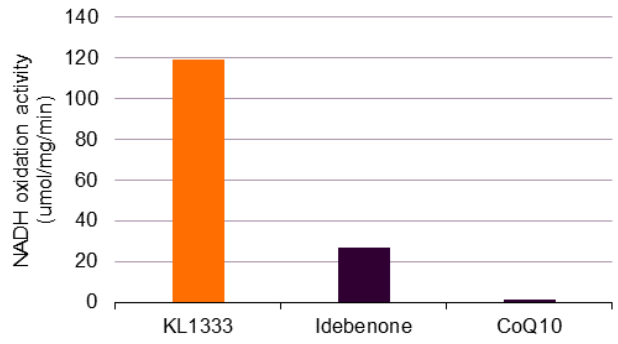
Table 3: FSS score change with idebenone or placebo treatment

Dose	N	FSS Score Change
Placebo	7	+4.3
900mg/day idebenone	7	-3.8
2,250mg/day idebenone	7	-1.3

Source: Clinicaltrials.gov, NCT00887562

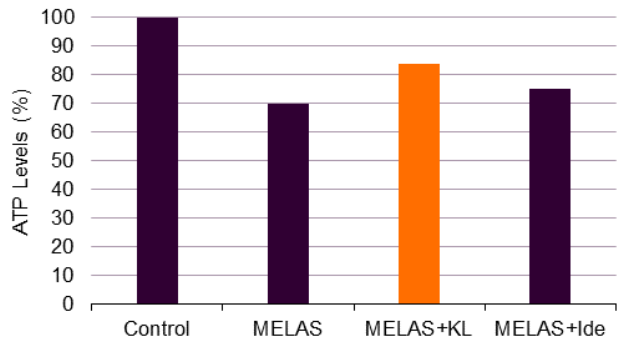
In preclinical trials, KL1333 exhibited much higher NADH oxidation activity than both idebenone and CoQ10 (bottom, left) and showed greater ATP production than idebenone in patient fibroblasts with MELAS (bottom, right). These findings give us comfort that KL1333 is also likely to show a clinical benefit and is highly likely to be approved if it does.

Chart 23: KL1333 has greatest NADH oxidation activity



Source: Seo et al, 2018

Chart 24: KL1333 (KL) was better at increasing ATP levels

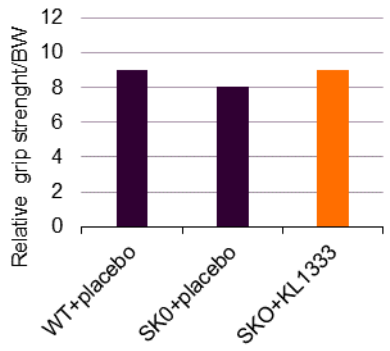


Source: Seo et al, 2018

Muscle Improvements Seen *In Vivo*

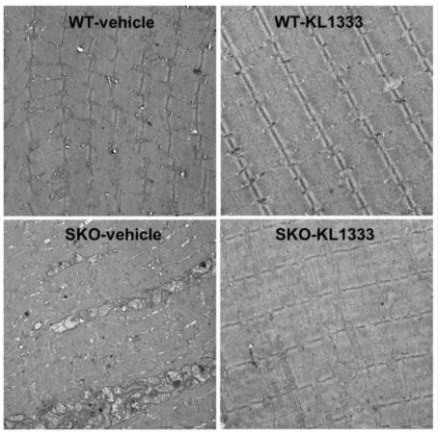
Patients with MELAS caused by a m.3243A>G mutation in mtDNA experience impaired protein translation of respiratory chain subunits. This can be modelled in mice by using a Crif1 skeletal knock-out (SKO). In an experiment, SKO mice were orally dosed for 4 weeks with either placebo or KL1333 and their relative grip strength measured and compared to healthy mice (WT). The SKO mice on KL1333 were found to have significantly increased grip strength and normalised muscle histology compared to those SKO mice that received placebo.

Chart 25: Improved Grip Strength



Source: Company reports

Chart 26: Muscle histology samples

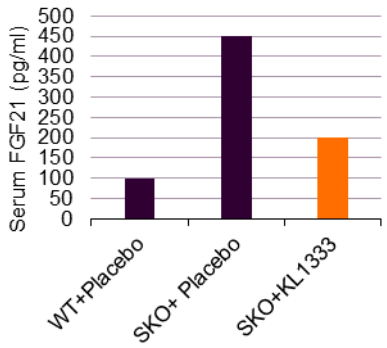


Source: Company reports

Mitochondrial damage biomarkers also improved

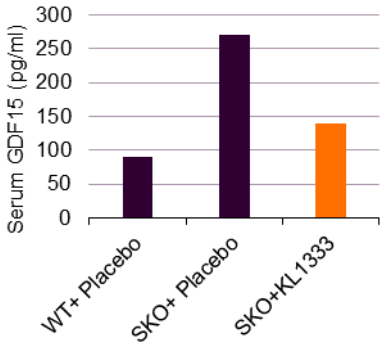
In the SKO mice, KL1333 administration significantly reduced the biomarkers FGF21 and GDF15. These biomarkers are good proxies for mitochondrial damage and reflect the NAD+/NADH ratio, so their reduction is likely to correlate with slower disease progression in our view. Likewise, creatine kinase, a marker of muscle injury, was normalised by KL1333 to that of healthy cells.

Chart 27: KL1333 reduced FGF21



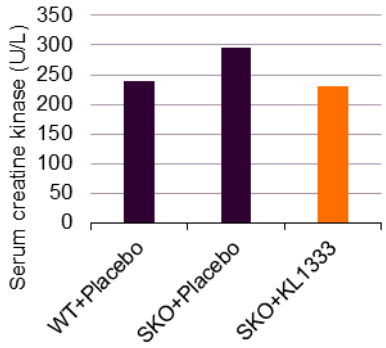
Source: Company reports

Chart 28: KL1333 reduced GDF15



Source: Company reports

Chart 29: KL1333 reduced creatine



Source: Company reports

Pivotal Phase II/III to Initiate in H2 2021

KL1333 is currently under investigation in an ongoing phase Ib trial and a drug-drug interaction safety study. This is part of an approved preparatory programme to enable the drug to move directly into a pivotal phase II/III trial in H2 2021. The phase Ib trial has already recruited over half of patients and we expect a readout by H1 2021. The pivotal phase II/III trial is expected to measure change in chronic fatigue symptoms and with an adaptive design of approximately 150 patients would have 80% statistical power.

Table 4: Phase II/III Pivotal Trial Outline

Trial Outline	
Design	Randomised, double-blind, parallel-group, placebo controlled
Patient Demographic	Adult Patients with • Genetically confirmed PMD consistent with MIDD-MELAS or KSS-(C)PEO spectrum disorders • Chronic fatigue and multisystemic disease - myopathy and metabolic dysfunction
N	150
Study Regimen	Upward dose titration in weekly increments 12 months of treatment (including dose titration phase)
Primary Endpoint	Patient-reported fatigue

Source: Company reports

First Patient Data Expected in H1 2021

KL1333 is currently under investigation in a double-blind placebo-controlled phase Ib study in adult PMD patients with first readout expected in H1 2021. The first-in-human study revealed that single and repeat daily dosing of KL1333 was tolerable with no severe adverse events exhibited in healthy volunteers. KL1333 was found to accumulate in the blood in a dose-dependent manner.

Table 5: NCT03888716 Trial Design

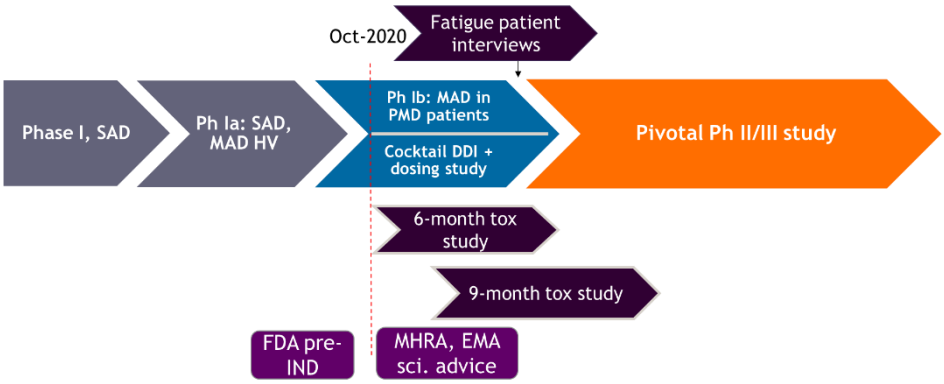
Trial	N	Primary Purpose	Primary Completion	Study Completion
NCT03888716	72	Safety and Pharmacokinetics Exploration of biomarker, PRO and functional endpoints e.g NAD+ and Daily Fatigue Severity	31/01/21	28/02/21

Source: Clinicaltrials.gov

KL1333 Acceleration is Guided by Regulators

Abliva has received positive regulatory feedback from the FDA and MHRA for the KL1333 phase II/III development plan. It was on the FDA's advice that Abliva initiated a drug-drug interaction study and a qualitative study validating specific patient reported outcome measures in parallel with the ongoing phase I trial. A long-term *in vivo* toxicology study will be initiated to run in parallel with the pivotal phase II/III trial in H2 2021. We expect this to accelerate development and could reduce time to market approval by around 2 years.

Chart 30: KL1333 Development Overview



Source: Company reports

Heavily Risk-Adjusted Sales of $150m

We see the primary target market for KL1333 as being patients with MELAS Spectrum disorder and moderate to severe fatigue. Across the US, EU and UK, this amounts to c. 27k patients, based on extrapolating a prevalence study in Northern England. We assume that the market size grows broadly in line with population growth and assume a peak penetration of 5% for those moderately fatigued and 20% for those severely afflicted, with a launch in 2024 and an 8-year ramp. Our penetration rates may seem low, but we have scaled down to reflect for those patients not on hospital rosters or who have not been diagnosed (66-75%). Thus, our penetrations assume that the majority of known patients end up on treatment. In addition, there a number of competitors that could also penetrate this market.

Table 6: Moderate and Severe Fatigue in MELAS Spectrum disorder patients in the US, UK and EU

Patients	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031
Moderate Fatigue												
US	**5,514**	**5,558**	**5,602**	**5,647**	**5,692**	**5,738**	**5,784**	**5,830**	**5,877**	**5,924**	**5,971**	**6,019**
KL1333 penetration	0%	0%	0%	0%	0.10%	0.50%	1.30%	2.50%	3.60%	4.40%	4.80%	5.00%
UK and EU	**8,615**	**8,658**	**8,702**	**8,745**	**8,789**	**8,833**	**8,877**	**8,921**	**8,966**	**9,011**	**9,056**	**9,101**
Penetration	0%	0%	0%	0%	0.10%	0.50%	1.30%	2.50%	3.60%	4.40%	4.80%	5.00%
Total moderately fatigued patients treated					**18**	**73**	**183**	**369**	**538**	**653**	**723**	**756**
Severe Fatigue												
US	**5,120**	**5,161**	**5,202**	**5,244**	**5,286**	**5,328**	**5,371**	**5,414**	**5,457**	**5,501**	**5,545**	**5,589**
Penetration	0%	0%	0%	0%	0.5%	2.0%	5.0%	10.0%	14.5%	17.5%	19.3%	20.0%
ROW	**8,000**	**8,040**	**8,080**	**8,121**	**8,161**	**8,202**	**8,243**	**8,284**	**8,326**	**8,367**	**8,409**	**8,451**
Penetration	0%	0%	0%	0%	0.5%	2.0%	5.0%	10.0%	14.5%	17.5%	19.3%	20.0%
Total severely fatigued patients treated					**67**	**271**	**681**	**1,370**	**1,998**	**2,427**	**2,686**	**2,808**
Total patients treated					**85**	**343**	**864**	**1,739**	**2,537**	**3,080**	**3,409**	**3,564**
In US					34	135	341	687	1,004	1,222	1,355	1,419
In EU/UK					52	208	523	1,051	1,532	1,858	2,055	2,145

Source: Intron Health estimates

- We only include US and EU/UK sales as Abliva does not have rights in Japan/Korea
- Pivotal trial begins in H221 and is expected to take 1 year, hence 2024 launch is a reasonable estimate given 1 year regulatory time

- As discussed later, there are competing products being developed which may take some market share away from Abliva
- More moderately fatigued patients may not be eligible for KL1333 treatment given the likely high cost

Orphan drug pricing and 20% risk adjustment gives $150m sales

Abliva has received orphan drug designation from the FDA, so we assume appropriate orphan drug pricing of $250k in the US and $180k in the UK/EU. We use a risk adjustment of 20% given the absence of clinical data, which gives rise to our 2031 peak sales forecast of $153m. Due to the very high risk adjustment used, if the phase Ib data in H121 is positive, we envisage a significant revenue upgrade to KL1333.

Table 7: Sales forecasts for KL1333 by region

$m	2024	2025	2026	2027	2028	2029	2030	2031
US pricing ($)	250,000	253,000	255,000	258,000	260,000	263,000	265,000	268,000
Risk adjustment	20%							
US risk-adjusted sales	**2**	**7**	**17**	**35**	**52**	**64**	**72**	**76**
UK/EU pricing ($)	180,000	180,000	180,000	180,000	180,000	180,000	180,000	180,000
Risk adjustment	20%							
UK/EU Risk-adjusted sales	**2**	**7**	**19**	**38**	**55**	**67**	**74**	**77**
Total risk-adj. sales (base case)	**4**	**14**	**36**	**73**	**107**	**131**	**146**	**153**
Total de-risked sales	*18*	*72*	*181*	*366*	*537*	*656*	*729*	*766*

Source: Intron Health estimates

KL1333 Could Be Worth 2.5x Current Market Cap

We have valued KL1333 carefully, factoring in all pay-away's and the impact from tax losses carried forwards. We anticipate that Abliva will pay milestone payments of $1m in 2021 upon the completion of the phase I trial and another $12m in 2023 for the completion of pivotal trials. We also record $42m of payouts over 2024-26 to account for regulatory approvals and gaining drug reimbursement from payers. Abliva is also obliged to pay a royalty ranging from single digit to low double digits – we forecast an average rate of 12% by 2029. Swedish tax rates are 21.4%, but allow for losses to be carried forward, so we bake in a benefit from c. $120m of tax losses that we believe they will generate. At a WACC of 12% and EBIT margin of 80%, we show that KL1333 is worth $165m to Abliva, or SEK 1.90/share (in Dec-2022, after significant dilution).

Table 8: NPV for KL1333

$m	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035
Sales	0	0	0	0	4	14	36	73	107	131	146	153	153	153	153	153
EBIT (pre-payaways)	**0**	**0**	**0**	**0**	**3**	**11**	**29**	**59**	**86**	**105**	**117**	**123**	**123**	**123**	**123**	**123**
EBIT margin					80%	80%	80%	80%	80%	80%	80%	80%	80%	80%	80%	80%
Royalties paid					0	-11	-33	-7	-12	-16	-18	-18	-18	-18	-18	-18
Milestones paid		-1	0	-12	-14	-14	-14									
Total EBIT	**0**	**-1**	**0**	**-12**	**-11**	**-14**	**-18**	**51**	**74**	**89**	**99**	**104**	**104**	**104**	**104**	**104**
Taxation (21.4%)	0	0	0	0	0	0	0	0	0	-19	-21	-22	-22	-22	-22	-22
NOPAT	**0**	**-1**	**0**	**-12**	**-11**	**-14**	**-18**	**51**	**74**	**70**	**78**	**82**	**82**	**82**	**82**	**82**
NPV ($m)	165															
NPV (SEKm)	**1,348**															
NPV/share (2022)	1.90															
as % share price	**250%**															

Source: Intron Health estimates

- We have assumed zero sales ex-US, UK/EU and no terminal value beyond 2035
- We have assumed no sales outside of MELAS spectrum disorders

Licensing Agreement with Yungjin Pharm

Yungjin Pharm has granted Abliva exclusive global rights (excluding Japan and Korea) to develop and commercialise KL1333 worldwide. Under the terms of the agreement, Yungjin Pharm received $1m at signing, another $1m one year later and will receive an additional $1m after the completion of a successful phase I trial. A further $12m will be paid upon successful achievement of clinical development and up to another $42m for market and reimbursement approval milestones. Yungjin is also eligible to receive approval and sales milestone payments as well as tiered single to low double-digit royalties on future net sales.

Note continued overleaf…

A prodrug is an inactive compound that is converted in the body to its active therapeutic form.

NV354: ~$40m Sales in CI Dysfunction

NV354 is an oral succinate prodrug which unlike succinate, is cell-permeable. It is in development for mitochondrial diseases attributed to defects in Complex I of the Electron Transport Chain (ETC), as intracellular succinate is known to enable the ETC to bypass the faulty Complex I. With strong pre-clinical data already achieved, phase I trials are planned to initiate in H2 2021, with a potential early market entry in 2026. We forecast that highly risk-adjusted peak sales could be up to ~$40m a year by 2033 (and almost $200m when fully de-risked).

NV354 is New Treatment for Complex I Dysfunction

Mitochondrial complex I deficiency is the most prevalent defect of the respiratory chain in paediatric mitochondrial diseases, accounting for c. 30% of cases. This spectrum of disorders includes Leigh syndrome and Leber's hereditary optic neuropathy (LHON).

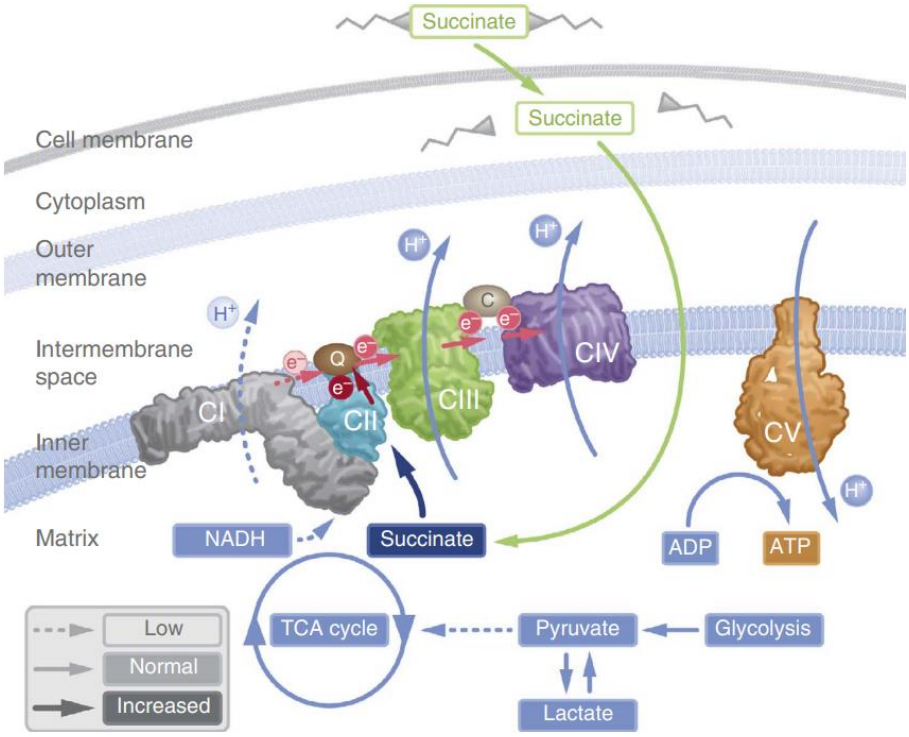
Succinate is a potential treatment, but has poor cell uptake

Succinate is a mitochondrial substrate which is metabolised through Complex II. This is an obvious potential treatment for mitochondrial diseases caused by a Complex I deficiency, as succinate can donate its electrons through the Electron Transport Chain without requiring a functional Complex I. However, in practice, succinate is ineffective as it is not able to pass through cell membranes, so it has minimal uptake into cells.

NV354 can efficiently supply Complex II with Succinate

The prodrug NV354 is able to easily pass through cell membranes and once in the cell, it is converted into succinate. Once succinate is in the cell, as a substrate for Complex II, it supplies it with electrons, bypassing the dysfunctional Complex I. Thus, the Electron Transport Chain can function normally again, generating sufficient ATP. This also results in lower lactate production, as it alleviates the dependence on glycolysis to produce ATP. Therefore, NVP354 has the potential to both protect mitochondria and prevent loss of organ function.

Chart 31: Prodrug strategy provides succinate to Complex II



Source: Ehinger et al, 2016

Pre-Clinical Work Expected to Complete in H1 2021

NV354 was originally selected as a succinate prodrug based on its tolerability, oral bioavailability, plasma stability and delivery to organs including the brain. Abliva has already generated pre-clinical proof of concept in experimental models of Complex I dysfunction, showing that it can facilitate bypassing of the mutation. The published pre-clinical data which we have seen demonstrates that NV354:

- Has good delivery to the brain and other organs
- Improves mitochondrial function
- Exhibits efficacy in rodent models of Leigh Syndrome

Good bioavailability in the brain

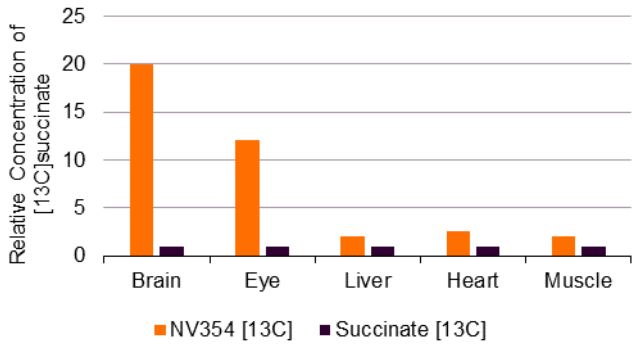
NV354 was able to successfully deliver succinate into cells including within the brain. This bioavailability was significantly higher than orally administered succinate, affirming the efficacy of Abliva's cell permeable pro-drug formulation.

Improved mitochondrial function in Leigh Syndrome patient cells

An earlier analogue of NV354 was found to significantly improve the functioning of mitochondria in cells from Leigh syndrome patients, as revealed by a greater oxygen consumption rate post-administration. Importantly, the increase in oxygen consumption is sufficient to bring the Leigh syndrome cell oxygen consumption to the same level as healthy

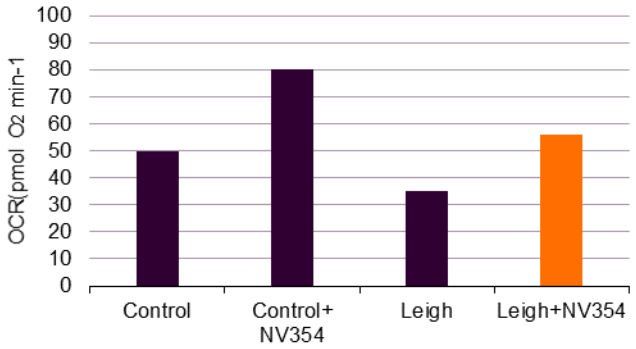
cells. This normalised rate of cellular respiration implies that the NV354-analogue enables a complete bypass of the Complex I dysfunction, which causes the underlying disease.

Chart 32: Higher succinate delivery to organs with NV354*



*Source: Company reports * NV354-analogue succinate prodrug*

Chart 33: Normalised mitochondrial function in Leigh cells



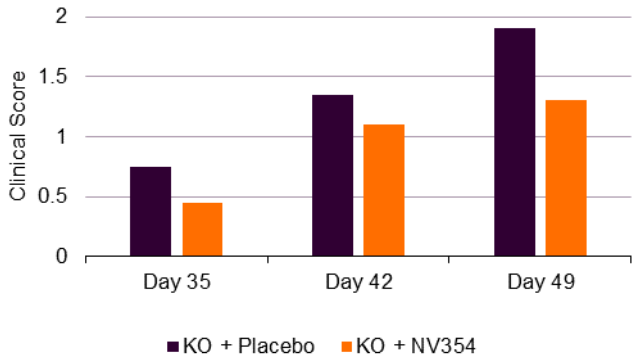
*Source: Company reports * NV354-analogue succinate prodrug*

Ndufs4 KO mice have mutated forms of the NDUFS4 gene, which is responsible for coding for subunits that are part of Complex I. As a result, these mice have mitochondrial Complex I dysfunction and make for suitable models of Leigh's syndrome in humans.

Strong early signs of *in vivo* efficacy

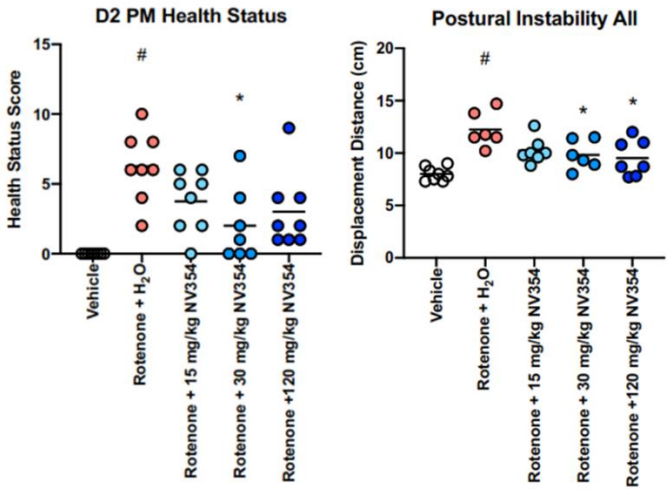
In rodent models of Leigh Syndrome (see margin), NV354 was found to decrease disease severity and improve health status. It reduced the severity of disease progression as revealed by lower clinical scores that assessed neuromuscular degeneration such as paralysis (bottom, left). Furthermore, in rats that were administered a toxin to induce Complex I dysfunction, NV354 was found to reduce the displacement distance of postural instability and improved the health score in comparison to toxin-administered rats not receiving treatment (bottom, right).

Chart 34: NV354 slows motor dysfunction



Source: Company reports
Clinical Score: 1= ataxia, 2= paresia/tilted head, 3= hindlimb paralysis

Chart 35: NV354 improves health status



Source: Company reports

Fast to Market - Phase I in H2 2021

Pre-clinical safety studies are expected to be completed in H1 2021 and are anticipated to pave the way for a phase I start in H2 2021. Given the promising efficacy signals in the pre-clinical data and the lack of current treatments for these severe diseases, we agree with Abliva that NV354 is likely to achieve Fast Track and PRIME designations. It also has the potential to expand to other PMD indications. In a best-case scenario, market entry could potentially be as soon as 2026.

Highly Risk-Adjusted Sales of $38m

In the US and EU/UK, we estimate there are around 1.2k patients with Leigh Syndrome based on an incidence of 1/34k births and a median age of 2.4 years. Given the severity of the disease (life expectancy is <5 years) and the lack of any current treatment options or competitors with drugs specifically targeting Leigh Syndrome, we expect NV354 to achieve a market penetration of ~70% in the US and 50% in the EU/UK. With the first clinical data not expected until 2024, we do not see a launch for this drug until 2026 and have peak sales being reached in 2033.

Table 9: Leigh Syndrome population in the US, EU & UK

Patients	**2020**	**2021**	**2022**	**2023**	**2024**	**2025**	**2026**	**2027**	**2028**	**2029**	**2030**	**2031**	**2032**	**2033**
US	559	564	568	573	578	582	587	592	596	601	606	611	616	621
Penetration	0%	0%	0%	0%	0%	0%	1%	4%	10%	29%	38%	44%	67%	70%
EU & UK	693	697	700	704	707	711	714	718	722	725	729	732	736	740
Penetration	0%	0%	0%	0%	0%	0%	1%	3%	3%	22%	26%	25%	48%	50%
Total patients treated							**12**	**48**	**77**	**332**	**418**	**450**	**769**	**804**
In US							7	26	58	172	228	267	415	434
In EU/UK							5	22	19	160	190	183	354	370

Source: Intron Health estimates

If we assume an annual net price of $250k in the US and $180k in the EU & UK (in-line with orphan drug pricing and the $5m lifetime cost of treating these patients), applying a risk adjustment of 20% results in our 2033 sales forecast of $38m (and $190m without risk-adjustment).

Table 10: Sales forecasts for NV354

$m	**2026**	**2027**	**2028**	**2029**	**2030**	**2031**	**2032**	**2033**
US ($)	250,000	255,000	260,000	265,000	271,000	276,000	282,000	287,000
Risk adjustment	20%							
Risk-adjusted sales	**0**	**1**	**3**	**9**	**12**	**15**	**23**	**25**
EU & UK ($)	180,000	180,000	180,000	180,000	180,000	180,000	180,000	180,000
Risk adjustment	20%							
Risk-adjusted sales	**0**	**1**	**1**	**6**	**7**	**7**	**13**	**13**
Total risk-adj. sales (base case)	**1**	**2**	**4**	**15**	**19**	**21**	**36**	**38**
Total de-risked sales	*3*	*11*	*18*	*74*	*96*	*107*	*181*	*191*

Source: Intron Health estimates

NV354 Could Be Worth ~60% of Current Market Cap

Unlike KL1333, NV354 is wholly-owned by Abliva, so there are no material pay-aways to model. At a WACC of 12% and EBIT margin of 80%, we show that NV354 is worth SEK334m to Abliva, or 62% of the total market capitalisation on 2022 shares.

Table 11: NPV for NV354

US & EU5	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035	2036	2037
Sales	1	2	4	15	19	21	36	38	38	38	38	38
Total EBIT	0	2	3	12	15	17	29	31	31	31	31	31
Taxation (21.4%)	0	0	-1	-3	-3	-4	-6	-7	-7	-7	-7	-7
NOPAT	**0**	**1**	**2**	**9**	**12**	**13**	**23**	**24**	**24**	**24**	**24**	**24**
NPV ($m)	41											
NPV (SEKm)	**334**											
NPV/share	0.47											
as % share price	**62%**											

Source: Intron Health estimates

- We have assumed no sales from ex-US and EU/UK or from disorders other than Leigh Syndrome
- No terminal value beyond 2037

Few Competitors For a Multi-$Bn Market

There are no FDA-approved therapies for the treatment of mitochondrial disease, with the current standard of care focused on symptom alleviation. New drugs in development by other players have the potential to improve clinical symptoms further, but only Abliva's pipeline has the potential to help treat the underlying biology of the disease. Furthermore, Abliva is specifically targeting a subset of the 12.5/100k population with mitochondrial disease – the 5/100k with MELAS/CPEO, whereas their competitors are generally targeting a broader population. Therefore, whilst we anticipate that Abliva will have to share the market, we also think they have the potential to be the market leaders in their niche.

Table 12: Therapeutics in Development for Mitochondrial Disease

Company	Therapeutic	Description	Indication	Phase
PTC Therapeutics	PTC743	Small molecule (oral), modulates oxidative stress	PMD epilepsies	Phase II
Khondrion	Sonlicromanol	Small molecule (oral), modulates oxidative stress	MELAS	Phase II
Reneo	REN001	Small molecule PPARd modulator (oral)	PMM	Phase I
Mitobridge (Astellas)	MA-0211	Small molecule PPARd modulator (oral)	PMM	Phase I
Abliva	KL1333	Small molecule (oral), NAD+ modulator	PMD	Phase I
Abliva	NV354	Prodrug(oral), energy substrate	Leigh	Pre-clinical
Stealth Biotherapeutics	Elamipretide	Cardiolipin-binding peptide	Barth syndrome	Failed at Phase III

Source: Company reports PMM: Primary mitochondrial myopathies

Sonlicromanol pushed into phase IIb despite failure at phase IIa

Khondrion's asset sonlicromanol (KH176) is a novel redox modulator that prevents lipid peroxidation-dependant cell death and counters inflammation. It has been granted orphan designation for MELAS-MIDD spectrum disorders and Leigh Syndrome in Europe and all hereditary mitochondrial respiratory chain disorders in the US. Although the results of the first PIIa KHENERGY study failed to meet movement-related efficacy endpoints, it remains under assessment in a PIIb study due to positive effects observed in mood-related exploratory outcomes and alertness. The KHENERGYZE study is therefore evaluating sonlicromanol in adults with a 3243A>G mutation in their MT-TL1 gene (responsible for MELAS spectrum disorders). A paediatric study is also expected to start later in 2020.

REN001 is taking a different approach

Reneo's REN001 is a selective peroxisome proliferator-activated receptor delta (PPARδ) agonist in development for primary mitochondrial myopathies. PPARδ increases the expression of enzymes that metabolise fats for energy generation, hence its over-activation by REN001 results in the production of excess energy substrates. The mechanism of action is hypothecated to be that this will induce the transcription of genes needed to create new mitochondria. A 12-week clinical study of REN001 in patients with primary mitochondrial myopathies showed it was well tolerated and that its use led to improvements in walking and disease symptoms. Therefore, a clinical trial of 200 adult PMM patients is expected to start in H1 2021.

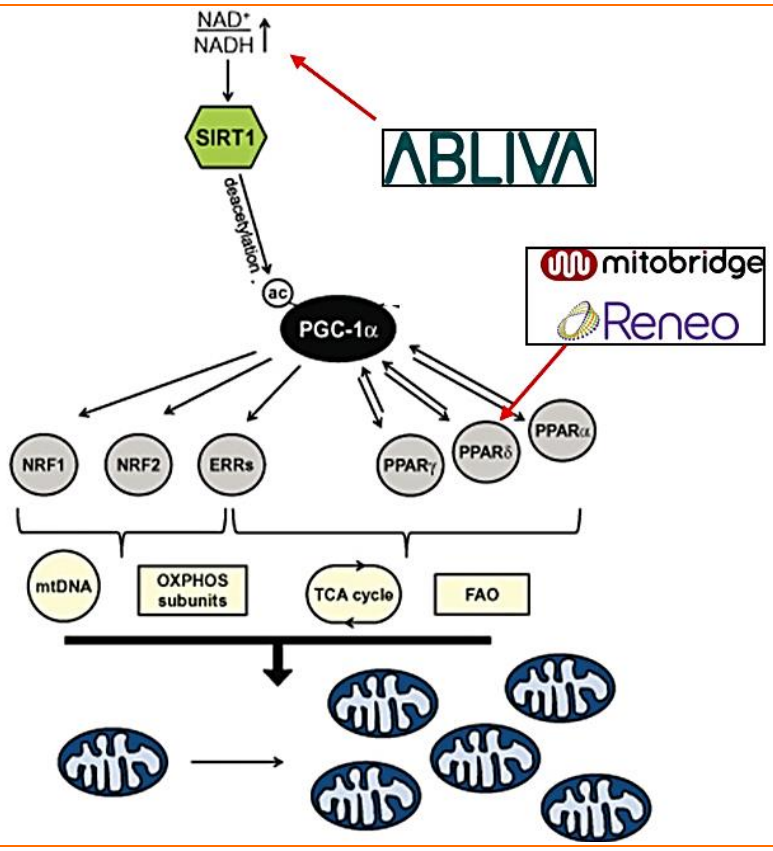
MA-0211 (ASP0367) is another PPARδ agonist

Like REN001, Mitobridge's MA-0211 is another selective PPARδ agonist; it has also been granted Fast Track Designation from the FDA. Pre-clinical data suggests that it could improve exercise intolerance and fatigue in primary mitochondrial myopathy patients by increasing the number and enhancing the function of mitochondria in patient cells. It is anticipated to enter a phase II/III study to validate the efficacy and safety in these patients.

Abliva's Drug May Have Competitive Edge

Reneo and Mitobridge both target the same genetic mutation as Abliva's KL1333, but they solely act on PPARδ, which is implicated in fatty acid oxidation. By contrast, KL1333 acts on an upstream target (PGC-1α) by increasing the NAD+/NADH ratio, which ultimately has a much broader effect on mitochondria function and proliferation. We believe this could give Abliva's pipeline an edge over the competition by potentially leading to higher efficacy in these very sick patients.

Chart 36: KL1333 has a differentiated mode of action



Source: Company reports

Financials

Total Revenues

We combine our forecasts from KL1333 and NV354 to show that the company reaches an inflection point in 2024, when revenues move sharply upwards.

Table 13: Abliva total revenues forecast

SEK (000s)	2019A	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030
KL1333 Revenues	0	0	0	0	0	28,936	117,039	295,885	598,445	877,569	1,071,170	1,191,725
NV354 Revenues	0	0	0	0	0	0	0	4,277	17,423	30,085	121,635	156,667
Total Pipeline Revenue	**0**	**0**	**0**	**0**	**0**	**28,936**	**117,039**	**300,162**	**615,868**	**907,654**	**1,192,805**	**1,348,392**
growth	N/A	N/A	N/A	N/A	N/A	N/A	304%	156%	105%	47%	31%	13%
Other Revenues	134	305	305	305	305	305	305	305	305	305	305	305
growth		56%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
Total Revenues	**134**	**305**	**305**	**305**	**305**	**29,241**	**117,344**	**300,467**	**616,173**	**907,959**	**1,193,110**	**1,348,697**
growth	**N/A**	**128%**	**0%**	**0%**	**0%**	**9487%**	**301%**	**156%**	**105%**	**47%**	**31%**	**13%**

Source: Intron Health estimates

P&L Heading Towards Breakeven

We have carefully modelled all the lines in our P&L, including the royalties paid on KL1333 sales alongside COGS, though we put the milestones into the cash flow and do not include in the P&L. We have included an incremental SEK3m ($400k) of launch costs in 2024 and another SEK11m ($1.4m) in 2025. As KL1333 is a rare disease drug with no existing market, we expect the initial ramp to be very slow and hence little marketing spend in the first year as very few treatment centres will be targeted.

Table 14: Abliva summary P&L

SEK (000s)	2020	2021	2022	2023	2024	2025
Revenues	**305**	**305**	**305**	**305**	**29,241**	**117,344**
Cost of goods	0	0	0	0	-2,924	-11,734
as % of sales	0.0%	0.0%	0.0%	0.0%	-10.0%	-10.0%
Royalties paid out	0	0	0	0	-248	-11,460
Gross profit	**305**	**305**	**305**	**305**	**26,069**	**94,149**
Gross margin	100%	100%	100%	100%	89%	80%
External expenses	-60,000	-63,000	-66,150	-69,458	-72,930	-76,577
growth	-5.0%	5.0%	5.0%	5.0%	5.0%	5.0%
Personnel cost	-16,000	-16,800	-17,640	-18,522	-22,226	-33,340
growth	8%	5%	5%	5%	20%	50%
D&A	-2,553	-2,560	-2,589	-2,596	-2,869	-3,190
EBIT	**-78,248**	**-82,055**	**-86,074**	**-90,271**	**-71,957**	**-18,957**
Net interest	194	875	1,402	938	474	184
Tax	0	0	0	0	0	0
Net income	**-78,054**	**-81,179**	**-84,671**	**-89,333**	**-71,482**	**-18,774**
Number of shares (basic, 000s)	241,767	489,673	709,673	709,673	709,673	709,673
EPS	**-0.32**	**-0.17**	**-0.12**	**-0.13**	**-0.10**	**-0.03**

Source: Intron Health estimates

Rights Issues Keep Abliva Cash Positive

Abliva raised SEK54.1m in new equity in May 2020 by issuing 83.7m new shares. We expect this cash to keep them afloat until mid-2021, when another raise will be necessary (Abliva has no debt). To simplify things, we assume that the company raises ~SEK377m on 1st July 2021 in order to remain cash positive until 2025. In practice, annual rights issues may be employed as opposed to a large equity issuance in one year. Our valuation takes this into account as we value the shares using the 2022 share number (c. 710m).

Table 15: History of rights issues to make ends meet

Shares (m)	2019	2020	2021	2022	2023	2024	2025
Starting	**78,500**	**171,575**	**241,767**	**489,673**	**709,673**	**709,673**	**709,673**
Issuance	93,075	70,192	220,000	0	0	0	0
Buybacks		0	0	0	0	0	0
Other		0	27,907*	220,000*	0	0	0
Ending	**171,575**	**241,767**	**489,673**	**709,673**	**709,673**	**709,673**	**709,673**
Average share price (assumption)		N/A	0.86	0.94	1.04	1.14	1.26
Share issuance raise		**54,098**	**377,520**	**0**	**0**	**0**	**0**
Cash and cash equivalents	**58,319**	**35,167**	**331,431**	**247,797**	**147,648**	**54,523**	**8,106**

*Source: Company reports, Intron Health estimates * Adjustments for phasing of share issuances*

We see ~3x Upside to Current Price

We value Abliva using a Sum-of-the-Parts methodology. We only value the cash flows over 2021-37 and do not include a terminal value in our calculations. We also use very high risk adjustments and ignore some potential sources of value including off-label sales, regional sales outside of US/EU/UK and all of the R&D programmes outside of KL1333 and NV354. For example, we do not ascribe any value to the NVP015 project out-licensed to BridgeBio/Fortify, which has potential value of $60m. Our SOTP implies a target price of SEK2.25/share and assumptions include:

- WACC of 11%
- Tax of 21.4% (Swedish corporate tax rate after tax losses used up)
- R&D and G&A expenses to 2027 are NPV'd and deducted; this is sufficient to bring the two major drugs to approval

Table 16: Intron Health valuation of Abliva

	Value (SEKm)	Value / share (SEK)
KL1333 (risk-adjusted)	1,348	1.90
NV354 (risk-adjusted)	334	0.47
Other revenues to 2025	1	0.00
Corporate & R&D costs to 2027	-343	-0.48
Net (debt) cash in 2022	248	0.35
Total (risk-adjusted) value	**1,588**	**2.24**
Share price	0.76	
Upside multiple	**2.9x**	

Source: Intron Health estimates

De-risking Over Time to Add Incremental Upside

If we were to remove our risk adjustments for KL1333 and NV354 (currently risk-adjusted down to 20%), then we calculate that the potential upside increases from 3x to 17x. However, it is important to stress that we would expect such a de-risking to take many years and is obviously dependent on there being successive positive readouts.

2 Deals in the Space Show Abliva Likely Undervalued

In January 2018, Astellas acquired Mitobridge for $225m plus another potential $225m, dependent on the progress of various programmes in clinical development. Mitobridge has a small molecule PPARδ modulator in phase I trials for primary mitochondrial myopathies (PMM) so is at a similar stage to Abliva.

Reneo Pharma also has a PPARδ modulator and in December 2020, Novo Ventures and Abingworth backed a $95m Series B financing round following on from an initial $50m financing agreed two years ago. The company has planned a phase II trial in PMM this year and expect the $95m will last until readout. We are therefore satisfied that the mitochondrial disease space is an active one and that valuations multiples higher than Abliva's can be easily supported.

Non-Core and Partnering Projects

We exclude all three of Abliva's non-core research programmes from our company valuation due to the early stage of development, lack of data and uncertainty around partnering. Nevertheless, these projects could bring real value to the company in time and so we view them as potential sources of future upside.

NVP025 in Mitochondrial Myopathies

Abliva's mitochondrial myopathy drug candidate, NVP025, is a potent cyclophilin inhibitor. It is currently undergoing preparation ahead of its entry into pre-clinical trials.

NVP025 aims to protect the mitochondria in muscles from disturbed calcium handling and subsequent muscular dystrophy, thus preventing the muscle weakness associated with this disease. Working in collaboration with the Karolinska Institute in Stockholm, Abliva has found that cyclophilins have the ability to slow disease progression and increase survival in an experimental model of mitochondrial myopathy. As this drug has yet to enter pre-clinical trials, in the absence of any data we exclude it from our valuation of the company.

NeuroSTAT in Traumatic Brain Injury

This technology is Abliva's only treatment under development for an indication outside of primary mitochondrial disease. NeuroSTAT is a

patented non-allergenic lipid emulsion formulation of cyclosporin A indicated for the treatment of severe traumatic brain injury. It has orphan drug designation in Europe and the US, has IND approval and is phase II-ready with positive phase Ib patient safety data as well as biomarker efficacy signals, but Abliva are seeking to partner it before committing to bringing it to the next stage of development. Once a partner is found, Abliva estimate that it is likely to take another 6-7 years of development before commercial launch. Given the uncertainties around this, we currently exclude this programme from our company valuation.

Traumatic brain injury impacts 3 million per year in the US

Around three million people each year suffer from severe traumatic brain injury in the US alone, defined as a blow to the head or a penetrating head injury that disrupts the normal function of the brain. Of these, around 290k are hospitalised and 80-90k people experience the onset of long-term disability resulting from it. This is the population that we would envisage as being the target market.

Cyclosporin prevents further brain cell damage

Cyclosporin inhibits activation of the mitochondrial permeability transition pore, a key event in TBI pathology. This means the mitochondria can withstand more stress and secondary neuronal injury and cell death can be prevented. The novel and patented lipid emulsion formulation of cyclosporin A prevents chremophore associated anaphylaxis and is efficacious and safe.

Studies suggest candidate is efficacious

Studies like that of the 2017 Copenhagen Head Injury Cyclosporin (CHIC) Study have demonstrated it is safe and well-tolerated, confirmed that the drug passes the blood-brain barrier in patients with TBI and showed signs of clinical effectiveness, as measured by biomarkers (GFAP, NF-L, Tau, and UCH-L).

FDA have approved development plans

Abliva has a ready design for future development including an FDA approved phase II trial design that constitutes a placebo-controlled study in 75-105 patients across 5 countries. This will be succeeded by a larger study of 2-3 years duration. The FDA has also awarded the drug fast track designation.

NV556 in Non-Alcoholic Steatohepatitis

NV556 is in development for NASH, which afflicts up to 3-5% of the global population. NASH is a progressive liver disease involving the incorporation of fat which leads to scarring and inflammation. With hepatitis C becoming rarer, it is expected to become the #1 cause of liver transplantations in the US in the near future.

NV566 is a potent cyclophilin inhibitor derived from Abliva's Sangamide class of compounds and is thought to have a direct anti-fibrotic effect to counter liver fibrosis. Pre-clinical findings revealed it may be more effective in patients with more advanced NASH may also have efficacy in other liver fibrosis conditions such as Primary Biliary Cholangitis and Primary Sclerosing Cholangitis. Abliva is awaiting a partnering agreement to progress this candidate into further development. Given the amount of interest in the NASH space today, we believe it is entirely possible that Abliva could find an effective partner for this drug, but until we see an update, we do not currently include this drug in our company valuation.

Company Background

Abliva, previously known as NeuroVive, is a Swedish biotech company founded in 2000 by Dr Eskil Elmer. The company was listed on Aktietorget in 2008 before entering the Swedish NASDAQ in 2013. The company originally focused on the development of cyclosporin A for mitochondrial protection during heart surgery and cardiac reperfusion injury (CicloMulsion). Both programs failed at the clinical trial stage and were subsequently discontinued. Abliva's new strategy is to develop or in-license compounds for the treatment of mitochondrial diseases, with two candidates KL1333 and NV354 publishing positive data.

Board and Management

Erik Kinnman - CEO

Erik is a seasoned life science executive having held several leadership positions in biopharmaceutical companies including AstraZeneca and Sobi. His expertise includes clinical development, business strategy and business development. He is an M.D. certified in Neurology and Pain Management and holds a PhD and Executive MBA from the Stockholm School of Economics.

Catharina Johansson - CFO

Catharina joined Abliva in 2013, having previously held roles in medtech growth enterprises with multinational operations including the medical device company Cellavision. She holds a MSc in Business and Economics.

Eskil Elmer - CSO & Vice President Discovery

Eskil is a consultant physician, adjunct professor of basal and clinical neurophysiology at Lund University (Sweden) and group leader of the Mitochondrial Medicine lab at the department of Clinical Neurophysiology. Dr Elmer is a patentee and cofounder of Abliva AB.

Magnus Hanson - CMO

Magnus joined Abliva AB in 2008 and has previously served as a consultant physician at Skane University Hospital and as a Senior Scientist at Abliva. Dr Hansson has extensive experience in the area of mitochondrial medicine and has overall charge of Abliva's pre-clinical and clinical development programs. He holds a PhD in experimental brain research from Lund University, Sweden.

David Laskow-Pooley - Chairman

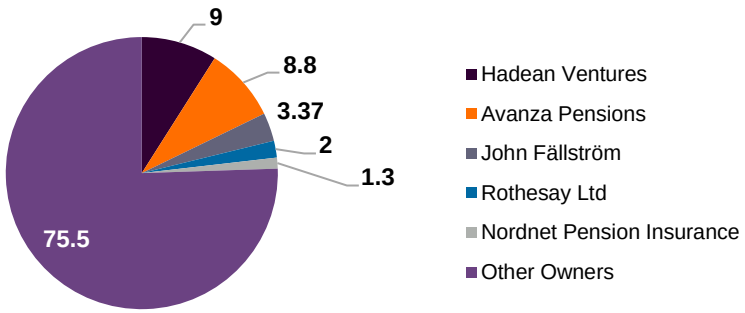
David was elected to the board of Abliva in 2016 and became Chairman in 2017. In addition, he is CEO of Pharmafor and a board member of Marker Therapeutics and LREsystem. David holds a pharmacy degree from the Sunderland School of Pharmacy with more than 40 years in the pharmaceutical industry and extensive experience in starting and developing companies in the industry.

Shareholder Structure

As of 29th May 2020, Abliva has been listed on the Stockholm Nasdaq OMX Small Cap under its current name (previously known as Neurovive Pharmaceutical AB).

The company is currently owned mainly by small private investors. The market capitalisation amounts to SEK230m (as of January 2021). Hadean Ventures is the largest shareholder, a European life science specialist venture capital firm based in Oslo with a particular focus on the Nordic region. This €85m fund is backed by leading private and institutional investors and has a strategic focus on 'under-ventured' regions. Following significant due diligence, Hadean invested in Abliva in Q3 2020 and now holds a seat on the Board.

Chart 37: Shareholder Structure by Percentage



Source: Company reports

Financial Statements

Group P&L

Table 17: Abliva AB P&L

SEK (000s)	2019A	2020	2021	2022	2023	2024	2025	CAGR 20-25
Revenues	**134**	**305**	**305**	**305**	**305**	**29,241**	**117,344**	**228.9%**
growth	2580%	128%	0%	0%	0%	9487%	301%	
Cost of goods	0	0	0	0	0	-2,924	-11,734	
growth							301.3%	
as % of sales	0.0%	0.0%	0.0%	0.0%	0.0%	-10.0%	-10.0%	
Royalties paid out	0	0	0	0	0	-248	-11,460	
Gross profit	**134**	**305**	**305**	**305**	**305**	**26,069**	**94,149**	**214.7%**
Gross margin	**N/A**	**100.0%**	**100.0%**	**100.0%**	**100.0%**	**89.2%**	**80.2%**	
External expenses	-63,133	-60,000	-63,000	-66,150	-69,458	-72,930	-76,577	5.0%
growth	13.1%	-5.0%	5.0%	5.0%	5.0%	5.0%	5.0%	
as % of sales	N/A	N/A	N/A	N/A	N/A	N/A	N/A	
Personnel cost	-14,872	-16,000	-16,800	-17,640	-18,522	-22,226	-33,340	15.8%
growth	2.9%	7.6%	5.0%	5.0%	5.0%	20.0%	50.0%	
as % of sales	N/A	N/A	N/A	N/A	N/A	N/A	N/A	
D&A	-2,379	-2,553	-2,560	-2,589	-2,596	-2,869	-3,190	
as % of sales	N/A	N/A	N/A	N/A	N/A	N/A	N/A	
OOI/OOE	3,175	0	0	0	0	0	0	#DIV/0!
as % of sales	N/A	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	
EBIT	**-77,075**	**-78,248**	**-82,055**	**-86,074**	**-90,271**	**-71,957**	**-18,957**	**-24.7%**
EBIT margin	N/A	N/A	N/A	N/A	N/A	N/A	-16.2%	
growth	5.1%	1.5%	4.9%	4.9%	4.9%	-20.3%	-73.7%	
Interest expense	-46	-40	-40	-40	-40	-40	-40	
Interest received	0	234	915	1,442	978	514	224	
Associates/JVs	121	0	0	0	0	0	0	
Pre-tax profit	**-77,000**	**-78,054**	**-81,179**	**-84,671**	**-89,333**	**-71,482**	**-18,774**	
Tax	0	0	0	0	0	0	0	
Effective tax rate	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	0.0%	
Net profit	**-77,000**	**-78,054**	**-81,179**	**-84,671**	**-89,333**	**-71,482**	**-18,774**	
Minorities	6	0	0	0	0	0	0	
Net income	**-76,994**	**-78,054**	**-81,179**	**-84,671**	**-89,333**	**-71,482**	**-18,774**	**-24.8%**
Number of shares (basic, 000s)	171,575	241,767	489,673	709,673	709,673	709,673	709,673	
EPS	**-0.45**	**-0.32**	**-0.17**	**-0.12**	**-0.13**	**-0.10**	**-0.03**	**-39.4%**
growth	-52.1%	-28.1%	-48.7%	-28.0%	5.5%	-20.0%	-73.7%	

Source: Intron Health estimates

Group Balance Sheet

Table 18: Abliva AB balance sheet

SEK (000s)	2019A	2020	2021	2022	2023	2024	2025
ASSETS							
Intangible assets	74,686	75,230	76,784	77,367	89,959	104,778	119,864
PP&E	786	747	709	674	640	608	578
Associates	13,101	13,101	13,101	13,101	13,101	13,101	13,101
Other non-current assets	0	0	0	0	0	0	0
Non-current assets	**88,573**	**89,078**	**90,595**	**91,142**	**103,700**	**118,488**	**133,543**
Inventories	0	0	0	0	0	8,772	23,469
Trade/other receivables	1,141	1,255	1,381	1,519	1,671	1,838	2,021
Other current assets	459	459	459	459	459	459	459
Cash & cash equivalents	58,319	35,167	331,431	247,797	147,648	54,523	8,106
Non-current assets	**59,919**	**36,881**	**333,271**	**249,775**	**149,778**	**65,592**	**34,055**
Total assets	**148,492**	**125,959**	**423,865**	**340,916**	**253,478**	**184,080**	**167,598**
LIABILITIES							
Borrowings	0	0	0	0	0	0	0
Other non-current liabilities	361	361	361	361	361	361	361
Non-current liabilities	**361**	**361**	**361**	**361**	**361**	**361**	**361**
Borrowings	0	0	0	0	0	0	0
Trade payables	14,234	15,657	17,223	18,945	20,840	22,924	25,216
Provisions	0	0	0	0	0	0	0
Other current liabilities	6,102	6,102	6,102	6,102	6,102	6,102	6,102
Current liabilities	**20,336**	**21,759**	**23,325**	**25,047**	**26,942**	**29,026**	**31,318**
Total liabilities	**20,697**	**22,120**	**23,686**	**25,408**	**27,303**	**29,387**	**31,679**
EQUITY							
Share capital	9,298	9,298	9,298	9,298	9,298	9,298	9,298
Additional paid in capital	592,980	647,078	1,024,598	1,024,598	1,024,598	1,024,598	1,024,598
Translational reserves	619	619	619	619	619	619	619
Retained earnings (losses)	-475,107	-553,161	-634,341	-719,012	-808,345	-879,827	-898,601
Minority interests	5	5	5	5	5	5	5
Total equity	**127,795**	**103,839**	**400,179**	**315,508**	**226,175**	**154,693**	**135,919**
Total liabilities and equity	**148,492**	**125,959**	**423,865**	**340,916**	**253,478**	**184,080**	**167,598**

Source: Intron Health estimates

Group Cash Flow

Table 19: Abliva AB Cash Flow

SEK (000s)	2019A	2020	2021	2022	2023	2024	2025
Operating income	-77,074	-78,248	-82,055	-86,074	-90,271	-71,957	-18,957
D&A	2,379	2,553	2,560	2,589	2,596	2,869	3,190
Other non-cash adjustments	121	0	0	0	0	0	0
Change in inventories	0	0	0	0	0	-8,772	-14,697
Change in trade receivables	0	-114	-126	-138	-152	-167	-184
Change in trade payables	0	1,423	1,566	1,722	1,895	2,084	2,292
Other working capital movements	2,208	0	0	0	0	0	0
Interest received	0	234	915	1,442	978	514	224
Interest paid	-46	-40	-40	-40	-40	-40	-40
Tax paid	0	0	0	0	0	0	0
Cash flow from operations	**-72,412**	**-74,192**	**-77,180**	**-80,499**	**-84,994**	**-75,469**	**-28,172**
Purchase of PP&E	-69	-71	-67	-64	-61	-58	-55
Disposals of PP&E	0	0	0	0	0	0	0
Purchase of intangibles	-2,626	-2,987	-3,009	-3,071	-3,095	-3,598	-4,191
Milestones paid out	0	0	-1,000	0	-12,000	-14,000	-14,000
Cash flow from investment	**-2,695**	**-3,058**	**-4,076**	**-3,135**	**-15,155**	**-17,656**	**-18,246**
Proceeds from share issuance	107,780	54,098	377,520	0	0	0	0
Other	-309	0	0	0	0	0	0
Cash flow from financing	**107,471**	**54,098**	**377,520**	**0**	**0**	**0**	**0**
Beginning cash & cash equivalents	**25,951**	**58,319**	**35,167**	**331,431**	**247,797**	**147,648**	**54,523**
Change in cash	32,364	-23,152	296,264	-83,634	-100,149	-93,125	-46,417
FX impact	4	0	0	0	0	0	0
Ending cash & cash equivalents	**58,319**	**35,167**	**331,431**	**247,797**	**147,648**	**54,523**	**8,106**

Source: Intron Health estimates

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Full 12-month historical recommendation changes are available on request

This report was produced by New Street Research LLP. 52 Cornhill, London, EC3V 3PD Tel: +44 20 7375 9111.

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