

Plan clears for final phase

Abliva is approaching pivotal study with KL1333

Plans are becoming clearer for Abliva's registrational Phase 2/3 study for KL1333 in patients with primary mitochondrial disease. Patients will be given up to twice as high doses as in the Phase 1b study, signaling that management is not concerned about the adverse events profile and that the preliminary efficacy demonstrated in Phase 1b may be amplified in a new study.

Double endpoints of energy shortages

Abliva's study is intended to be conducted in 120-180 patients with mutations in mitochondrial DNA, the largest of which is the MELAS-MIDD patient group. The study looks at two different primary endpoints for energy deficiency: a patient estimate of perceived fatigue and a functional test of muscle strength over 30 seconds. Both measures are evaluated after 12 months of double-blinded treatment.

Most registrational studies use a primary endpoint, which forms the basis for the application for permission to sell a new drug. However, within the mitochondrial disease spectrum, we notice a more varied construction of endpoints, which may allow Abliva to seek approval even if only one of the two primary endpoints is positive in the study.

NV354 takes steps towards human testing

Abliva recently announced that the company is completing the preclinical package for NV354, the company's second drug candidate. The plan is to start studies on healthy volunteers next year. NV354 is a pro-drug of succinate, a chemical preparation to get the energy substrate succinate to cross the cell's membranes to help patients with the deadly disease Leigh syndrome.

Funding for new study remains to be secured

Now management needs to find funding to secure the implementation of the KL1333 study. We estimate that the company's cash position will amount to SEK 35 million at the end of the year unless financiers inject new capital before then. We estimate that management is seeking approximately SEK 300 million from institutional investors.

We link the recent weak price development to this extensive overhang of new shares. From our perspective, the unique opportunity to move KL1333 with orphan drug status to market approval in 2025 dominates through a limited investment. The fair value for the company, which is partly a function of the share issue price, amounts to SEK 1.6.

Abliva

Analysis	
Date	6 October 2021
Analyst	Sten Westerberg
Basfakta	
Industry	Drug Development
Chairman	David Laskow-Pooley
Ceo	Ellen K. Donnelly
Noteringsår	2013
Listing	Nasdaq Stockholm
Ticker	ABLI
Share price	SEK 0.55
Number of shares, million	403.0
Market capitalization, SEK million	302
Net cash 2021p, SEK million	35

Website: www.abliva.com

Price development last year



Source: Refinitiv

Forecasts & Key figures, SEK million

	2019	2020	2021p	2022p	
Turnover	0.1	0.2	0	0	
Results f. tax	-77	-60	-81	-150	
Net profit	-77	-60	-81	-150	
Earnings per share	-0.5 SEK	-0.1 SEK	-0.2 SEK	-0.4 SEK	
Rörelsemarginal	neg	neg	neg	neg	
Likvida medel	58	62	348	199	
New share issue	108	87	398	0	
Direktavkastning	0%	0%	0%	0%	

Source: Company, Analysis Guide

Investment thesis

Rare diseases need treatments

Abliva conducts drug research within a group of different primary mitochondrial diseases (PMD). Mitochondria are the cell power plant and the morbid conditions often stem from severe metabolic disorders, which can sometimes be linked to mutations and gene changes. The course of the disease often starts early in life and significantly impacts the quality of life for those affected.

KL1333 is being developed for the treatment of PMD patients with mitochondrial DNA mutations, the largest of which is MELAS-MIDD, a syndrome linked to mutation m.3243A>G. The syndrome affects about 20-30 people per million inhabitants. There is currently no other documented effective treatment that can similarly increase energy production in these weak patients, potentially satisfying a major underlying medical need.

Promising results in Phase 1 study

The company's strength largely rests on a relatively large documentation of the safety and dosage of the substance in Phase 1. This data also includes efficacy in eight PMD patients during the 10-day dosing of 50 mg or placebo. These data show a positive trend in both muscle fatigue and general fatigue:

Signals of increased efficacy with higher exposure in the blood of KL1333,

Positive trend for three various placebocontrolled clinical measurements;

Side effects that can be resolved with dividing the dose over the day

Clear medical need in a small group of patients.

Short-term challenges and long-term potential

In discussions with the FDA, Abliva has been given the go-ahead to focus on a registrational Phase 2b/3 study to test whether KL1333 can be approved. The cost of conducting this study on 120-180 patients is estimated by the company at USD 30-40 million, money that is currently missing.

For this limited amount, shareholders have the option to advance KL1333 on the basis of the Phase 1 data towards a possible launch in 2025. Our forecast for the MELAS population eligible for treatment amounts to at least 12,000 patients in the US and EU, representing a future market of approximately USD 1.5 billion.

Taking into account dilutive new issues, we see a fair value for the share of SEK 1.6, well above today's share price.

We estimate the probability of launch in 2025 at 23 percent.

Plan for KL1333 study complete

Abliva's plan for a registrational Phase 2b/3 study with KL1333 depends on the company finding funding. A recent meeting of company management revealed that they intend to gradually increase the dose in the Phase 2/3 study to 100 mg compared to the Phase 1 dose of 50 mg. The dose is given twice daily to reduce the incidence of side effects on the gastro-intestinal tract.

The study will recruit 120-180 patients and the company expects to be able to do this over a 12-month period. The study would then at be fully recruited in the first half of 2023. Thereafter, patients should be on treatment for 12 months with continuous evaluation of two primary endpoints, fatigue and muscle weakness. An initial evaluation is made approximately halfway through the study and a final result may be completed in 2024. In our main scenario, KL1333 has a 23 percent chance to launch in 2025.

Two-part endpoint may increase the chance of success

The study protocol will contain two primary endpoints. One measure is a patient record of perceived fatigue. This scale is based on previous fatigue scales for patients with neurological disorders but has been reworked by Abliva together with a specialist to better measure the symptoms of fatigue experienced by patients with primary mitochondrial disease (PMD). The form contains a number of questions where the patient is asked to record the impact of fatigue on their daily activities.

The second primary endpoint is a functional test of the patient's muscle weakness. The patient rises from a chair and sits again as many times as they can within 30 seconds, a so-called 30 second Sit-To-Stand (STS) test. The patient's ability to do this movement may vary greatly depending on disease impact. A stroke patient can manage STS as little as 8 times in one minute, while a young healthy man can manage an average of 50 times in a minute.

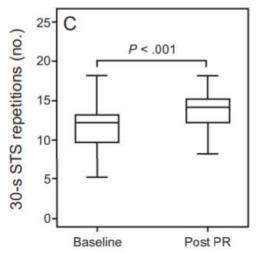
In patients with chronic obstructive pulmonary disease, the scores in a 30-second test ranged from 5-15 times STS with an average of 10¹. In the light of this figure, Abliva's results in 5 out of 6 patients in Phase 1b may appear very promising, namely to improve the number of STS by just over 2 in just over 10 days of treatment compared to 0.5 in placebo (see picture below).

In another study in patients with chronic obstructive pulmonary disease (COPD), 96 people showed an ability of 10-15 STS at the study classification with an average of 12. After a period of lung rehabilitation, patients improved the number of sit-to-stand

¹ Int J Chron Obstruct Pulmon Dis. 2012; 7:537-42.

average twice during a 30-second test, which was considered a clinically meaningful improvement (see picture below).

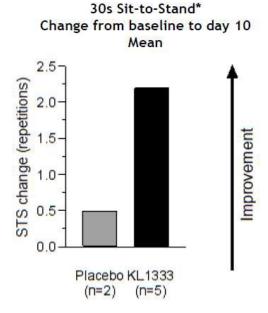
Number of STS for thirty seconds in COPD sufferers



Zanini (Zanini) and to the Respiratory Care October 2019, 64 (10) 1261-1269 (Post PR = After four weeks of lung rehabilitation)

Source:

Outcome for KL1333 on STS 30s in



*) One of the six patients receiving KL1333 was unable to perform the STS test and was excluded.

Dual primary endpoints in PMD

Registrational studies usually contain only one primary endpoint, such as survival in cancer patients or number of heart attack in cardiovascular disease, but there are exceptions to that rule. Studies on primary mitochondrial disease have a history of sometimes including two primary endpoints, as Abliva intends to do.

A large Phase 3 study presented by Stealth Therapeutics in December 2019 in patients with primary mitochondrial myopathy

(PMM), a breakdown of muscles in patients with damaged mitochondria, combined a functional measure, six minutes of walking (Six Minute Walk Test, 6MWT) with a patient-rated goal of fatigue (PMMSA). This study failed to demonstrate an improvement in 6MWT after 24 weeks of treatment compared to placebo.

In the 200 PMM patients' large STRIDE study that Reneo Pharmaceuticals is currently recruiting for, the primary endpoint is a functional 12-minute walking test (see below). The difference between baseline and treatment is determined after 24 weeks of treatment.

A possible positive effect of Abliva choosing a dual primary endpoint could be that the study could then be viewed as positive if one of the measurements is unable to show positive effect. Should both endpoints be positive, we believe this may be reflected in a broader approval. This study design can be distinguished from so-called 'coprimary endpoints', a design in which both endpoints need to be positive for the study to be said have reached their goal.

Mitochondrial disease Landscape

				Nι	ımber Primary		
Project	Company	Phase	Type	Time	pt.	readout	Endpoint
REN001	Reneo	Phase 2/3	PMM	24	200	2023	12MWT
KENUUI	Pharmaceuticals	rhase 2/3		weeks	200	2023	1 2 IVI W 1
ASP0367	Astellas	Phase 2/3	PMM 	52	149	October 2023	6MWT
				weeks	17)	October 2023	OIVI VV I
PTC743	PTC Therapeutics	Phase 2/3	MELAS	48	60	September 1,	Epileptic
				weeks		2021	seizure
CY6463	Cyclerion	Phase 2a	MELAS	29	20	November, 2021	Safety
	Therapeutics	r nase za		days	20		
Sonlicro-	Khondrian B.V.	Phase 2	MELAS	29	27		Cognitive
manol	Kilolidilali B.V.	rnase 2	MELAS	Days	21		functions

^{*)} Primary Mitochondrial

Source: clinicaltrials.gov, corporate communications

Reneo begins Phase 2b trial

Reneo Pharmaceutical (NASDAQ: RPHM) is a company active in the development of medicines for rare mitochondrial diseases. The company announced in July that the first patient had been dosed in the STRIDE study, a registrational study designed to evaluate the efficacy and safety of REN001 in patients with primary mitochondrial myopathy (PMM), a slightly different population than Abliva. Reneo is valued at USD 215 million with cash at USD 167 million at the end of the latest quarter. This valuation is based on the REN001 project, which is also evaluated in a couple of indications other than PMM.

REN001 is a PPAR δ -agonist, which accelerates the conversion of fat and glucose into energy in the patient's mitochondria. The substance is given 100 mg once a day during the 24 weeks of STRIDE. Approximately 200 adult patients with PMM caused by

changes in mitochondrial DNA are expected to participate and an initial topline result may come as early as 2023.

The primary endpoint in STRIDE is the change in distance that the patient has time to walk during a 12-minute walking test, i.e. a functional endpoint. Secondary endpoints consist of two different changes in patient-rated exhaustion and fatigue. We note that the company registered the STRIDE study on clinicaltrials.gov in September 2020 and that the first patient under treatment was estimated at the beginning of 2021. In July this year, the company announced that the first patient was treated in the study, possibly illustrating a certain amount of time that large studies in rare patient groups are often associated with before recruitment takes off.

STRIDE decision based on Phase 1 study

Reneo Pharmaceuticals based its decision to initiate STRIDE on a 12-week Phase 1b open-label study in 23 patients with PMM completed in 2020. A majority of patients then went on to participate in a 36-week extension of treatment. Compared to baseline, the patients, all of whom received REN001 for 12 weeks, showed an average increase of 104 metres in a 12-minute walking test (12MWT) and an increase in oxygen consumption (VO2).

Financial discussion and valuation

In the valuation of Abliva, we have assumed that before the end of the year, the company issues shares for SEK 318 million, or USD 37 million, at a price of 55 cents, just below the current share price. This would entail a new issue of 577 million shares in addition to the 403 million currently issued. This transaction is significant and the uncertainty about the transaction is likely an explanation for the share price's decline during the year. Any announcement of this new share issue is anticipated with great interest from the company's shareholders.

Given the uncertainty surrounding an impending large new share issue, we treat Abliva in a present value calculation with a relatively high return requirement, 15 percent, which can be lowered once the step to finance the Phase 2/3 study is completed. In addition, a long-term valuation of KL1333 should include a further major new share issue to reflect market investments in launch and manufacturing in the US and EU, which we roughly estimate at just over SEK 1 billion. More likely, the company will sell the rights to KL1333 after a successful Phase 3 study, but we expect that the value of such a deal will be based on the opportunity cost for Abliva's shareholders to build their own organization in the US and EU (see table below).

Sales forecast scenario

In our valuation of the Abliva share, we assume a conservative scenario for KL1333 and its chances of reaching the market. We start from the population of 12,000 patients with primary genetic mitochondrial disease in MELAS-MIDD and KSS-CPEO, which the

most seriously ill. With prevalence rates of 3.5 per 100,000 and 1.5 per 100,000 respectively, the total population of these two diseases is 42,000 in the US and Europe, but at present we have too little information to determine whether a larger proportion of this population is eligible for medicine. In our forecasts, we have therefore set the company's target group, Target Patient Population, at 28 percent of 42,000.

Pricing medicines for rare diseases (orphan medicines) is difficult to ascertain. Prices of upwards of USD 500,000 per patient per year can be required for gene therapy-based treatments. Some enzyme inhibitors sell for up to USD 250,000 per patient per year. In a 2019 study, the average price for Orphan Drugs in 2017 in the U.S. was USD 123,000 per patient and year². Evaluate Pharmaceuticals concluded in its 2018 compilation that the average price of an orphan drug in the United States was USD 150,000 per patient for year. Two to outliers, the median price ended up at USD 110,000 for patient for year.

In our calculation, we have adopted a sales price in the US for KL1333 of USD 150,000 per patient per year, slightly higher than the median price for orphan drugs in the US. In Europe, we expect a price of USD 85,000 per year, in line with reimbursements for other orphan medicinal products. Furthermore, we expect the company to reach up to 70 percent of the seriously ill part of the patient group by 2032, which means peak sales in 2031-32 of USD 1.1 billion. In order to reach these sales figures, we have assumed that the company is building its own sales organization in the US and the EU, but it is also possible that the company licenses its compound to a company with already established sales channels. In the forecasts below, we assume that KL1333, thanks to its orphan drug status, will have 7 years of market exclusivity in the US and 10 years of market exclusivity in the EU.

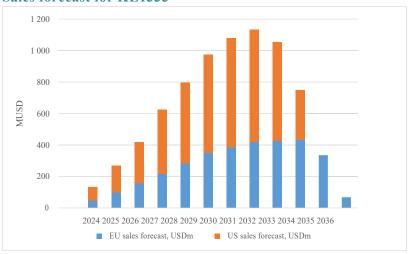
Other assets are not financed

Our valuation of the company's other assets, primarily NV354 and NeuroSTAT, is characterized by the absence of funding and limited clinical data. Recently, the company announced that NV354, a prodrug of the endogenous energy substrate succinate, has completed its preclinical studies and that the plan is to start studies on healthy volunteers next year. Given Abliva's low market value, it is not unreasonable that the stock market will start to put a value on NV354 over the course of next year.

For the time being, the company's task remains to finance a registrational study for KL1333 and in our valuation of the company, this task overshadows other value generating activities in the company.

² The Rise of Orphan Drugs, America's Health Insurer Plans, September

Sales forecast for KL1333



Source: Analysis Guide forecasts

Assumptions for the present value calculation of

MSEK (unless otherwise	e stated)	2020	2021p	2022p	2023p	2024p	2025p	2026p	2030p	2031p
G . 1 1 . CWI	1222	4.1	20		100	20	0			
Costs, development of KL	1333	-41	-20	-111	-100	-20		0	0	
Milestones to Yungjim		0	0	-20	-67	0	-265	-100	0	
Calculation for the US	market									
Number of patients within	n the target group	5	for	100,000 in	nhabitants,	, total	19,321 U	J.S. citizens		
Number of possible paties	nts to treat Sales	28% o	f the target				5,455	5,565	6,023	6,144
Price, USD/Patient Year							151,500	153,015	159,228	160,820
Percentage of seriously ill	people receiving						10%	20%	65%	70%
Sales USA, MUSD							83	170	623	692
Calculation for the Eur	opean market									
Number of patients within	n the target group	5	per	100,000 in	habitants,	total	25,000	25,000	25,000	25,000
Number of possible patier	nts to treat	28% o	f the target	group			7,059	7,059	7,059	7,059
Försäljningspris, USD/pati	entår						85,850	86,709	90,229	91,132
Percentage of seriously ill people receiving treatment						5%	10%	55%	60%	
Sales EU, MUSD							30	61	350	386
Total Sales, MUSD							113	231	974	1,078
EBIT, MSEK		-60	-81	-150	-341	-629	-498	898	6 646	7 186
Net profit, MSEK		-60	-81	-150	-341	-629	-498	898	5 316	5 749
Riskjustering	gsfaktor			1.00	0.40	0.26	0.23	0.23	0.23	0.23
Present value calculation of	f KL1333,	2 081			-103	-108	-67	105	354	333
The verdict per	2.1 SEK, 4	ofter new issue 1 at subscription price SEK 0.55					Total numb	er of shares	s, mln	980
•	1.5 SEK, 4	after new issu	e 2 at subsc	ription pric	e SEK	3.00	Total numb	er of shares	, mln	1 356
SEK/USD	8,7	7								
Discount Factor Tax	15%	Ď								
Rate	20%	Ď								
Positive phase 1	100%	Ď								
Successful phase 2/3	26%	Ď								
Successful	90%	Ď								
application	23%	,								

Overview of the target group for KL1333

The drug candidate KL1333 increases the cell's energy production by restoring the balance of nicotinamide adenine dinucleotide (NAD+/NADH) in the so-called Respiratory Complex I reaction. This balance is important for the effective degradation of food and in the formation of new mitochondria. We cannot find any other projects with similar mechanisms that are under clinical development.

Researchers estimate that primary mitochondrial diseases (PMD) occur in about 125 people per million inhabitants. PMD is primarily caused by mutations in the mitochondrial genome, inherited by the mother, and characterized by the failure of mitochondria, the cell's energy factories, to function in the desired way. The disease can also be caused by genetic changes in the DNA of the nucleus.

One of the most common mutations, m.3243A>G, is found and will cause disease in about 3.5 births per 100,000 inhabitants. Thus, in a population of 10 million, this would mean that 350 have a disease caused by this mitochondrial mutation. This mutation gives rise, among other things, to the rare MELAS-MIDD disease spectrum According to the National Board of Health and Welfare, MELAS affects about 1–2 Swedes per 100,000 inhabitants. According to the National Board of Health and Welfare, there are about 30 mutations that can give rise to the mitochondrial dysfunction MELAS, but the mutation m.3243A>g is believed to be the main source of the disease.

Abliva intends to treat the MELAS-MIDD spectrum disorders with the NAD modulator KL1333. MELAS is an acronym for Mitochondrial Encephalopathy, Lactic Acidosis, and Stroke-like episodes syndrome. MIDD stands for Maternally Inherited Diabetes and Deafness. Abliva's focus is on patients within the MELAS-MIDD spectrum disorders with pronounced fatigue, muscle weakness, and diabetes as the main symptoms.

MELAS-MIDD can be a very severe disease with continuous status degradation that mainly affects musculature and metabolism, leading to pronounced fatigue, muscle weakness and severe mitochondrial diabetes. In 5-10% of patients with MELAS-MIDD, severe central nervous system symptoms occur resulting in stroke-like episodes and loss of important brain functions. Most of the patients have peripheral symptoms such as muscle fatigue. The disease is chronic, comes and goes in relapses and the first symptoms usually occur between the ages of five and 15, almost always before the age of 40. Common symptoms in children are attacks of severe headache with vomiting. Epilepsy is a common consequence of the disease that eventually turns into unconsciousness and severe neurological conditions. The attacks can last a few hours up to a few days.

Kearns-Sayre Syndrome (KSS), another of the genetic diseases that Abliva intends to treat with KL1333, is reported to be known in to affect about 20 Swedes, i.e. an extremely rare disease (one case per

million). The first symptoms of Kearns-Sayre syndrome usually appear between the ages of five and 20 and are then progressive. Later onset, up to the age of 60 years, may occur but is very rare. When symptoms appear early in childhood, this usually leads to a more difficult course of the disease.

CPEO is a mitochondrial disease related to KSS that Abliva also wants to treat. CPEO stands for Chronic Progressive External Ophthalmoplegia (CPEO), two serious Neuromuscular eye diseases that debut before the age of 20. According to the National Board of Health and Welfare, the current incidence is not known, but is estimated at 1–2 people per 100,000 inhabitants. KSS-CPEO is considered to be caused by a deletion (loss of genome) in a different sequence in mtDNA than in MELAS-MIDD. According to Abliva, deletions that lead to diseases within the KSS-CPEO spectrum affect about 15 children in a million.

Mitochondrial diseases are distinguished by high intracellular levels of free reactive oxygen radicals (ROS) and a decrease in levels of the ATP molecule (adenosine triphosphate), the cell's actual fuel. Mitochondria's main task is to release energy from blood sugar and fat in the form of ATP consumed during the cell's various tasks. In addition, mitochondria are involved in the production of reactive free oxygen radicals, which at high levels can damage the cell. Mitochondria also affect other signalling systems inside the cell, cell death and the cell's own metabolism. As we have explained above, mitochondria have their own DNA, which comes exclusively from the child's mother and therefore differs from the DNA of chromosomes in the nucleus.

Introduction and description of KL1333

KL1333 was developed by south Korean company KT&G (Korean Tobacco & Ginseng Company). In 2017, Abliva entered into an agreement on the right to develop KL1333 against primary mitochondrial diseases with Yungjin Pharmaceutical, a subsidiary of KT&G.

The molecule KL1333 is developed as a derivative of a known family of molecules, known as β lapachons. This class of molecules was discovered during studies of the health-giving properties of the bark of the Pau d'Arco tree, which grows in South and Central America. Several attempts have been made to show that the substance has, among other things, anticancer effect, but at present the bark is sold mainly as a health supplement. In addition to cancer, different molecules in this class have been shown to have an effect on cell metabolism and energy regulation.

KL1333 is an oral treatment, which restores the balance of the two forms of intracellular nicotinamide adenine dinucleotide (NAD+/NADH), a coenzyme that is central to the energy metabolism of mitochondria and cells. In preclinical studies, the substance has been shown to increase mitochondria's energy production, reduce the accumulation of

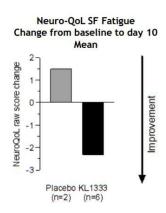
lactate, counteract the formation of free oxygen radicals and mediate long-term positive effects on energy metabolism.

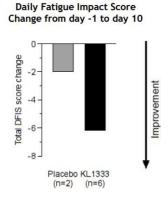
NAD⁺ and its reduced form NADH are regulators of intracellular redox-homeostasis, energy metabolism and several other signaling pathways in the cell. NAD⁺ can be produced by the de novo cell or by salvage pathways when the cell consumes large amounts of energy. NAD⁺ can also be generated by converting NADH via various enzymes or substrates, such as NQO1. It is via this later enzyme, NQO1, that the NAD modulator KL1333 acts. KL1333 raises NAD⁺ levels by oxidizing the reduced form NADH. This positively affects the NAD⁺/NADH intracellular ratio, which increases the cell's energy production and stimulates the production of new mitochondria. Due to the dysfunctional mitochondria, the NAD⁺/NADH ratio is low in patients with primary mitochondrial diseases.

This summer, Abliva presented results from a Phase 1 study with KL1333 in healthy volunteers. The study also included a final Phase 1b part in eight patients with different forms of primary mitochondrial disease (PMD). The primary purpose of the study was achieved, i.e. that KL1333 showed a favorable safety profile, allowing the company to move forward towards testing KL1333 on larger groups of patients in a Phase 2/3 study. The company's ambition is to start this study at the end of the year, which may be described as optimistic but not unthinkable. In our main scenario, the first patient in the Phase 2/3 study will be treated at the beginning of the next year, provided that the company has until then solved the funding.

In the now reported Phase 1b part, KL1333 was tested at 50 mg once a day on six PMD patients with some form of mitochondrial disease. The treatment lasted 10 days. Two more patients were given placebo to gain a better understanding of the efficacy of the active substance. The company said in its press release that no serious side effects were found. From previous studies in healthy volunteers at doses up to 250 mg, it is known that KL1333 causes intestinal problems that correlate with the dose strength. Abliva intends to counteract this by dividing the dose in two or three occasions per day.

KL1333 shows effect on fatigue in Phase 1 study





Source: Abliva

Quality of Life in Neurological Disorders Short Form (Neuro QoL SF) is a form in which the patient himself estimates different symptoms of fatigue in eight different subjects graduated 1-5. The chart on the previous page shows that the two patients who did not receive KL1333 reported, on the scale, that they experienced slightly rising symptoms of fatigue during the evaluation. In contrast, the six patients who received KL1333 over 10 days reported that their symptoms of fatigue decreased. The difference (delta) between the bars is just under 4 points. Abliva writes in its presentation that a difference between 2.5 and 5 is considered clinically meaningful in other therapeutic contexts, all of which suggests that the outcome in these six patients makes clinical sense.

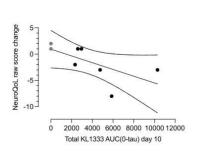
According to another patient-reported scale, the Daily Fatigue Impact Score, even the two placebo patients experienced an improvement over the short period, probably a traditional placebo effect. In contrast, the six patients treated with KL1333 experienced significantly greater efficacy and the difference between the groups was also about 4 points, a signal of clinically meaningful improvement, based on previous evaluations highlighted by Abliva.

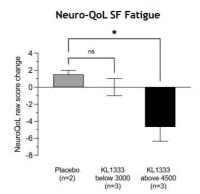
Dose response signal strengthens belief in KL1333

Another interesting finding in the study is that the three patients who best took up the dose of 50 mg of KL1333 in blood circulation were also those who showed the clearest effect on Neuro QoL scale (right chart below). Below, two of the three black dots representing the patients with the lowest uptake of KL1333 in the blood circulation showed No clear change on the scale while the other three patients, with an uptake of more than 4,500ng/mL, had clear effects on the scale. We stress that this signal is uncertain and, like the other clinical observations, is based on data from too few observations to draw conclusions.

Reduced fatigue follows exposure to KL1333

Neuro-QoL SF Fatigue





Source: Abliva

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