MAXIM GROUP

Equity Research
Initiation

Biotechnology

ABS.PA – NXT PA	December 18, 2025
Closing Price 12/17/25	**€1.08**
Rating:	Buy
12-Month Target Price:	€4.00
52-Week Range:	€0.77 - €1.51
Market Cap (M):	€71.5
Shares O/S (M):	66.2
Float:	70.0%
Avg. Daily Volume (000):	0.0
Debt (M):	€15.8
Dividend:	€0.00
Dividend Yield:	0.0%
Risk Profile:	Speculative
Fiscal Year End:	December

Total Revenues ('000)

	2024A	2025E	2026E
H1	€560	€515A	€500
H2	€512	€500	€500
CY	€1,072	€1,015	€1,000

Total Expenses ('000)

	2024A	2025E	2026E
H1	€4,049	€2,879A	€3,348
H2	€3,282	€3,167	€3,579
CY	€7,331	€6,046	€6,927

AB Science is based in France, and trades on the Euronext exchange. Financial results and our estimates and price target are expressed in Euros (€).

Naz Rahman, CFA
(212) 895-3642
nrahman@maximgrp.com

AB Science S.A.

Buy

Mastering Mast Cell Inhibition for Neurodegenerative Diseases Starting with ALS – Initiating with a Buy Rating and €4 PT

Summary

- AB Science is a Phase 3-ready company developing its lead asset, masitinib, for treating neurodegenerative disorders, with its lead indication being amyotrophic lateral sclerosis (ALS). AB Science plans to initiate a Phase 3 trial for ALS in 1Q26.
- In a Phase 2 study, masitinib showed promising survival and functional preservation data in normally progressing ALS patients. Masitinib has also demonstrated positive data in treating progressive multiple sclerosis (MS) and mild Alzheimer's disease (AD). Taken as a whole, we believe the data support masitinib's potential in treating neurodegenerative diseases.
- AB Science has an earlier-stage therapy, AB8939 for acute myeloid leukemia (AML), which has shown positive response rates in MECOM-rearranged patients.
- Exiting 1H25 (Jun), the company held €5.0M in cash and raised €2.8M on 10/17/25. AB Science has a total of €15.8M in debt due between June 2026 (€250K due) and 2029. There are also conditional advances of €13M and management is currently evaluating financing options for the ALS program. Excluding the Phase 3 ALS study costs, there should be sufficient cash runway into 2027.
- Masitinib has generated promising benefits across three neurodegenerative diseases, which we believe validates the mast cell inhibition approach. Considering the underlying efficacy data and safety profile, we view the risk-benefit profile of masitinib as positive. Given the data and opportunity, we initiate coverage with a Buy rating and €4.00 price target.

Details

Masitinib is a tyrosine kinase inhibitor (TKI) with a dual mechanism of action targeting mast cells, as well as microglia and macrophages.

- Mast cells are found in connective tissues and in almost every organ. They mediate inflammatory cascades. Pathological mast cells have been found in autopsied ALS, MS, and AD patients, which has been associated with degenerating motor axons, NMJs, and myofibers that correlate with disease severity. Masitinib has been found to reduce mast cell infiltration.
- Masitinib 4.5mg was found to have statistically significant effects in normally progressing ALS patients in a Phase 2 study. Demonstrated stat-sig slowing of functional decline and a stat-sig PFS benefit.
- Phase 3 study design has been agreed upon with regulators. ALS represents the fastest and cheapest path to market among all pipeline indications.
- Positive data in progressive MS and in mild AD further support its neuroprotective potential. We do not model AD or MS and consider them upside.

AB8939 for acute myeloid leukemia (AML) – microtubule destabilizer and ALDH1/2 inhibitor. Currently in Phase 1 studies.

- Promising data thus far – in MECOM rearranged patients, disease control rate of 100% (3/3), partial response rate of 100% (3/3), 1 patient in complete remission.
- Full Phase 1 data expected in mid-2026.
- We consider AB8939 to be upside but expect P1 results to be a meaningful near-term event for the shares.

Compelling valuation. We forecast masitinib launching in 2030 for the treatment of amyotrophic lateral sclerosis (ALS). We do not model the rest of the pipeline or indications and assume them to be upside. We apply a 60% sales risk adjustment to masitinib for ALS based on the stage of development, regulatory risk, and commercial risks. A 30% discount is applied to the free cash flow, discounted EPS, and sum-of-the-parts models, which are equally weighted to derive a 12-month price target of €4.00.

CORPORATE PROFILE

Ownership:
Institutional: <5%
Insiders: 30%*
**CEO Alain Moussy owns 20% of shares*

Balance Sheet Summary:
(as of 6/30/25 in EUR)
Cash: €5.0M (raised €2.8M in October 2025)
Debt: €28.8M (€15.8M in debt + €13M in conditional advance)

Revenue Estimates:

2025E:	€1.0M
2026E:	€1.0M
2027E:	€1.0M

Operating Expense Estimates:

2025E:	€6.0M
2026E:	€6.9M
2027E:	€7.9M

Analysts covering the stock: 3

# of Buys:	3
# of Holds:	0
# of Sells:	0

(*Excluding Maxim Group LLC*)

Investor Relations Contact:
Email: investors@ab-science.com

AB Science Corporate Headquarters
3, Avenue George V 75008,
75008 Paris – France
https://www.ab-science.com/

Company Background: AB Science is a late-stage, Phase 3-ready biopharmaceutical company developing its lead asset, masitinib, for the treatment of various neurodegenerative disorders. Masitinib's lead indication is amyotrophic lateral sclerosis (ALS). Masitinib has generated positive data in ALS, progressive multiple sclerosis (MS), and mild Alzheimer's disease (AD). AB Science is preparing to initiate a Phase 3 trial for ALS. The company is also developing AB8939 for acute myeloid leukemia (AML). In addition, AB Science commercializes Masivet, a veterinary product for mast cell tumors (MCT) in dogs; however, the product is not a strategic priority and generates minimal revenue. AB Science is based in Paris, France, and trades on Euronext (ENX).

Senior Management:

Alain Moussy, Co-Founder and Chief Executive Officer. Mr. Moussy founded AB Science and has a background in both consulting and corporate strategy, having served as a strategic consultant at Booz, Allen & Hamilton and later as Head of Corporate Development at Carrefour. He also currently serves as President of AFIRMM, an association dedicated to mastocytosis patients. Mr. Moussy studied engineering at the École nationale supérieure de techniques avancées and has an M.B.A. from The Wharton School.

Laurent Guy, Chief Financial Officer. Mr. Guy has a strong background in banking, having previously worked at Société Générale and BNP Paribas. He was also previously a strategy consultant at Accenture. Mr. Guy holds a M.B.A. from Université Paris Dauphine-PSL.

Christian Fassotte, Global Chief Medical Officer. Dr. Fassotte has >30 years of experience. He previously held executive positions at Sanofi and Roche in Medical, Regulatory Affairs, and R&D. Dr. Fassote has a Doctor of Medicine (MD) from the University of Liège.

For more information about AB Science's senior management, please see the company website.

Investment Thesis

AB Science is a late-stage, Phase 3-ready biopharmaceutical company developing its lead asset, masitinib, for the treatment of various neurodegenerative disorders. Masitinib's lead indication is amyotrophic lateral sclerosis (ALS), and AB Science has previously reported promising progression-free survival (PFS) and function-preservation data in a subset of normally progressing patients. Masitinib has also been evaluated in progressive multiple sclerosis (MS) and mild Alzheimer's disease (AD), where it demonstrated positive results. Taken as a whole, we believe the data support masitinib's potential in treating neurodegenerative diseases. AB Science also commercializes Masivet, a veterinary product for mast cell tumors (MCT) in dogs; however, the product is not a strategic priority and generates minimal revenue.

Masitinib is a tyrosine kinase inhibitor with a dual mechanism of action targeting mast cells as well as microglia and macrophages. It is a potent and selective inhibitor of c-Kit, Lyn, and Fyn kinases, which are important in the mast cell activation. Masitinib is also a potent and selective inhibitor of macrophage colony-stimulating factor receptor 1 (MCSFR-1). Mast cells are a target in neurodegenerative disease, and macrophages are a target in oncology.

Mast cells are immune cells found in connective tissues throughout the body and in almost every organ. They are known for their ability to mediate inflammatory cascades. When activated, mast cells degranulate and release various mediators, the most notable of which is histamine. Mast cells can become pathological when they undergo degranulation. They have been found to infiltrate skeletal muscle, nerves, and neuromuscular junctions (NMJs) in autopsied ALS patients. Mast cell infiltration has been associated with degenerating motor axons, NMJs, and myofibers, which correlate with disease severity. Mast cell activation is believed to contribute to muscle inflammation, fibrosis, and NMJ degradation. Consequently, targeting mast cells appears to us to be a novel way to approach neurodegenerative diseases, many of which are complex, heterogenous conditions that likely require combination therapies. In preclinical studies, masitinib has been shown to reduce immune cell infiltration, which led to preserved nerve structure and neuroprotective effects.

Masitinib 4.5mg was found to have stat-sig benefits in "normally progressing" ALS patients in a Phase 2 study, with minimal effect in "fast progressors". Masitinib was evaluated in a 48-week Phase 2 trial in a heterogeneous ALS population. The primary endpoint was the change in ALSFRS-R score at 48 weeks (ΔALSFRS-R). The masitinib 4.5mg dose demonstrated statistically significant slowing of functional decline (27%) in normally progressing patients when fast progressors were excluded. Masitinib 4.5mg also demonstrated a statistically significant progression-free survival (PFS) benefit (+4 months), although overall survival (OS) did not reach stat-sig but trended positively. When evaluating strictly normally progressing patients and excluding fast progressors and those who had already experienced complete loss of function in any subdomain, masitinib demonstrated significantly better results than placebo for both function and survival (+9 months PFS, +12 months OS).

We note that the Phase 2 trial had baseline imbalances: ~20% of patients randomized to masitinib had complete loss of function (fast progressors), compared with only 8% of patients randomized to placebo. Once a patient has complete loss of function in any domain, they become significantly more difficult to treat. With these patient imbalances removed (~86% of the primary analysis population), masitinib 4.5mg demonstrated a consistent benefit across all endpoints. Masitinib 4.5mg was also found to be well tolerated.

A Phase 3 trial has been designed to evaluate masitinib 4.5mg over 48 weeks. The primary endpoint will differ for the FDA and EMA: the FDA will use CAFS (combined assessment of function and survival), while the EMA will use the change in ALSFRS-R. Survival will be a secondary endpoint. The Phase 3 trial will also be optimized by excluding fast progressors and patients who have already experienced a loss of function. Trial initiation is dependent on raising additional capital. AB Science is targeting 1Q26 for initiation.

Data generated with masitinib in progressive multiple sclerosis (MS) and Alzheimer's disease (AD) further support its neuroprotective potential. Microglia and mast cells have been found to be implicated in the pathophysiology of MS and AD. Masitinib 4.5mg was evaluated in a Phase 2 study in patients with mild-to-moderate AD. The study found that masitinib significantly improved cognition and function at week 24. It also showed a stat-sig effect on iARDs (integrated Alzheimer's Disease Rating Scale, combining ADAS-Cog and the ADCS-iADL). Masitinib was additionally found to slow the time to severe disease progression. Regarding MS, there are currently limited options for progressive MS, with no FDA-approved therapies for non-active secondary progressive MS and one for primary progressive MS. The majority of approved therapies are for relapsing MS. This leaves a significant market opportunity for masitinib. In a Phase 2 study of primary progressive MS (PPMS) and relapse-free secondary progressive MS (rfSPMS), masitinib generated promising data. Although the primary endpoint was not met, masitinib demonstrated directionally positive effects on MS-related impairment and showed a reduction in time to disability. AB Science has agreements with the FDA and EMA on Phase 3 trial designs for both MS and AD. Since ALS is likely the fastest and cheapest path to market, AB Science is prioritizing development in ALS; we view MS and AD as upside. AB Science is also evaluating masitinib for the treatment of mast cell diseases (MCD) and sickle-cell disease (SCD), which we also view as upside and do not include in our valuation.

AB8939 for acute myeloid leukemia (AML). AB Science's other key pipeline asset is AB8939, which it is developing to treat MECOM-rearranged AML patients. AB8939 is a novel, small-molecule, synthetic microtubule destabilizer and targeted stem cell ALDH1/2 inhibitor. AB8939 can overcome resistance currently seen with approved microtubule-targeting chemotherapies. AB8939 is currently being evaluated in a Phase 1 trial and has shown positive responses MECOM-rearranged patients. AB Science has completed Stages 1 and 2 of the Phase 1 trial and is enrolling patients in Stage 3, the combination arm of AB8939 + venetoclax. The data, while in a small number of patients has been highly promising thus far. In MECOM-rearranged patients, AB8939 has shown a disease control rate of 100% (3/3) and the partial response rate is 100% (3/3), including one patient in complete remission. These results were after the first cycle of treatment in patients receiving third- or fourth-line treatment, two of whom had previously progressed on venetoclax in combination with other chemotherapies. We currently consider masitinib

the lead asset and while we do not currently model in AB8939, we consider it upside. That being said, we believe the asset has significant promise and could be a share driver going forward. With the full Phase 1 data expected in mid-2026, AB8939 may represent a meaningful near-term catalyst for shares and we would pay attention to associated news.

Bottom line — Initiating with a Buy rating and 12-month price target of €4. Masitinib has generated promising benefits across three neurodegenerative diseases, which we believe validates the mast cell inhibition approach and the potential biological effect of masitinib. While the most promising data in ALS were in a subset of patients, we note that ALS is a very heterogeneous disease, and most developments and approvals have been seen in subsets of patients, with many receiving broad labels. Considering the underlying efficacy and broad safety data, we find the risk-benefit profile of masitinib to be positive. Consequently, we expect a Phase 3 success in ALS. We remain conservative in our estimates and risk-adjust our masitinib sales forecast for ALS by 60%. ALS may be the fastest path to market, but we remain bullish on the overall pipeline.

Finances. AB Science finished 1H25 with €5.0M. The company completed a €2.8M private placement on 10/17/25. AB Science also received a partial payment of €2.9M for the 2023 Research Tax Credit by the tax administration in 2025. AB Science has state-guaranteed loans with a principal of €3.5M due in March 2029, carrying an annual interest rate between 0.25% - 1.75%. AB Science also has a €250K loan due in June 2026 with an interest rate of 2.25%. The company has an additional loan with the European Investment Bank (EIB - private) with a principal of €12M due in December 2028. Of this amount, €6M carries an annual interest rate of 9% and €6M carries a rate of 7%. AB Science is also renegotiating this debt to push it to 2029.

The company also has a conditional advances of ~€13M (carrying value) (~€10 gross amount) on the balance sheet, received from Bpifrance ISI (private). The conditional advances are only paid if the projects are successful otherwise no payment is due.

- €5,764 received for masitinib's development in neurodegenerative diseases. If successful in ALS Phase 3 and commercialized, required to pay back €6,600 over 4 years and then pay 1% of sales over 3 years up to a cumulative of €7,000 (after initial €6,600 payment).
- €4,432 received for AB8939 development in oncology. If successful in Phase 3 and commercialized, required to pay 1% of sales up to a cumulative of €16,000 (capped at €4,000 per year).

AB Science completed multiple capital raises in 2025, raising €9.075M:

- 10/17/25: €2.8M private placement (2,477,877 ordinary shares + warrants to purchase up to 2,477,877 ordinary shares for €1.13/share). Warrants expire 10/17/2030.
- 8/4/25: €2.55M private placement (2,276,787 ordinary shares + warrants to purchase up to 2,276,787 ordinary shares for €1.12/share). Warrants expire 8/7/2030.
- 7/8/25: €1.925M private placement (1,645,302 ordinary shares + warrants to purchase up to 1,645,302 ordinary shares for €1.17/share). Warrants expire 7/8/2030.
- 5/23/25: €1.8M private placement (1,645,302 ordinary shares + warrants to purchase up to 1,645,302 ordinary shares for €1.17/share). Warrants expire 7/8/2030.

AB Science is currently evaluating financing options for the ALS program. Excluding capital required for the ALS study, AB Science's cash runway extends into 2027. The AB8939 study is fully funded. To initiate the ALS Phase 3 program, AB Science requires €22M. In addition to the study cost, development of the liquid formulation is expected to cost €5M and will occur after Phase 3. The Phase 3 trial will enroll 80% of patients in Europe and 20% in the U.S., significantly reducing costs, as European patients cost <€15K per patient vs. >€40K in the U.S. This is effectively the cheapest and quickest path to market for AB Science across all of its development programs. We believe that, given the data generated thus far, AB Science may successfully complete sufficient financing in the future.

Exhibit 1. AB Science Pipeline

Platform	Drug	Therapeutic area	Indication	Development Stage
Tyrosine Kinase Inhibitor	Masitinib (Oral)	Neuro-degenerative Diseases (NDD)	Amyotrophic Lateral Sclerosis	Phase 3
			Progressive Forms of Multiple Sclerosis	Phase 3
			Alzheimer's Disease	Phase 3
		Mast Cell Diseases	Indolent Systemic Mastocytosis	Phase 3
			Mast Cell Activation Syndrome	Phase 2
		Blood diseases	Sickle Cell Disease[1]	Phase 2
Oncology Platform	AB8939 (IV)	Hematology	Acute Myeloid Leukemia (AML)	Phase 1
	AB12319 (oral)	Oncology	Sarcoma, Solid Tumors	Preclinical
Drug Discovery Platform		Target 1: undisclosed for neurodegenerative diseases Target 2: undisclosed for neurodegenerative diseases		Drug Discovery

Source: Company Reports

Lead Asset Masitinib Being Developed to Treat Neurodegenerative Diseases

Masitinib's mechanism of action (MOA) targets mast cells and macrophages. Masitinib is a tyrosine kinase inhibitor with a dual mechanism of action, targeting mast cells and microglia/macrophages. Masitinib is a potent and selective inhibitor of c-Kit, Lyn, and Fyn kinases, which are important in the activation of mast cells. Masitinib is also a potent and selective inhibitor of macrophage colony-stimulating factor receptor 1 (MCSFR-1). Macrophages are a key target in oncology, while mast cells are a target in amyotrophic lateral sclerosis and Alzheimer's disease.

Mast cells are mononuclear immune cells that are found in connective tissues throughout the body in almost every organ. They contain secretory granules that often obscure the nucleus. Mast cell membranes contain IgE receptors that bind the Fc region of circulating IgE and two crosslinks induce cell degranulation (process by which cells release stored granules containing chemical substances).[1]

The two major types of mast cells are:

- MC(T) cells contain tryptase and are found mainly near mucosal tissues (e.g., respiratory, gastrointestinal). They are primarily involved in immune defense.
- MC(TC) cells contain tryptase, chymase, and carboxypeptidase. They are found in connective tissue (skin, submucosa) and are involved in tissue repair.

Mast cells are primarily known for their ability to mediate inflammatory cascades. When activated (i.e., an IgE-bound membrane binds to a foreign substance and two Fc receptors crosslink), mast cells degranulate and release mediators, most notably histamine, followed by tryptase, chymase, TNF-α, prostaglandins, and leukotrienes. Histamine induces white blood cell chemotaxis, airway smooth muscle constriction, and increased vascular permeability.

The inflammatory effects of mast cells are leveraged in immune responses. MC(T) cells can enhance mucus secretion and gut peristalsis to eliminate pathogens. Mast cells are also involved in tissue repair and angiogenesis. MC(TC) cells release factors that promote coagulation, fibroblast growth, endothelial cell development, and new blood vessel formation (via VEGF and FGF2). They also aid wound healing and nerve regeneration.

Mast cells can become pathological when they undergo degranulation.

Overactivation or proliferation of mast cells can cause several disorders. Mast cell IgE-mediated degranulation can lead to Type I hypersensitivity reactions, including allergic responses such as rhinitis, asthma, urticaria, and anaphylaxis.

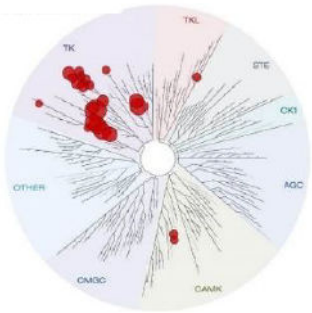
Clonal proliferation of mast cells due to KIT mutations can cause mastocytosis. Individuals may also experience cutaneous mastocytosis (CM), which affects the skin, including forms such as urticaria pigmentosa. They may also experience systemic mastocytosis (SM), which affects multiple organs and may be indolent or well-differentiated, potentially leading to organ dysfunction.

Masitinib has a neuroprotective effect on the central nervous system by targeting mast cells and microglia. Masitinib reduces microgliosis and aberrant glial cells via CSF-1R inhibition and prevents motor neuron degeneration. Masitinib has been found to significantly improve survival in post-paralytic $SOD1^{G93A}$ rats. It also exerts a neuroprotective effect on the peripheral nervous system by targeting mast cells. Mast cell infiltration and degranulation have been found to contribute to neuromuscular pathology in post-paralytic $SOD1^{G93A}$ rats. Masitinib's ability to inhibit mast cells significantly reduces the rate of neuromuscular junction (NMJ) and motor deficits. Significant infiltration of mast cells and neutrophils is seen in the muscle of ALS patients. Mast cells and neutrophils have also been found in the peripheral motor pathway in post-paralytic SOD1 rats. Masitinib has been found to prevent mast cell and neutrophil infiltration, axonal pathology, secondary demyelination, and the loss of type 2B myofibers in $SOD1^{G93A}$ rats following paralysis.

[1] Fong M, Crane JS. Histology, Mast Cells. [Updated 2023 May 1]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK499904/

Exhibit 2. Masitinib Kinase Inhibition Profile

Kinase inhibition profile of masitinib			
Cellular Target	Molecular Target	IC_{50} [nM]	Kd [µM]
Mast cells	KIT wild-type (WT)	20	0.008
	FYN	240	0.14
	LYN	225	0.061
Microglia/ Macrophages	MCSFR-1	90	0.0076



Source: Company Reports

Amyotrophic Lateral Sclerosis (ALS) – Phase 3 Ready to Initiate

Amyotrophic lateral sclerosis (ALS) is a progressive, adult-onset neurodegenerative disease that leads to muscle weakness and paralysis. ALS patients experience progressive degeneration of motor neurons in the spinal cord and brain, which are responsible for controlling voluntary muscle movement. Patients with ALS develop degeneration of the "lateral" area of the spinal cord, leading to scarring or hardening (sclerosis). The loss of motor neurons results in muscle weakness, loss of muscle mass, and an inability to control movement. The motor neurons affected by ALS impact voluntary movements and muscle control. As patients lose muscle control, they gradually lose their ability to speak, eat, move, and breathe. Patients may die as early as three to five years after their initial diagnosis. Approximately 20% of ALS patients live five years, 10% survive 10 years, and 5% live 20 years or longer.[2]

Epidemiology. There are two types of ALS, sporadic and familial. Approximately 90%–95% of all cases are sporadic ALS (SALS), with the remaining 5%–10% familial (FALS). FALS is inherited, and in affected patients, there is a 50% chance their offspring will inherit the gene mutation. It is estimated that the incidence of ALS is two per 100,000. In people over 60 years of age, there are three to four new cases per 100,000. ALS onset usually occurs between 40 and 60 years of age, but in rare instances it occurs in younger patients, including patients in their 20s and 30s. It is estimated that the overall prevalence is five to seven per 100,000, corresponding to an overall prevalence rate ~16,500 – 23,000 cases in the U.S. ALS is 1.56x more likely in men than women, but among patients over age 70, the incidence is similar. ALS is about twice as likely to occur in White individuals, compared to Black individuals. In addition, it is more common among non-Hispanics. The only known and consistent risk factors for sporadic ALS are age and gender. As mentioned, FALS is hereditary. It is believed there may be additional environmental contributors to ALS, but these have not been proven.[3]

Limited treatment paradigm. There are essentially only two approved compounds specifically for ALS and four therapies used for ALS: Radicava (edaravone), Rilutek (riluzole, generic), Tiglutik (thickened riluzole), and Nuedexta. None of the therapies substantially halt or reverse the disease. ALS treatments primarily aim to reduce the severity of symptoms but do not address the underlying disease. FDA-approved therapies have limited disease-modifying effects, with riluzole extending survival two to three months and edaravone slowing the decline of the ALSFRS-R score in a small subset of early stage patients. Tiglutik is a thickened liquid form of riluzole that helps patients with swallowing difficulties due to muscle weakness in the face and throat. Nuedexta is specifically indicated for pseudobulbar affect (PBA), which is

[2] https://www.als.org/understanding-als/what-is-als
[3] https://www.als.org/navigating-als/resources/fyi-epidemiology-als-and-suspected-clusters

characterized by frequent, involuntary, and often sudden episodes of exaggerated crying or laughing that ALS patients may experience. On 9/29/2022, Amylyx's (AMLX - NR) prior ALS therapy, Relyvrio, was granted conditional approval.[4] On 3/8/24, Amylyx announced that Relyvrio failed its confirmatory Phase 3 PHOENIX trial,[5] which ultimately led to its withdrawal from the market.[6] On 4/25/23, the FDA also approved Biogen's (BIIB - NR) Qalsody (tofersen) specifically for SOD1 ALS patients, a population of <500 people in the U.S.[7] To treat loss of respiratory function, patients may also receive non-invasive ventilation. It is estimated that ~$250K is spent annually out-of-pocket to care for a person with ALS. While current treatment options remain ineffective, the ALS market is estimated to increase to >$1B globally by 2029.

Masitinib Has Generated Positive Function Retention and Survival Data in ALS

What is the importance of mast cells in ALS? In ALS, degeneration of motor axons is associated with issues with axonal transport, mitochondrial function, and neuromuscular junction (NMJ) stability. While inflammation in the central nervous system involving glial and immune cells is well studied, the role of peripheral immune cells in ALS is less understood. It is known that monocytes and macrophages infiltrate nerves and muscles in ALS, but whether they are deleterious or protective is unclear. **Studies in SOD^{1G93A} rats have found that mast cells expressing the c-Kit receptor accumulate and interact directly with degenerating motor nerve endings in ALS models. Activation of mast cells has been found to correlate with disease progression. Masitinib, a c-Kit inhibitor, has been found to slow NMJ degeneration and motor decline in an ALS rat model, suggesting that mast cells contribute to disease worsening.** The exact mechanism by which mast cells interact with other immune cells to promote motor neuron degeneration remains unknown. Mast cells promote inflammation by releasing mediators that increase vascular permeability and recruit neutrophils, which may damage neuromuscular tissue. Neutrophils are a type of myeloid immune cell known for their cytotoxic roles in neurodegenerative diseases and muscle damage. They release enzymes and form neutrophil extracellular traps (NETs) that can harm surrounding tissues. However, their role at ALS motor nerve terminals has not been fully explored. Taken together, these findings suggest that masitinib's mechanism of action (MOA) may generate a positive benefit in treating ALS patients.

Mast cell infiltration found in autopsied ALS patients. In autopsied quadriceps muscles from ALS patients, a significant increase in mast cell number and activity was found vs. healthy controls. While control muscles contained small, inactive mast cells limited to perivascular regions, ALS patient muscles showed large, actively degranulating mast cells releasing granules into nearby muscle fibers. Mast cells have the ability to recruit and activate other immune cells through degranulation and release of inflammatory mediators, including macrophages and T cells infiltrating ALS-affected muscle. These mast cells expressed c-Kit and chymase, confirming their identity and involvement in chronic muscle inflammation. The findings were consistent with ALS rat models, suggesting mast cells contribute to disease-related muscle pathology.[8]

Exhibit 3. Baseline Characteristics of ALS and Control Patients in Study

Table 1. Clinical characteristics of ALS and control subjects included in the study

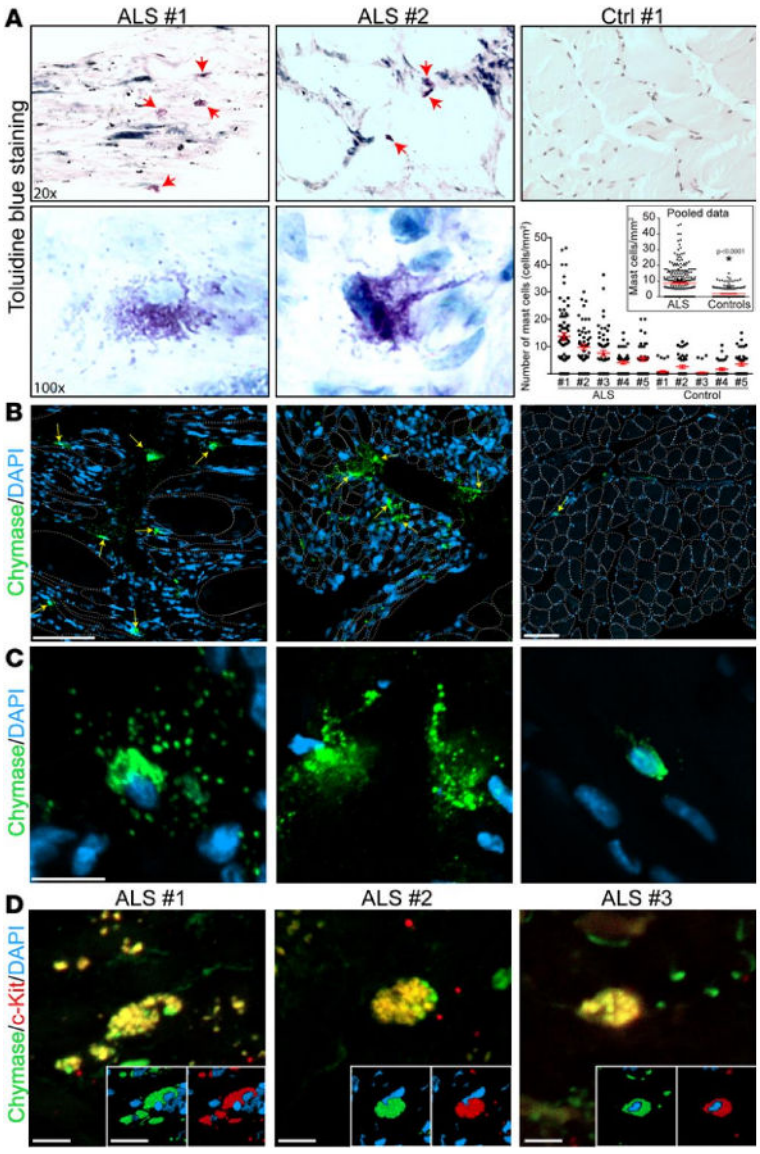
Subject	Muscle	MRC grade[A]	Age (years)	Sex	Disease onset	Survival (Months)	Postmortem tissue processing (hours)	EMG[B] denervation
ALS 1	vastus	3	63	M	Leg	44	7.0	Active and chronic
ALS 2	vastus	1	69	F	Leg	50	3.0	Active
ALS 3	vastus	3	64	M	Leg	35	6.5	Chronic
ALS 4	vastus	3[C]	59	F	Arm	26	13.0	Active
ALS 5	vastus	3	75	M	Bulbar	55	4.3	Active and chronic
Control 1	vastus	-	61	M	-	-	10.0	-
Control 2	vastus	-	68	M	-	-	19	-
Control 3	vastus	-	59	M	-	-	9.5	-
Control 4	vastus	-	90	M	-	-	4.5	-
Control 5	vastus	-	62	M	-	-	3.0	-

[A]Medical Research Council (MRC) muscle power grade at clinic visit prior to death. [B]EMG performed at time of ALS diagnosis. [C]Muscle showed predominant spasticity.

Source: Trias et al.

[4] https://www.amylyx.com/news/amylyx-pharmaceuticals-announces-fda-approval-of-relyvriotm-for-the-treatment-of-als
[5] https://www.amylyx.com/news/amylyx-pharmaceuticals-announces-topline-results-from-global-phase-3-phoenix-trial-of-amx0035-in-als
[6] https://www.amylyx.com/news/amylyx-pharmaceuticals-announces-formal-intention-to-remove-relyvrior/albriozatm-from-the-market-provides-updates-on-access-to-therapy-pipeline-corporate-restructuring-and-strategy
[7] https://www.fda.gov/drugs/news-events-human-drugs/fda-approves-treatment-amyotrophic-lateral-sclerosis-associated-mutation-sod1-gene
[8] Trias et al., Mast cells and neutrophils mediate peripheral motor pathway degeneration in ALS., JCI Insight. 2018;3(19):e123249. https://doi.org/10.1172/jci.insight.123249.

Exhibit 4. Mast cells infiltrate and degranulate in the skeletal muscle of ALS patients. (A) Representative images of toluidine blue staining show the infiltration of mast cells into the quadriceps muscles of ALS patients. The upper panels are low-magnification microphotographs demonstrating significant infiltration of mast cells (arrows) into degenerating muscle of ALS patients vs. controls, in which few mast cells are observed and are mostly associated with blood vessels. Note the pronounced infiltration and degranulation state of mast cells under ALS conditions (lower panels). The graph shows the analysis of mast cells in the quadriceps muscles of five ALS patients and five controls. The inset shows the quantitative analysis of mast cell infiltration for pooled ALS patients, compared with pooled controls. (B) Representative confocal tile reconstruction show chymase-positive mast cells (green, arrows) infiltrating quadriceps muscles from ALS and control donors. A marked infiltration of mast cells is seen in ALS patients compared with controls, in which fewer and smaller chymase-positive mast cells are observed, mostly associated with blood vessels. Dotted lines delimit myofibers. (C) Representative confocal microphotographs show an important number of chymase-positive cells with an irregular shape consistent with a degranulating state, which is not observed in any of the controls analyzed. (D) Confocal microphotographs show the co-expression of the tyrosine kinase receptor c-Kit (red) and chymase (green) in a subpopulation of cells resembling mast cell progenitors that infiltrate the muscle. Insets show 3D confocal reconstructions of separated channels. Scale bars: 5µm (colocalization) and 10µm (insets).



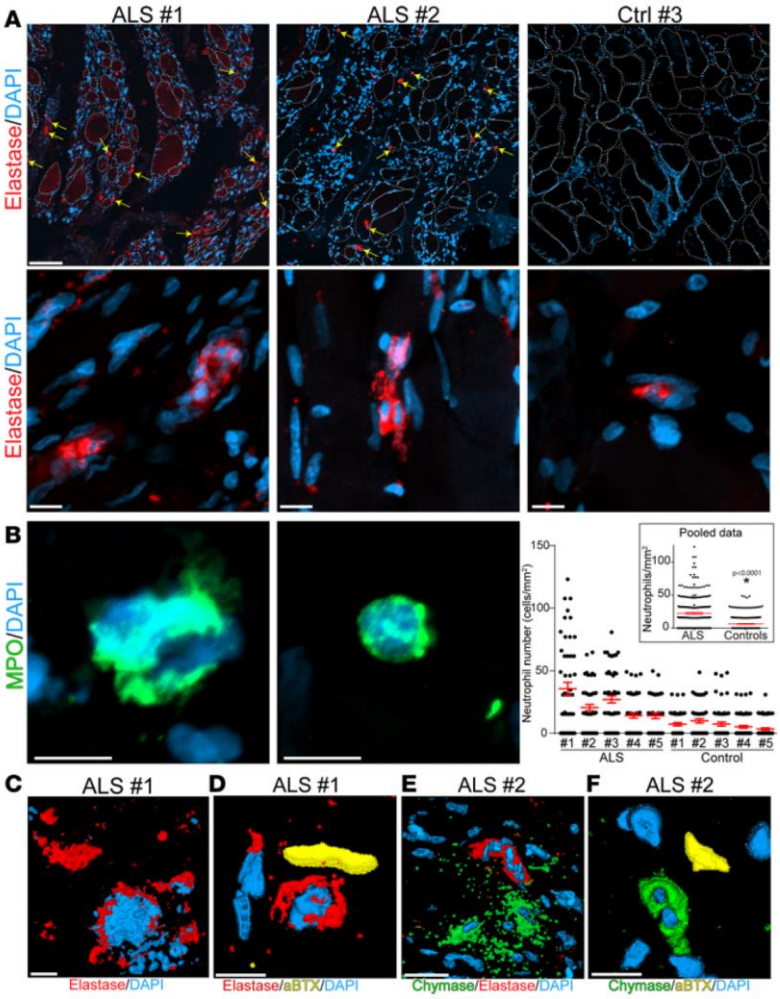
Source: Trias et al.

Neutrophil infiltration seen in ALS SOD1^{G93A} rat models. In SOD1^{G93A} rat models, neutrophil infiltration in skeletal muscle increased significantly during paralysis progression. Initially rare, neutrophils became abundant after paralysis onset, forming clusters and neutrophil

extracellular traps (NETs). They were often found in direct contact with neuromuscular junctions (NMJs) and motor nerve terminals, sometimes engulfing nerve fibers, suggesting a role in axonal degradation. A subset of neutrophils expressing myeloperoxidase (MPO) was also present in advanced stages, reinforcing their involvement in muscle inflammation and nerve damage.

Mast cells and neutrophils were also seen in peripheral nerves. In a SOD1^{G93A} rat model, mast cells and neutrophils were found to infiltrate the sciatic nerve and ventral roots of symptomatic animals. In healthy rats, mast cells were scarce and restricted to blood vessel regions, but in diseased rats, they accumulated along degenerating axons, showing explosive degranulation and a sharp increase in density as paralysis progressed. Immunostaining confirmed high levels of tryptase-positive granules invading nerve tissue. This infiltration pattern was specific to peripheral nerves, as no significant mast cell presence was found in the spinal cord or dorsal root ganglia.

Exhibit 5. Neutrophils infiltrate degenerating skeletal muscle of ALS patients and interact with mast cells and NMJs. (A) Representative confocal tile reconstruction showing the infiltration of elastase-positive (red) neutrophils into two postmortem quadriceps samples from ALS patients and one control donor (upper panels). High-magnification images in the second-row panels show neutrophils from ALS patients forming aggregates resembling neutrophil extracellular traps. Neutrophils from control donors were associated with blood vessels and mostly present as small, single cells that do not aggregate. Dotted lines delimit myofibers. (B) Images show infiltration of myeloperoxidase (MPO, green) into degenerating quadriceps ALS muscles. Few or no MPO-positive cells are observed in control donors. The graph to the right shows the analysis of total elastase-positive neutrophils present in the quadriceps muscles of each ALS patient and control. The inset shows the analysis of elastase-positive neutrophil infiltration for pooled ALS patients compared with pooled controls. (C) Neutrophil extracellular traps observed in the quadriceps of an ALS case. (D) Representative 3D confocal reconstruction shows the interaction of elastase-positive neutrophils (red) with NMJ motor endplates (yellow). (E) Shows the interaction of elastase-positive neutrophils (red) with degranulating chymase-positive mast cells (green). No degranulating mast cells or neutrophil–mast cell interactions are observed in control donors. (F) Shows the proximity of chymase-positive mast cells (green) to motor endplates (yellow) in the quadriceps muscles of an ALS patient.



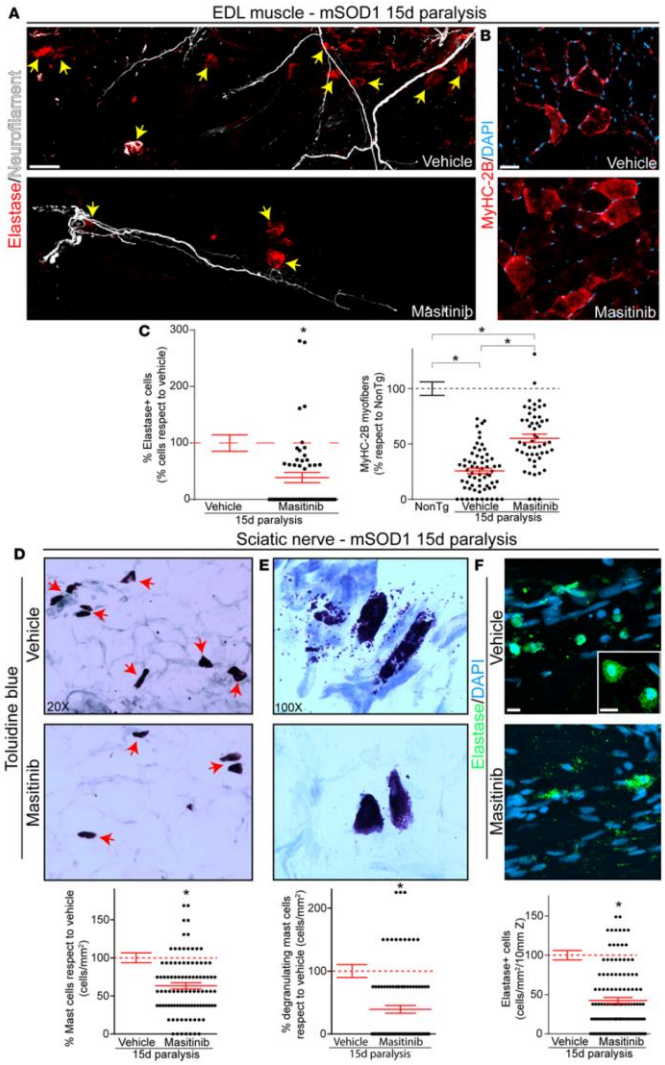
Source: Trias et al.

Masitinib Found to Have Neuroprotective Effects in ALS Rat Models

Masitinib was found to be neuroprotective and reduce immune cell infiltration in an ALS rat model. Masitinib was evaluated in SOD1^{G93A} ALS rats for its impact on inflammation and nerve degeneration. When administered orally after paralysis onset, masitinib significantly reduced mast cell and neutrophil infiltration in skeletal muscle and the sciatic nerve. Mast cell density decreased by about 40%, degranulation by 60%, and neutrophil infiltration by 50%. It also helped preserve type 2B fast-twitch muscle fibers, which are typically lost during ALS progression.

Masitinib's reduction of immune cell infiltration was also correlated with preserved nerve structure. Masitinib decreased axonal loss in the ventral roots and sciatic nerve by 30% and 20%, respectively, vs. untreated rats. It also reduced markers of demyelination and Schwann cell activation (GFAP and S100B) by roughly 60%–70%, indicating less nerve damage. Overall, masitinib protected motor pathways in ALS by downregulating mast cell- and neutrophil–mediated inflammation and slowing axonal degeneration.

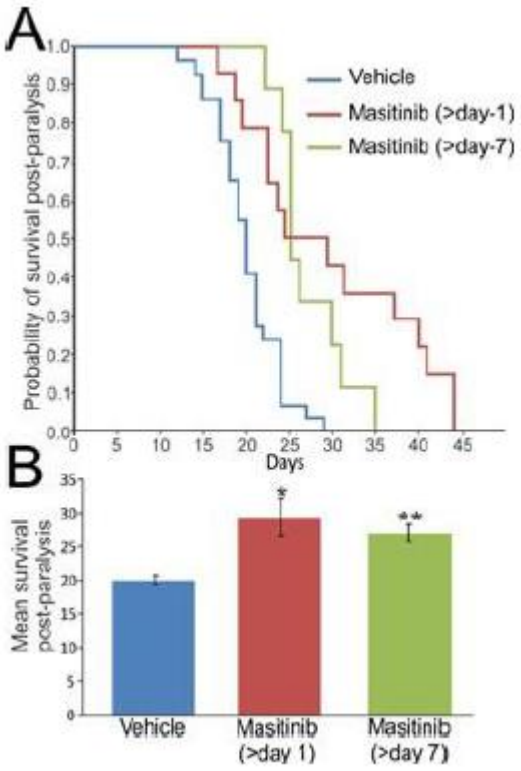
Exhibit 6. Masitinib treatment reduces mast cell and neutrophil infiltration into the sciatic nerve and prevents muscle atrophy in mSOD1 rats. Masitinib (30mg/kg/day) or vehicle was orally administered for 15 days after paralysis onset, and rat EDL muscle and sciatic nerves were processed for analysis. (A) Images show the infiltration of elastase-positive neutrophils (red, yellow arrows) in the surroundings of motor nerve terminals. (B) Transverse cryosections of EDL rat muscle showing staining with an anti–myosin heavy-chain 2B isoform (MyHC-2B, red). Masitinib treatment reduced the loss of 2B myofibers. n = 3 animals/condition (20 muscle slices per animal analyzed). (C) Left graph shows the analysis of elastase-positive neutrophils infiltrating masitinib-treated rats vs. vehicle-treated rats. n = 4 animals/condition. The right graph shows the quantification of 2B myofibers in EDL muscle across conditions. The number of MyHC-2B–positive fibers from nontransgenic (NonTg) animals was considered 100%. n = 3 animals/condition. (D and E) Images of sciatic nerve show the number and degranulation of mast cells. The graphs below show the analysis of the total number of mast cells (D) and degranulating mast cells (E). n = 4 animals/condition. (F) Images show elastase-positive neutrophils infiltrating symptomatic vehicle- and masitinib-treated sciatic nerves. The graph below shows the analysis of the total number of elastase-positive neutrophils infiltrating the sciatic nerve compared with vehicle-treated rats. n = 4 animals/condition.



Source: Trias et al.

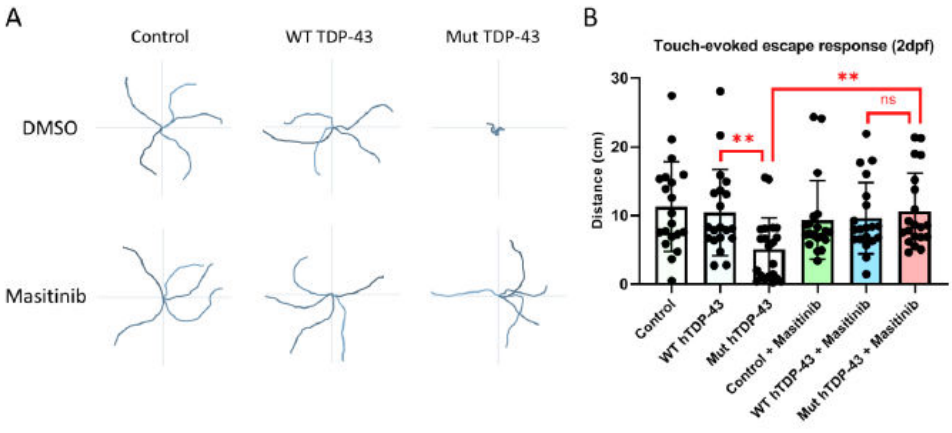
Masitinib was also found to be effective in two commonly used ALS models: SOD and zebrafish with TDP-43 overexpression

Exhibit 7. Masitinib prolonged survival by 40% following paralysis onset (Treatment initiated 7 days after paralysis onset)



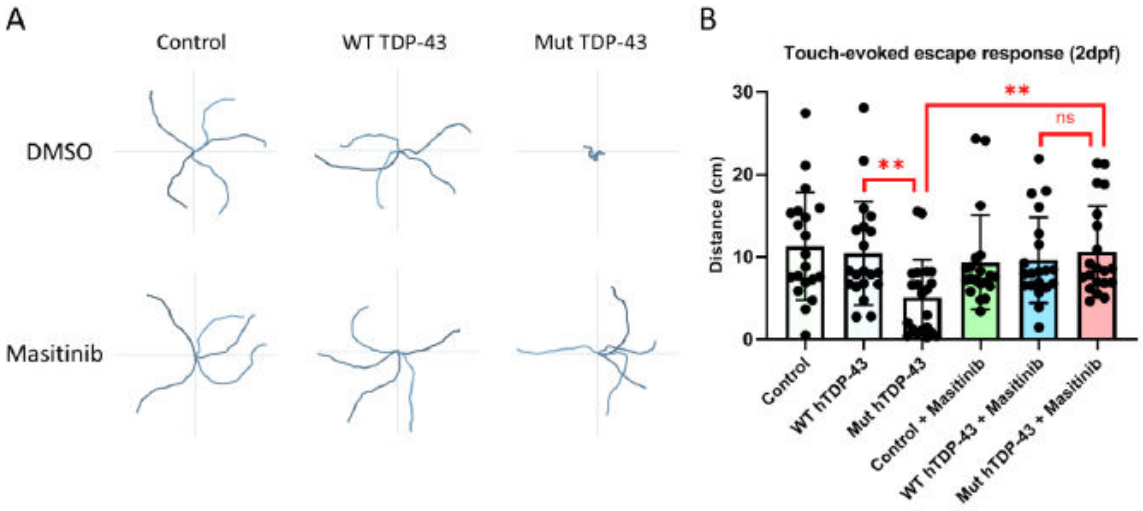
Source: Company reports

Exhibit 8. Masitinib restored motor function in a zebrafish model with mutant TDP-43 overexpression



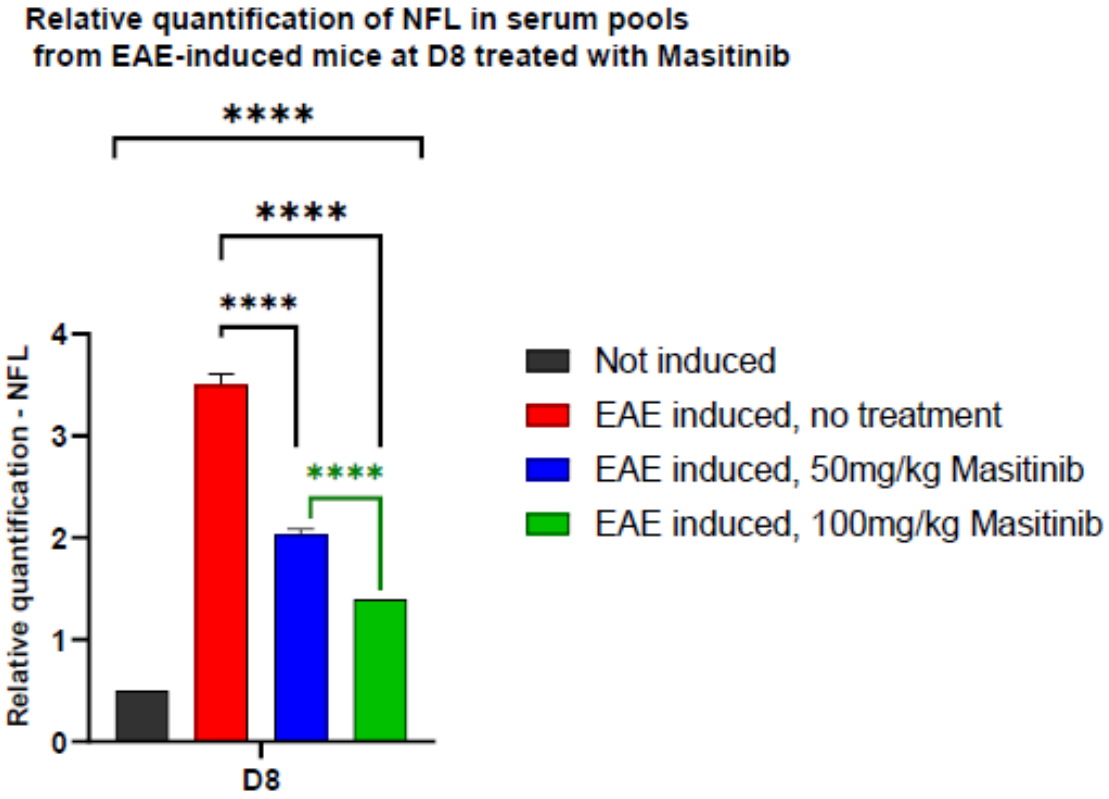
Source: Company reports

Exhibit 9. Masitinib restored motor function in a zebrafish model with mutant TDP-43 overexpression



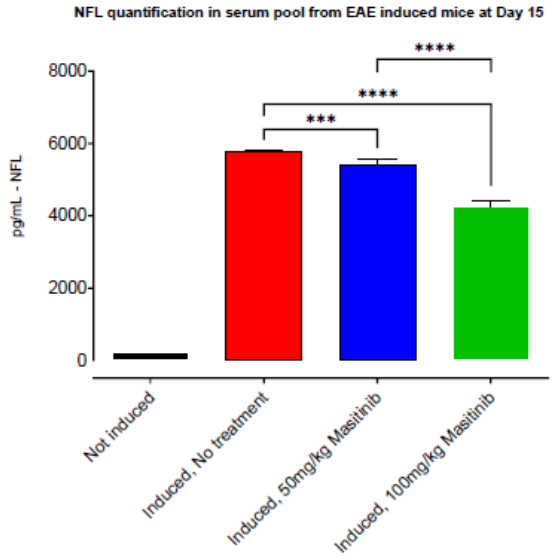
Source: Company reports

Exhibit 10. Masitinib Found to Reduce NfL Levels in a Neurodegenerative Disease Model (Experimental Autoimmune Encephalomyelitis Model (EAE)) – Day 8



Source: Company reports

Exhibit 11. Masitinib Found to Reduce NfL Levels in a Neurodegenerative Disease Model (Experimental Autoimmune Encephalomyelitis Model (EAE)) – Day 15



Source: Company reports

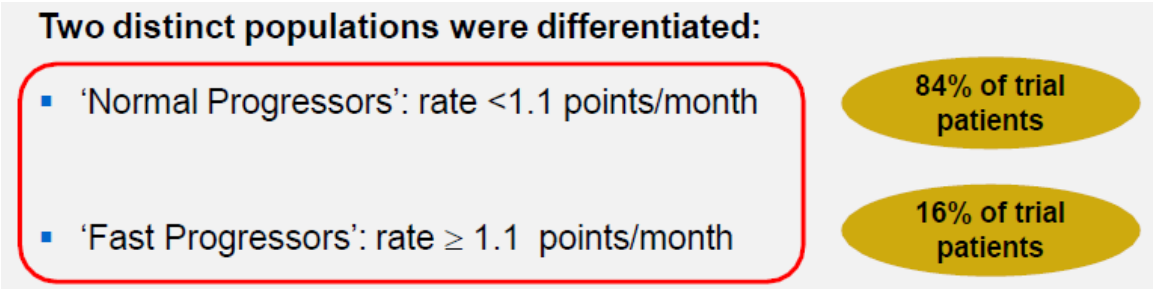
Phase 2b/3 – Masitinib was evaluated in a Phase 2b/3 study showing positive benefits in "normally progressing" ALS patients on both function and survival.

Phase 2b/3 study design. Masitinib was evaluated in a Phase 2b/3, double-blind, randomized, placebo-controlled trial, 48-week trial that enrolled two parallel groups. Riluzole was used as the standard of care in the trial. The trial enrolled 394 patients who were randomized to one of three cohorts: 1) masitinib 4.5mg/kg/day + riluzole (130 patients), 2) masitinib 3.0mg/kg/day + riluzole (131 patients), or 3) placebo + riluzole (133 patients). The primary endpoint was the change in the ALSFRS-R score at 48 weeks (ΔALSFRS-R).

Inclusion criteria. The study enrolled patients with probable or definite ALS, either sporadic or familial, without restriction on baseline ALSFRS-R score. Patients were required to be on a stable dose of riluzole for at least 30 days prior to screening. They were required to have a disease duration ≤ 36 months and a forced vital capacity (FVC) ≥ 60%.

For the statistical analysis, patients were categorized as "normal progressors" (<1.1 points/month; 84% of patients) or "fast progressors" (≥1.1 points/month; 16% of patients). The rate of ALSFRS-R progression was measured from the first symptom to randomization (points/month). The efficacy analysis was conducted in a stepwise manner.

Exhibit 12. Normal and Fast Progressors Evaluated



Source: Company reports

Exhibit 13. Efficacy analysis conducted in stepwise manner

	STEP	POPULATION
Primary Analysis	1	Normal Progressor; Masitinib 4.5 mg
Move to next step if previous step is positive	2	Normal Progressor; Masitinib 3.0 mg
	3	Normal + Fast Progressors; Masitinib 4.5 mg
	4	Normal + Fast Progressors; Masitinib 3.0 mg

Source: Company reports

Exhibit 14. Masitinib demonstrated statistically significant slowing of functional decline at week 48 at 4.5mg using multiple analysis methods in "normal progressors" and excluding "fast progressors"

Change from Baseline to WK48 in ALSFRS-R (Primary Endpoint)	Rule for Imputation of Missing Data	Primary Analysis Population (Normal Progressors; M4.5 vs P)		Reduction in Slope
LOCF Method (Primary Analysis)	Last observation collected is carried forward to week 48 time point	Diff. of mean	3.39	27%
		p-value	0.0157	

mITT population. N= 113 (placebo) and 105 (Masitinib 4.5 mg/kg/day)

Change from baseline to week 48 in ALSFRS-R (primary endpoint)	Rule for Imputation of Missing Data	Primary Analysis Population (Normal Progressors ; M4.5 vs P)		Reduction in slope
Copy Increment in Reference - CIR	• CIR assumes progressive return to placebo at the time of discontinuation • CIR is adapted to disease modifying treatment	**Diff. of mean**	**2.67**	**20%**
		p-value	**0.0462**	
Jump to Reference Analysis (JTR) for discontinuations due to Lack of Efficacy/ Toxicity/Travel	• JTR gives 2 penalties: i) a brutal alignment to placebo at the time of discontinuation, ii) then to follow the slope of placebo • JTR is adapted to symptomatic treatment	Diff. of mean	2.82	21%
		p-value	0.0372	

mITT population. N= 113 (placebo) and 105 (Masitinib 4.5 mg/kg/day)

Source: Company reports

Exhibit 15. Masitinib 4.5mg demonstrated statistically significant benefits across multiple different secondary endpoints, including progression-free survival (PFS) in "normal progressors". We note that overall survival (OS) did not meet stat-sig but trended positively.

ALSAQ-40 (mITT - Normal Progressors ; M4.5 vs P)

Analysis (M4.5 vs placebo)	Difference, [95% CI]	p-value
mLOCF Method	-7.76 [-13.45; -2.06]	0.0078

- There is a benefit in quality of life

FVC (mITT - Normal Progressors ; M4.5 vs P)

Analysis (M4.5 vs placebo)	Difference, [95% CI]	p-value
mLOCF Method	7.54 [0.76;14.32]	0.0296

- There is a benefit in respiratory function

Progression Free Survival analysis (mITT - Normal Progressors ; M4.5 vs P)

Treatment group	N	Median months [95% CI]	Wilcoxon p-value
Placebo	113	16 [11; 19]	0.0159
Masitinib 4.5	105	20 [14; 30]	

- Progression Free Survival (PFS) was prespecified and there was a benefit (+4 months) with a p=0.0159
- Event being the earlier event between 9 points decline in ALSFRS and death

Event Free Survival analysis (mITT - Normal Progressors ; M4.5 vs P)

Treatment group	N	Median months [95% CI]	Wilcoxon p-value
Placebo	113	11 [10; 17]	0.0162
Masitinib 4.5	105	14 [12; 20]	

- Event Free Survival (EFS) was prespecified and there was a benefit (+3 months) with a p=0.0162
- Event being the earlier event between progression, tracheotomy, ventilation or gastrostomy

OS analysis (ITT - Normal Progressors ; M4.5 vs P)

Treatment group	N	Median months [95% CI]	Wilcoxon p-value
Placebo	114	40 [30; 49]	0.0761
Masitinib 4.5	106	46 [33; 69]	

- Trend of survival benefit based on long-term follow-up

Source: Company reports

Exhibit 16. Upon further analysis, the study found an imbalance in 14% of the study population, defined as patients with a complete loss of function in at least one individual component of the ALSFRS-R. Nearly 20% of the patients with complete loss of function were in the masitinib 4.5mg arm vs. only 8% in the placebo arm. This affected the overall study results.

A greater proportion of patients with any complete loss of function were randomized in the Masitinib arm (20%) as compared with the control arm (8%)

Distribution of patients with complete loss of function in at least one individual component of the ALSFRS-R in the primary analysis population

Normal Progressor	Placebo N=113	Masitinib 4.5 mg/kg/d N=105
ALS with complete loss of function in any individual component of the ALSFRS-R (score of zero on any item)	8.0%	20.0%

The disease severity in patients with any complete loss of function was higher in the Masitinib arm

Population	Stat	Placebo	Masitinib 4.5 mg/kg/d
1 item with score 0	n [Q]	8 [8]	10 [10]
2 items with score 0	n [Q]	0	7 [10]
3 items with score 0	n [Q]	0	3 [9]
4 items with score 0	n [Q]	1 [4]	1 [4]
Total	**n [Q]**	**9 [12]**	**21 [33]**

The bracketed [Q] represents the number of items with a 0-score. Some patients (n) had a 0-score (Q) in more than one item

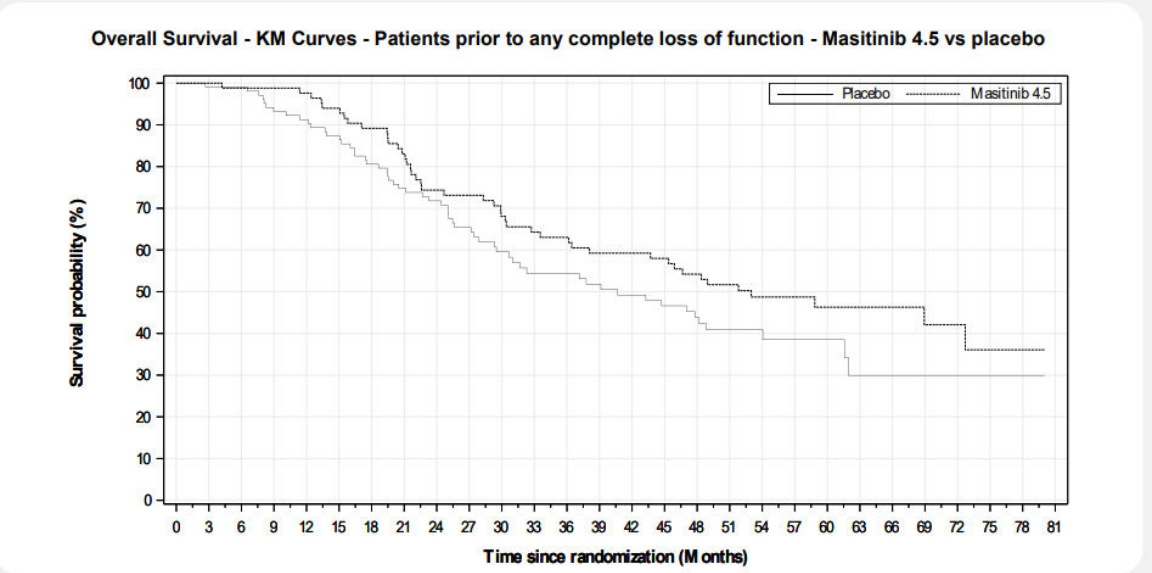
Source: Company reports

Exhibit 17. When the patient demographic imbalances in the study were removed (~86% of primary analysis population), masitinib 4.5mg demonstrated a consistent benefit across all endpoints

		AB10015 Primary Analysis Population (Normal Progressors)	AB10015 Patients with ALS prior to any loss of function (Subgroup)
ΔALSFRS-R (primary endpoint) (mLOCF – pirmary analysis	Diff. of mean	**3.39**	**4.04**
	p-value	**0.0157**	**0.0065**
ΔALSFRS-R (primary endpoint) (Copy Increment in Reference - CIR)	Diff. of mean	**2.68**	**3.13**
	p-value	**0.0462**	0.0308
Combined Assessment of Function and Survival (CAFS)	Relative benefit	+ 14.8%	**+ 20.2%**
	P-value	0.0776	0.0290
ALSAQ-40 (CIR)	Diff. of mean	-6.04 [-11.51;-0.57]	-6.22 [-12.27;-0.17]
	p-value	0.0305	0.044
FVC (CIR)	Diff. of mean	5.85 [-0.98;12.67]	7.59 [0.41;14.77]
	p-value	0.0931	0.0384
Median PFS	Gain	+ 4 months	**+ 9 months**
	Median [95% CI]	20 [14; 30] vs 16 [11; 19]	25 [17, NE] vs 16 [11, 19]
	p-value log rank	0.0159	0.0057
Median OS (Long-term) (censoring of placebo at time of switch to masitinib)	Gain	+ 6 months	**+ 12 months**
	Median [95% CI]	46 [33; 69] vs 40 [30; 49]	53 [36; NE] vs 41 [30; 54]
	p-value log rank	0.0761	0.0192

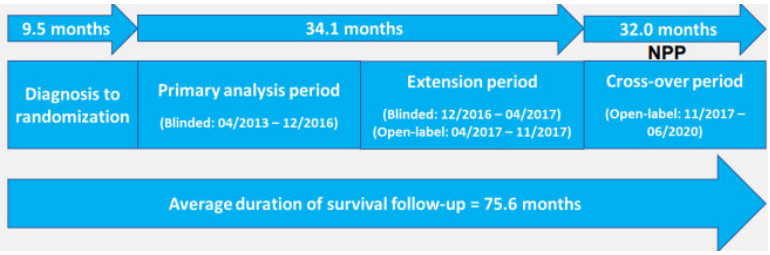
Source: Company reports

Exhibit 18. Patients who were treated earlier from the point of diagnosis and had no loss of function experienced significant survival benefits of 12+ months vs. placebo



Source: Company reports

Exhibit 19. Patients in the study were eligible to enter an extension program with long-term follow-up. The patients averaged 75 months of follow-up.



Source: Company reports

Exhibit 20. The long-term compassionate-use extension study found that a significant portion (43%) of patients receiving masitinib 4.5mg survived >5 years. Furthermore, for these patients, ENCALS models typically predict a survival of 45 months (<4 years), whereas the average survival time observed was 87 months (>7 years). Overall, while this is a subset of patients, we view it as a significant benefit.

A large proportion of patients treated with masitinib 4.5 mg/kg/day have a survival duration of more than 5 years from onset

Normal + Fast Progressors initially randomized in M4.5 treatment arm

Survival Duration	Masitinib 4.5 mg/kg/day (N=128)
Alive > 5yrs	**55 (43.0%)**
Alive > 6yrs	47 (36.7%)
Alive > 7yrs	29 (22.7%)
Alive > 8yrs	14 (10.9%)

* 22.7 % of patients entered the compassionate-use (CU) program

Survival for these patients is not explained by the published model that predicts survival based on baseline characteristics

Comparison of overall observed survival with ENCALS predicted data **Normal + Fast Progressors** initially randomized in M4.5 treatment arm **and alive more than 5 years from onset**

Survival Duration	Masitinib 4.5 mg/kg/day (N=55)
Observed overall survival (average in months)	87
ENCALS predicted overall survival (average in months)	45
Average benefit over ENCALS predicted (in months)	**42**

Source: Company reports

Masitinib has been found to be well tolerated across multiple parameters. Approximately 6,800 patients have received at least one dose of masitinib. More than 3,300 patients have received masitinib for ≥6 months and >2,000 for ≥1 year in oncology and non-oncology indications. Nearly 100 patients have been on therapy for ≥5 years.

Exhibit 21. Masitinib Evaluated in ~6,800 Patients Across All Studies/Indications

	Safety population	Patients exposed for at least			
	All	≥ 6 months	≥ 12 months	≥ 2 years	≥ 5 years
Healthy Volunteers subjects	114	0	0	0	0
Non Oncology subjects	3,338	2,124	1,519	665	50
Oncology subjects	3,333	1,191	574	221	51
Total	**6,785**	**3,315**	**2,093**	**886**	**101**

Source: Company reports

Exhibit 22. The most common treatment-emergent adverse events (TEAEs) were gastrointestinal (GI)-related, skin reactions, and infections (ALS-specific study).

Occurrence of Severe Treatment-Emergent Adverse Events -
Number and Percent of Subjects by System Organ Class, Preferred Term, and Treatment Group (W0-W48)
Safety Population(N=393)

	All TEAEs			Severe TEAEs		
	Placebo (N=133)	Masitinib 3.0 (N=131)	Masitinib 4.5 (N=129)	Placebo (N=133)	Masitinib 3.0 (N=131)	Masitinib 4.5 (N=131)
SYSTEM ORGAN CLASS	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
All	104 (78.2)	111 (84.7)	114 (88.4)	26 (19.5)	28 (21.4)	39 (30.2)
Blood And Lymphatic System Disorders	2 (1.5)	11 (8.4)	19 (14.7)	0 (0.0)	3 (2.3)	2 (1.6)
Cardiac Disorders	4 (3.0)	9 (6.9)	5 (3.9)	2 (1.5)	5 (3.8)	2 (1.6)
Ear And Labyrinth Disorders	2 (1.5)	2 (1.5)	2 (1.6)	0 (0.0)	0 (0.0)	0 (0.0)
Endocrine Disorders	0 (0.0)	0 (0.0)	1 (0.8)	0 (0.0)	0 (0.0)	0 (0.0)
Eye Disorders	1 (0.8)	2 (1.5)	7 (5.4)	0 (0.0)	0 (0.0)	1 (0.8)
Gastrointestinal Disorders	33 (24.8)	48 (36.6)	61 (47.3)	4 (3.0)	4 (3.1)	7 (5.4)
General Disorders And Administration Site Conditions	9 (6.8)	12 (9.2)	20 (15.5)	1 (0.8)	0 (0.0)	0 (0.0)
Hepatobiliary Disorders	3 (2.3)	4 (3.1)	4 (3.1)	0 (0.0)	0 (0.0)	1 (0.8)
Immune System Disorders	0 (0.0)	0 (0.0)	1 (0.8)	0 (0.0)	0 (0.0)	0 (0.0)
Infections And Infestations	28 (21.1)	33 (25.2)	45 (34.9)	3 (2.3)	2 (1.5)	1 (0.8)
Injury, Poisoning And Procedural Complications	15 (11.3)	13 (9.9)	21 (16.3)	0 (0.0)	2 (1.5)	4 (3.1)
Investigations	34 (25.6)	30 (22.9)	38 (29.5)	5 (3.8)	6 (4.6)	6 (4.7)
Metabolism And Nutrition Disorders	11 (8.3)	7 (5.3)	6 (4.7)	3 (2.3)	2 (1.5)	2 (1.6)
Musculoskeletal And Connective Tissue Disorders	13 (9.8)	12 (9.2)	13 (10.1)	0 (0.0)	0 (0.0)	0 (0.0)
Neoplasms Benign, Malignant And Unspecified	3 (2.3)	0 (0.0)	1 (0.8)	2 (1.5)	0 (0.0)	0 (0.0)
Nervous System Disorders	13 (9.8)	18 (13.7)	24 (18.6)	1 (0.8)	4 (3.1)	4 (3.1)
Psychiatric Disorders	18 (13.5)	22 (16.8)	22 (17.1)	0 (0.0)	1 (0.8)	0 (0.0)
Renal And Urinary Disorders	2 (1.5)	6 (4.6)	3 (2.3)	0 (0.0)	0 (0.0)	0 (0.0)
Reproductive System And Breast Disorders	2 (1.5)	4 (3.1)	3 (2.3)	0 (0.0)	0 (0.0)	0 (0.0)
Respiratory, Thoracic And Mediastinal Disorders	16 (12.0)	19 (14.5)	35 (27.1)	8 (6.0)	12 (9.2)	13 (10.1)
Skin And Subcutaneous Tissue Disorders	16 (12.0)	27 (20.6)	39 (30.2)	0 (0.0)	1 (0.8)	2 (1.6)
Surgical And Medical Procedures	1 (0.8)	0 (0.0)	1 (0.8)	0 (0.0)	0 (0.0)	0 (0.0)
Vascular Disorders	6 (4.5)	8 (6.1)	9 (7.0)	1 (0.8)	0 (0.0)	0 (0.0)

Source: Company reports

Exhibit 23. Masitinib was well-tolerated, with the discontinuation rate at week 47 similar between placebo and the masitinib 4.5mg arm. The discontinuation rates also compare favorably to other ALS therapies in development.

Rate of discontinuation at week 48

Patient Status	Placebo	M4.5
Normal	26.5%	30.5%
Fast	52.6%	56.5%

Source: Company reports

Exhibit 24. Masitinib's discontinuation rate at week 48 was favorable compared with other therapies

Comparison of discontinuation rates at week 48 timepoint

Study discontinuation rate	Investigation Arm	Control Arm
Masitinib AB10015 (DB 48-week) [a]	31.1% (33/106)	27.7% (31/114)
AMX0035 NCT05021536 (DB 48-week)	42% (167/397)	43% (115/267)
Edaravone NCT01492686 (Overall, 48-week) [b]	20.9% (14/67)	39.4% (26/66)
Levosimendan NCT03505021 (DB 48-week)	34.0% (112/329)	32.9% (55/167)
Tirasemtiv NCT02496767 (DB 48-week)		
Tirasemtiv 250 mg	36.5% (46/126)	29.8% (56/188)
Tirasemtiv 375 mg	48.4% (61/126)	
Tirasemtiv 500 mg	52.8% (66/125)	
Tirasemtiv (All)	45.9% (173/377)	
Ozanezumab NCT01753076 (DB 48-week)	30.3% (46/152)	27.1% (41/151)

Source: Company reports

Exhibit 25. Masitinib patients had a longer treatment exposure than those receiving placebo (>17 months). Notably, at two-year mark, >2x the patients were in the masitinib arm than placebo.

Treatment Exposure duration by treatment Arm – Blinded Period – Safety population

Treatment arm	All(N)	Average Treatment exposure (months)	% Exposure > 01 Year	% Exposure > 18 Months	% Exposure > 02 Years
Normal + Fast progressors					
Masitinib 4,5 + riluzole	129	17,2	**57%**	43%	**30%**
Masitinib 3 + riluzole	131	17,0	59%	47%	29%
Placebo + riluzole	133	15,9	**61%**	32%	**14%**
Normal progressors					
Masitinib 4,5 + riluzole	105	18.8	**64%**	50%	**35%**
Masitinib 3 + riluzole	110	17.7	62%	51%	30%
Placebo + riluzole	114	16.6	**66%**	34%	**16%**
Normal progressor without loss of function					
Masitinib 4,5 + riluzole	84	19.5	**66%**	50%	**39%**
Masitinib 3 + riluzole	94	18.3	64%	53%	31%
Placebo + riluzole	104	16.9	**67%**	36%	**16%**

Source: Company reports

EMA rejected Conditional Approval Pathway. AB Science initially intended to pursue the Conditional Approval (Accelerated Approval) pathway for masitinib, but it was rejected by the EMA, and the company will have to conduct a Phase 3 confirmatory trial. The pathway was rejected for three reasons:

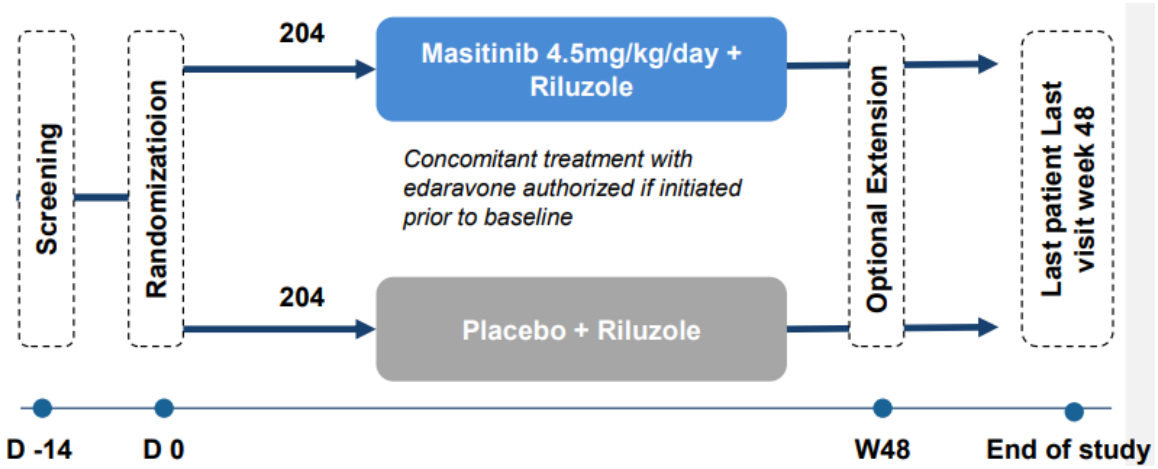
1. Amylyx's (AMLX - NR) Relyvrio was initially given conditional approval in the U.S. and then pulled from the market when its Phase 3 failed. This led the EMA to be more cautious on the Conditional Approval pathway.
2. The EMA is more cautious than the FDA regarding biomarker data, and whether it translates to clinical benefits thus wants to see functional data for an approval.
3. AB Science, in its Phase 2 study, modified the protocol of the study during the study and changed its primary endpoint to "normal progressors". Since these analyses and protocol changes were not pre-specified, the EMA believed an accurate, objective assessment of the data could not be conducted, including with deviations from the inclusion/exclusion criterion. Taken as a whole, the EMA stated the dataset should not be relied upon for confirmatory analysis.

Phase 3 Study Design. AB Science has designed a Phase 3 (AB23005) study optimized based on the results found in the Phase 2. The Phase 3 study is a multicenter, randomized, placebo-controlled, 408-patient, 48-week trial. Patients will be randomized to either masitinib 4.5mg/kg/day + riluzole or placebo + riluzole in a 1:1 ratio between the active and control arms. The primary endpoint differs for the FDA vs. the EMA: CAFS will be used for the FDA filing, and the change in ALSFRS-R for the EMA. The secondary endpoints will include progression-free survival (PFS), overall survival (OS), and quality of life. Surrogate endpoints will include NfL and other biomarkers.

We note that the shift to a nine-month study vs. the six-month studies used in prior ALS development programs, is because it is very difficult to detect a treatment effect in ALSFRS-R at six months; patients typically do not progress quickly enough at six months, and function doesn't deteriorate rapidly enough to observe an effect.

The Phase 3 program is estimated to cost ~€22M. In addition to the study cost, development of the liquid formulation is estimated at €5M, which will occur after the Phase 3 completion. This represents the most cost-effective and fastest path to market for AB Science across its development programs.

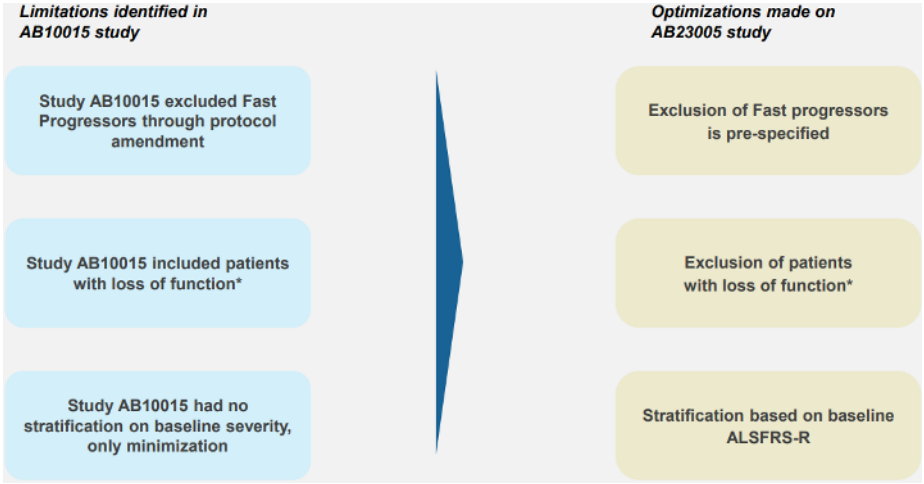
Exhibit 26. Phase 3 Study Design



Source: Company reports

The Phase 3 study includes multiple optimizations to improve potential outcomes. In this study, the exclusion of fast progressors is pre-specified. Notably, patients who have already experienced a loss of function will be excluded. The Phase 3 will also stratify patients based on baseline ALSFRS-R scores. These optimizations should remove the statistical impact of patients who cannot be effectively treated and whose motor neuron function cannot be preserved or recovered.

Exhibit 27. Phase 3 Study Optimizations



Source: Company reports

The Phase 3 study is also optimized for the CAFS endpoint for the FDA submission. Phase 2 was not powered for CAFS, but a trend in the treatment effect was observed. In a post-hoc analysis of patients who had not already experienced a loss of function, a 20%+ stat-sig benefit (p<0.0290) was observed on CAFS. AB Science also conservatively used modest treatment effect assumptions, assuming a 14% effect for CAFS instead of the 20% benefit seen in Phase 2. The 14% corresponds to the effect observed in normal progressors in the Phase 2 study. These more conservative assumptions provide us with greater confidence in the study's success rate, as they include a larger sample size to detect a treatment effect.

Exhibit 28. Phase 3 Optimized for CAFS Endpoint; Phase 2 Found Benefit in Patients Without Prior Loss of Function

Study AB10015 was not powered for CAFS but there was a trend of treatment effect

CAFS	Primary Analysis Population (Normal Progressors)
Relative benefit	+ 14.8%
P-value	0.0776

Optimization

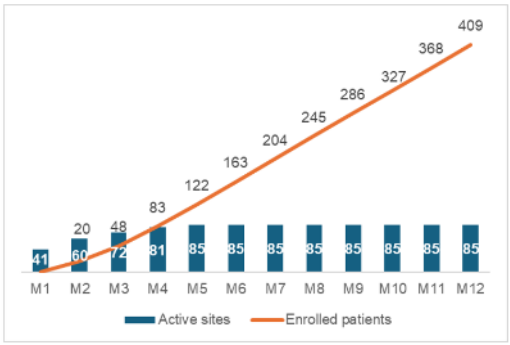
Significant effect on CAFS achieved with patients prior to loss if function

CAFS	Patients with ALS prior to any loss of function
Relative benefit	**+ 20.2%**
P-value	0.0290

Source: Company reports

Exhibit 29. AB Science believes, based on a feasibility analysis, that it will take 12 months to enroll 400 patients

	Pre-selected sites in retained countries	
	Number of Sites	Enrolment per 12 Month
USA	**36**	**262**
EU	**43**	**352**
Belgium	1	10
Denmark	1	7
France	6	21
Germany	8	82
Greece	3	36
Italy	13	123
Latvia	1	7
Norway	1	10
Portugal	1	6
Slovenia	1	5
Spain	5	34
Sweden	2	11
Other Non-EU	**6**	**57**
Argentina	5	47
Serbia	1	10
Total	**85**	**671**



Source: Company reports

Overall, we believe masitinib is positioned for success in a Phase 3 trial for ALS.

The masitinib 4.5mg data have been promising. We acknowledge that the data are from a subset of patients for which Phase 2 was not powered and the analysis was not pre-specified. That being said, we believe the data in normally progressing patients without complete loss of function show clear, statistically significant benefits in both functional improvement and, notably, survival. The recent two ALS approvals in the U.S. were influenced by post-hoc analysis: for Relyvrio, which was later removed from the market, and for Qalsody. In addition, the current FDA administration appears to be emphasizing more supportive pathways and data for rare disease development. The FDA also appears to be emphasizing NfL for regulatory approval purposes in ALS, with survival viewed as corroborative data. Qalsody's approval in April 2023 was based on demonstrating a 55% reduction in NfL over 28 weeks and a 73% reduction in risk of death over 12 months. While Relyvrio was ultimately pulled from the market, its conditional approval was based on biomarker changes and observed survival benefits.

Based on current data, AB Science has optimized the Phase 3 trial to exclude likely non-responders. We believe AB Science has also remained conservative in powering assumptions, which gives us greater confidence in a successful trial. AB Science has an agreement with the FDA on the CAFS endpoint and the EMA on ALSFRS-R. We would expect that if masitinib meets the primary endpoint, AB Science will have a straightforward path to approval. We recognize that most ALS therapies have struggled or have generated mixed results in studies. With that in mind, prior therapies have paved the way for clinical trial design and development knowledge for both regulators and AB Science. The longer timepoints in AB Science's study may generate positive results; most prior studies were conducted over six months rather than nine–12 months.

To our knowledge, Clene (CLNN - Buy) is currently the most advanced ALS therapy in development and has generated survival data across >500 ALS patients, with >1,000 patient-years of safety exposure. Despite the survival benefit, the FDA did not initially agree with Clene's proposal for a conditional approval pathway, as CNM-Au8 missed the primary endpoint in prior studies. The FDA requested NfL data from Clene's open-label expanded access program to determine filing appropriateness, which Clene has provided with positive results. Clene may be the next ALS therapy approved in 2026, and we believe the outcomes of Clene's development may influence AB Science's development. While AB Science is earlier in development, ALS is a very complex, heterogeneous disease, and we believe long-term market may support multiple players. Overall, we are bullish on masitinib's development in ALS.

Masitinib for Amyotrophic Lateral Sclerosis (ALS) Market Model Assumptions

1. We model sales in the U.S. and EU. separately.
2. We assume there are 340M individuals in the U.S., growing at a rate of 0.5% annually.
3. We assume an ALS prevalence rate of 5–7 per 100,000 individuals.
4. We assume 85% of ALS patients are diagnosed and treated.
5. We assume 75% of those patients are the target patient profile for masitinib.
6. We assume 80% of those patients are covered by insurance.
7. We assume this is the target patient population in the U.S.
8. We assume a commercial launch in 2030 in the U.S.
9. We assume a WAC price of $150,000 per prescription in the U.S.
10. We assume 5% annual gross price increases.
11. We estimate gross-to-net discounts of 10% annually.
12. We assume a EUR to USD conversion ratio of €0.90 EUR/USD.
13. We assume there are 450M individuals in the EU, growing at a rate of 0.5% annually.
14. We assume an ALS prevalence rate of 5–7 per 100,000 individuals.
15. We assume 90% of EU ALS patients are diagnosed and treated.
16. We assume 75% of those EU patients fit the target patient profile for masitinib.
17. We assume 90% of those EU patients are covered by insurance or government payers.
18. We assume this is the target patient population in the EU.
19. We assume a commercial launch in 2030 in the EU.
20. We assume a WAC price of $100,000 per prescription in the EU.
21. We assume 3% annual gross price increases.
22. We estimate gross-to-net discounts of 10% annually.
23. We assume a 60% sales risk adjustment to account for stage of development, PDUFA, and commercial risks.

Exhibit 30. Masitinib for Amyotrophic Lateral Sclerosis (ALS) Market Model

Masitinib for Amyotrophic Lateral Sclerosis (ALS)	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E	2034E	2035E
Total U.S. Population	340,000,000	341,700,000	343,408,500	345,125,543	346,851,170	348,585,426	350,328,353	352,079,995	353,840,395	355,609,597	357,387,645
% Growth	0.5%	0.5%	0.5%	0.5%	0.5%	0.5%	0.5%	0.5%	0.5%	0.5%	0.5%
Prevalence of ALS (5-7 per 100,000)	0.01%	0.01%	0.01%	0.01%	0.01%	0.01%	0.01%	0.01%	0.01%	0.01%	0.01%
# of ALS Patients	23,800	23,919	24,039	24,159	24,280	24,401	24,523	24,646	24,769	24,893	25,017
% of ALS Patients Diagnosed and Treated	85%	85%	85%	85%	85%	85%	85%	85%	85%	85%	85%
# of ALS Patients Diagnosed and Treated (U.S. Market)	20,230	20,331	20,433	20,535	20,638	20,741	20,845	20,949	21,054	21,159	21,265
% of Target Patients within Disease	75%	75%	75%	75%	75%	75%	75%	75%	75%	75%	75%
# of Target Patients within Disease	15,173	15,248	15,325	15,401	15,478	15,556	15,633	15,712	15,790	15,869	15,948
% of Patients Covered by Insurance	80%	80%	80%	80%	80%	80%	80%	80%	80%	80%	80%
# of Patients Covered by Insurance	12,138	12,199	12,260	12,321	12,383	12,444	12,507	12,569	12,632	12,695	12,759
Target U.S. Market	12,138	12,199	12,260	12,321	12,383	12,444	12,507	12,569	12,632	12,695	12,759
Market Penetration in U.S.						**3.0%**	**8.0%**	**14.0%**	**20.0%**	**26.0%**	**32.0%**
Total Patients Treated (U.S)						373	1,001	1,760	2,526	3,301	4,083
Total WAC Price Per Rx ($ USD)						$150,000	$157,500	$165,375	$173,644	$182,326	$191,442
Net Change in price						5%	5%	5%	5%	5%	5%
Gross-To-Net Discount						10%	10%	10%	10%	10%	10%
Net Price						$135,000	$141,750	$148,838	$156,279	$164,093	$172,298
Total Revenue (000) ($ USD)						**$50,400**	**$141,826**	**$261,909**	**$394,827**	**$541,634**	**$703,458**
Assumed USD to EUR Conversion Rate						€ 0.90	€ 0.90	€ 0.90	€ 0.90	€ 0.90	€ 0.90
Total Revenue (€000) (EUR)						**€ 45,360**	**€ 127,644**	**€ 235,718**	**€ 355,345**	**€ 487,471**	**€ 633,112**
Total EU Population	450,000,000	452,250,000	454,511,250	456,783,806	459,067,725	461,363,064	463,669,879	465,988,229	468,318,170	470,659,761	473,013,059
% Growth	0.5%	0.5%	0.5%	0.5%	0.5%	0.5%	0.5%	0.5%	0.5%	0.5%	0.5%
Prevalence of ALS (5-7 per 100,000)	0.01%	0.01%	0.01%	0.01%	0.01%	0.01%	0.01%	0.01%	0.01%	0.01%	0.01%
# of ALS Patients	31,500	31,658	31,816	31,975	32,135	32,295	32,457	32,619	32,782	32,946	33,111
% of ALS Patients Diagnosed and Treated	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%
# of ALS Patients Diagnosed and Treated (U.S. Market)	28,350	28,492	28,634	28,777	28,921	29,066	29,211	29,357	29,504	29,652	29,800
% of Target Patients within Disease	75%	75%	75%	75%	75%	75%	75%	75%	75%	75%	75%
# of Target Patients within Disease	21,263	21,369	21,476	21,583	21,691	21,799	21,908	22,018	22,128	22,239	22,350
% of Patients Covered by Insurance	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%	90%
# of Patients Covered by Insurance	19,136	19,232	19,328	19,425	19,522	19,619	19,718	19,816	19,915	20,015	20,115
Target EU Market	19,136	19,232	19,328	19,425	19,522	19,619	19,718	19,816	19,915	20,015	20,115
Market Penetration in EU						**5.0%**	**10.0%**	**16.0%**	**22.0%**	**28.0%**	**34.0%**
Total Patients Treated (EU)						981	1,972	3,171	4,381	5,604	6,839
Total WAC Price Per Rx (€ EUR)						€ 100,000	€ 102,500	€ 105,063	€ 107,689	€ 110,381	€ 113,141
Net Change in price						0%	3%	3%	3%	3%	3%
Gross-To-Net Discount						10%	10%	10%	10%	10%	10%
Net Price						€ 90,000	€ 92,250	€ 94,556	€ 96,920	€ 99,343	€ 101,827
Total Revenue (000) (€ EUR)						**$88,288**	**$181,895**	**$299,799**	**$424,641**	**$556,734**	**$696,399**
Total # of Patients Treated Globally						**1,354**	**2,972**	**4,930**	**6,908**	**8,905**	**10,922**
Total Revenue Globally (000) (€ EUR)						**€ 133,648**	**€ 309,538**	**€ 535,516**	**€ 779,986**	**€ 1,044,204**	**€ 1,329,511**
Risk Adjustment						60%	60%	60%	60%	60%	60%
Total Revenue (€000) - Masitinib for ALS						**€ 53,459**	**€ 123,815**	**€ 214,207**	**€ 311,994**	**€ 417,682**	**€ 531,804**

Source: Maxim Group estimates, Company reports

Alzheimer's Disease

Alzheimer's disease background. Alzheimer's disease (AD) is a chronic, progressive neurodegenerative disorder that begins subtly and gradually worsens, ultimately leading to death. Early symptoms often include short-term memory loss, language difficulties, disorientation, mood changes, reduced motivation, and behavioral shifts. As the disease advances, it severely impairs cognitive and physical functions, resulting in complete dependency. AD accounts for approximately 70% of all dementia cases, with an estimated 6.9 million people affected in the U.S. The condition progresses through four stages of cognitive decline, beginning with pre-dementia or mild cognitive impairment (MCI), which is frequently mistaken for normal aging. Approximately 10%–15% of individuals with MCI develop AD each year. In early stage AD, memory lapses and reduced language fluency become noticeable, especially to those close to the individual, though independence is largely retained. The moderate stage brings more pronounced cognitive deterioration, affecting long-term memory, recognition, and speech, often requiring full-time care. In the advanced stage, patients lose the ability to communicate or carry out basic tasks, becoming entirely reliant on caregivers and eventually bedridden. The disease typically progresses over eight to 10 years from early diagnosis to death, during which patients may also experience anxiety, agitation, and aggression. In its terminal phase, all communicative abilities are lost, and palliative care becomes essential, focusing on symptomatic relief, psychosocial support, and caregiving. AD has a poor prognosis, with most patients ultimately dying from complications such as sepsis, pneumonia, or congestive heart failure. The mortality rate for those with AD is approximately 10% per year.

Current standard of care. Alzheimer's disease treatments can be categorized into three main drug classes: cholinesterase inhibitors, glutamate (NMDA) receptor antagonists, and amyloid-targeting therapies. Cholinesterase inhibitors include drugs such as Cognex (tacrine), Aricept (donepezil), Razadyne (galantamine), and Exelon (rivastigmine), which have been approved for the treatment of mild-to-moderate stages of AD. These medications increase the availability of acetylcholine (ACh) by binding to the enzyme acetylcholinesterase, preventing the breakdown and reuptake of ACh at synaptic junctions. ACh is an important neurotransmitter that regulates mood, behavior, memory, and other cognitive functions. Its depletion in AD patients is associated with the cognitive and behavioral symptoms of the disease. While cholinesterase inhibitors provide modest, temporary benefits in early stages of AD by boosting ACh levels, their effectiveness is limited as they do not address the underlying neurodegeneration and, over time, these drugs lose their efficacy. Namenda (memantine), used in moderate-to-severe AD, is a glutamate receptor modulator that helps reduce excessive glutamate levels in the brain. Overstimulation of NMDA receptors by glutamate can lead to excitotoxicity and neuronal damage. However, memantine also does not address disease pathology, and as the disease advances, neuronal loss becomes more extensive.

In recent years, amyloid-targeting therapies (such as lecanemab and donanemab) have emerged as novel disease-modifying treatments for AD. These monoclonal antibodies aim to slow disease progression by reducing amyloid burden in the brain, marking a shift toward modifying the disease course rather than just alleviating symptoms. However, their use comes with notable safety concerns, including the risk of ARIAs, such as brain swelling or microbleeds, necessitating regular MRI monitoring. Despite these risks, the clinical benefit remains modest, with these therapies slowing the rate of cognitive decline by about 20%–30%.

Alzheimer's pathology: shifting focus from plaques to oligomers. The pathogenesis of Alzheimer's disease has traditionally centered around the accumulation of beta-amyloid (Aβ) plaques as a defining pathological hallmark and primary driver of neurodegeneration. These plaques are insoluble extracellular deposits composed of aggregated Aβ peptides, derived from the sequential cleavage of amyloid precursor protein (APP), a transmembrane protein expressed in neurons. Under normal conditions, APP is cleaved by α- and γ-secretases, producing non-toxic, soluble fragments. In AD, however, APP is aberrantly processed by β- and γ-secretases, resulting in the generation of Aβ peptides, most notably the aggregation-prone 42-amino acid isoform (Aβ42). Once formed, Aβ peptides can follow multiple aggregation pathways: they may persist as monomers, assemble into soluble oligomers, or progressively aggregate into protofibrils, fibrils, and ultimately the insoluble plaques. As mentioned, while plaques have historically defined AD neuropathology, clinical and experimental evidence has increasingly challenged whether plaques are causally linked to cognitive decline. Despite being the most abundant and visible Aβ species, monomers and plaques show poor correlation with disease severity. In contrast, soluble Aβ oligomers, though less abundant, have been demonstrated to disrupt synaptic transmission by embedding into neuronal membranes, altering ion channel activity, and binding to receptors and intracellular signaling proteins. Oligomers also trigger tau hyperphosphorylation, leading to microtubule destabilization, the formation of neurofibrillary tangles, and progressive neuronal loss. Moreover, the extent of cognitive impairment in AD correlates more strongly with the presence of Aβ oligomers than with plaque burden. Although current treatments, such as lecanemab, can effectively reduce plaque levels, their clinical benefit remains limited, providing only modest and temporary slowing of cognitive decline. Symptoms often return once treatment is paused, suggesting continued underlying toxicity from reemerging oligomers. Additionally, serious adverse effects that commonly arise, including brain swelling and microhemorrhages, are largely attributed to off-target interactions with vascular amyloids.

Overview of development of amyloid therapies. The amyloid hypothesis has driven the development of many AD therapies aimed at targeting amyloid-beta, but most have been unsuccessful in delivering meaningful clinical benefit. The lack of success can be attributed to early efforts largely focusing on monoclonal antibodies (mAbs) targeting monomers and plaques, which are now understood to be less central to the disease process. Below is an overview of some key developments in amyloid-targeting therapies, several of which have failed in clinical studies, while few have gained regulatory approval and are currently in use.

- **Bapineuzumab (Pfizer/J&J)**, one of the earliest anti-amyloid drugs, was a mAb that targets Aβ monomers. The drug failed to demonstrate clinical benefit in two Phase 3 studies involving patients with mild-to-moderate AD. The primary endpoints were changes in cognitive function as measured by the ADAS-cog11 and functional ability via the Disability Assessment for Dementia scale over 78 weeks, which showed no significant improvement compared to placebo. Although biomarker changes, such as reductions in amyloid

burden on PET imaging and decreased cerebrospinal fluid phospho-tau levels, were observed in APOE ε4 carriers, these did not translate into meaningful clinical outcomes. Additionally, safety concerns emerged, most notably ARIA, which were more common at higher doses and in APOE ε4 carriers.[9]

- **Crenezumab (Roche/Genentech)** was an mAb designed to target Aβ oligomers and was being developed for the treatment of early AD. Two global P3 studies (CREAD and CREAD2) evaluated its effects over 100 weeks, but both were discontinued in 2019 after interim analyses revealed they were unlikely to meet their primary endpoints. While crenezumab demonstrated a favorable safety profile and low incidence of ARIA, it failed to show significant cognitive benefit (measured by Clinical Dementia Rating–Sum of Boxes [CDR-SB]) or changes in AD biomarkers.[10] Importantly, this failure was not due to targeting the wrong species but rather insufficient selectivity — crenezumab bound broadly to multiple forms of Aβ rather than specifically to the toxic oligomers believed to drive disease progression.

- **Gantenerumab (Roche)** exhibits broad binding to multiple aggregated Aβ species, including plaques and fibrils. The GRADUATE I and II P3 studies evaluated its efficacy in patients with MCI or mild dementia due to AD, all with confirmed amyloid pathology via PET or cerebrospinal fluid (CSF) testing. Patients received gantenerumab or placebo every two weeks, and the primary outcome was the change in CDR-SB score at week 116. While gantenerumab significantly reduced amyloid plaque burden — as evidenced by PET imaging — and showed favorable shifts in CSF biomarkers, it failed to significantly reduce CDR-SB scores.[11] Moreover, the incidence of ARIA-E was relatively high, occurring in nearly 25% of treated patients. The trials' failure in 2022 was a notable setback, reinforcing the idea that broad targeting of aggregated Aβ may be less effective than strategies focused on the most neurotoxic species — soluble Aβ oligomers.

- **Aducanumab (Biogen)**, marketed as Aduhelm**,** was once hailed as a breakthrough in AD treatment as the first disease-modifying drug (DMD) approved to target the underlying pathology rather than just symptoms. Its mechanism of action (MOA) focused on targeting aggregated Aβ plaques in the brain, a hallmark of AD, with the goal of slowing cognitive decline. In June 2021, the FDA granted Aduhelm accelerated approval based on its ability to reduce amyloid plaque levels, despite inconsistent clinical study results and no clear evidence of cognitive improvement. The approval was highly controversial, as many experts questioned whether plaque removal alone was sufficient to produce meaningful improvements. The drug faced backlash from clinicians, limited insurance coverage, and low uptake among patients, ultimately leading to Aduhelm being pulled from the market in November 2024.

- **Lecanemab (Biogen/Eisai)**, marketed as Leqembi, initially received an accelerated approval in January 2023, which was followed by full approval in July 2023 based on clinical data demonstrating modest slowing of cognitive decline in early AD. The drug targets soluble Aβ protofibrils, intermediate aggregates in the Aβ aggregation pathway that are thought to be closer to the toxic species driving AD than the insoluble plaques targeted by earlier therapies. However, growing evidence points to smaller, highly toxic Aβ oligomers as the real drivers of neurodegeneration, meaning protofibrils may still miss the mark. In the P3 CLARITY study, significant risks including ARIA (both brain swelling and microhemorrhages) were commonly observed, particularly among patients with the APOE ε4 genotype. Several deaths were reported in the study, including some linked to intracerebral hemorrhage in patients receiving concurrent treatments like anticoagulants. Given its limited benefits, safety concerns, and the burden of biweekly infusions, the overall risk-benefit ratio of lecanemab remains a subject of ongoing scrutiny.

- **Donanemab (Eli Lilly)**, marketed as Kisunula, is the most recent addition to the class of amyloid plaque-targeting antibodies, approved for early symptomatic AD in July 2024. In the P3 TRAILBLAZER-ALZ 2 study, donanemab modestly slowed the rate of cognitive and functional decline, particularly in patients with intermediate tau levels, with nearly half of patients showing no progression at one year. Safety concerns, however, were notable, including ARIA observed in about a quarter of patients treated in this study. Due to this safety issue, regular MRI monitoring is required, especially during the first few months. In March 2025, European regulators decided not to approve donanemab in Europe due to safety concerns, further highlighting the challenges of this therapeutic class.

Masitinib and targeting mast cells represents a novel approach to tacking Alzheimer's disease

Based on prior studies, masitinib has been found to have neuroprotective effects by targeting microglia and macrophages. AB Science is pursuing an unorthodox development target for AD. Studies have found that mast cells and microglia are implicated in the pathophysiology of Alzheimer's disease (AD). Since masitinib has been found to inhibit mast cell and microglia proliferation, it may be an applicable treatment for early stage AD. In an AD mouse model, it restored learning and protected synapses via mast cell inhibition.

Masitinib demonstrated positive benefits in a Phase 2b/3 trial in mild Alzheimer's patients

Study design. The Phase 2b/3 study was a multicenter (118 sites), randomized, double-blind, placebo-controlled, two parallel-group (four-arm) trial with a primary treatment period of 24 weeks. The study enrolled 718 patients, with 51.5% of the patients enrolled in the EU. Patients were aged >50 years, with a clinical diagnosis of mild-to-moderate probable AD and a Mini-Mental State Examination (MMSE) score of 12–25.

[9] Salloway et al. N Engl J Med 2014;370:322-333
[10] Ostrowitzki et al. JAMA Neurol. 2022;79(11):1113-1121
[11] Bateman et al. N Engl J Med 2023;389:1862-1876

Masitinib was administered orally as an add-on therapy to the patients' existing standard care, which consisted of a stable dose of cholinesterase inhibitors (donepezil, rivastigmine, or galantamine) and/or memantine.

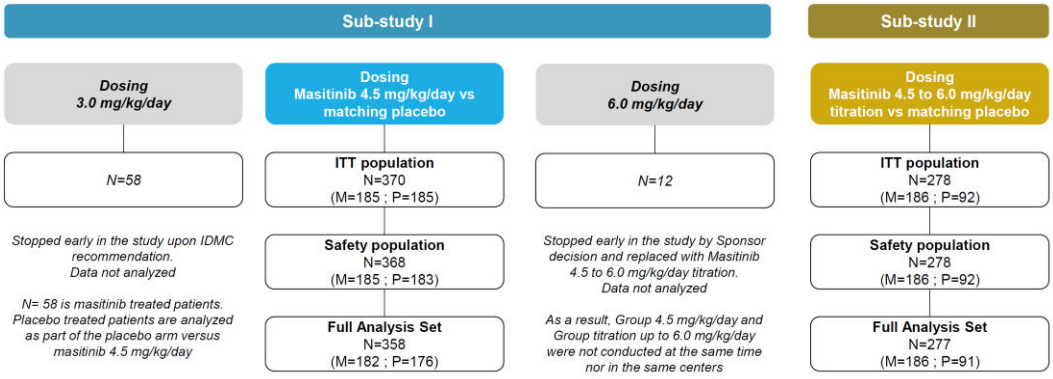
The trial had two independent parallel groups with different randomization ratios:

- **Group 1 (primary analysis):** Patients were randomized 1:1 to either masitinib 4.5mg/kg/day (fixed dose, administered as two intakes) or matched placebo.
- **Group 2 (secondary/exploratory analysis):** Patients were randomized 2:1 to either masitinib 6.0mg/kg/day (initial dose 4.5mg/kg/day for 12 weeks, then titrated up to 6.0mg/kg/day) or matched placebo.

The study had two co-primary endpoints, which include:

1) **Cognitive Function:** Alzheimer's Disease Assessment Scale – Cognitive Subscale (ADAS-Cog)
2) **Functional Ability:** Alzheimer's Disease Cooperative Study Activities of Daily Living Inventory scale (ADCS-ADL)

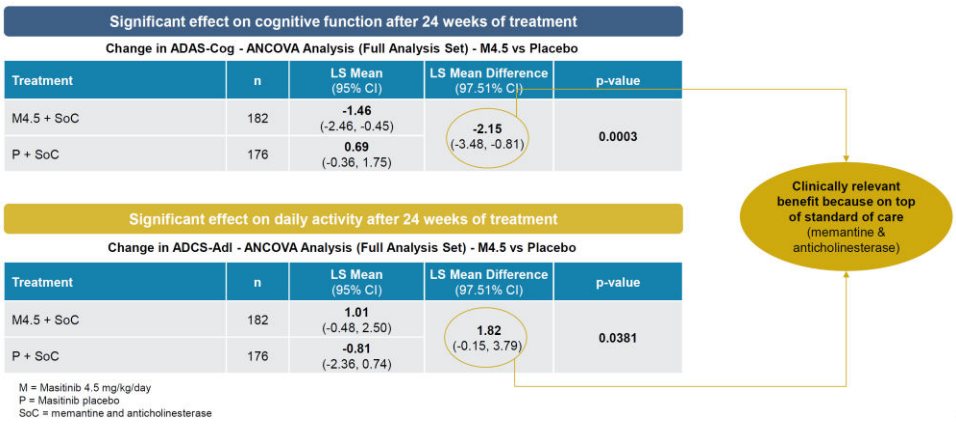
Exhibit 31. Masitinib Phase 2b/3 Study Design in Alzheimer's Disease



Source: Company reports

Masitinib 4.5mg met the primary endpoints on ADAS-Cog and ADCS-ADL. The study found that masitinib met the primary endpoint of the Alzheimer's Disease Assessment Scale – Cognitive Subscale (ADAS-Cog). Patients on masitinib experienced a -1.46 change from baseline on ADAS-Cog vs. +0.69 placebo (p = 0.0003). Masitinib patients also demonstrated a statistically significant improvement from baseline vs. the placebo group on the Alzheimer's Disease Cooperative Study Activities of Daily Living (ADCS-ADL) score, which assesses self-care and daily function. Patients in the masitinib group experienced a change of +1.01, indicating an improvement in function, vs. -0.81 placebo, indicating a loss of function (p=0.0381). There were also significantly fewer patients in the masitinib 4.5mg group who reached the stage of severe dementia (defined as MMSE < 10) vs. the placebo group after 24 weeks of treatment. The 3.0mg and 6.0mg groups did not demonstrate statistically significant benefits. Masitinib 4.5mg was also found to be well-tolerated. The safety profile was consistent with prior masitinib studies, including maculopapular rash, neutropenia, hypoalbuminemia.

Exhibit 32. Statistically Significant Effects Seen at 24 Weeks on Cognition and Function



Source: Company reports

Exhibit 33. Statistically Significant Effect Seen on iARDs (*integrated Alzheimer's Disease Rating Scale (iADRS) combining scores from, the Alzheimer's Disease Assessment Scale-Cognitive subscale (ADAS-Cog) and the Alzheimer's Disease Cooperative Study – instrumental Activities of Daily Living (ADCS-iADL)***).**

Mean (+/SEM) Plot of Alzheimer's Disease Assessment Composite Cog-ADL (iARDS) - Masitinib 4.5 versus Placebo (Full Analysis Set Population)

Treatment	n	LS Mean (95% CI)	LS Mean Diff. (9(% CI)	p-value
M4.5 + SoC	182	2.24 (0.980)	3.45 (0.90, 6.00)	0.0025
P + SoC	176	-1.21 (1.023)		

Source: Company reports

Exhibit 34. Masitinib 4.5mg Was Found to Reduce the Number of Patients Reaching Severe Dementia and the Time to Severe Dementia

Dementia- M4.5 vs Placebo Pooled (FAS)

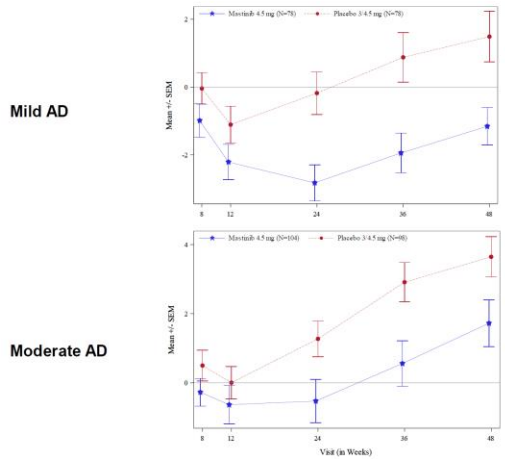
							p-value		Hazard	
Treatment group	Total	No. of Events	Percentage Events	No. Censored	Percentage censored	Median [95% CI]	KM p-Value	Log Rank	Ratio (95% CI)	p-Value
M4.5 + SoC	182	2	1.10	180	98.90	Not reached [;]	0.0446	0.0403	0.19 (0.0,0.8)	0.0276
Pooled P + SoC	267	15	5.62	252	94.38	6.3 [5.9;6.3]				

Baseline MMSE (FAS)	Masitinib + SoC (N = 182)	Pooled Placebo + SoC (N =267)
< 14	18 (9.9)	30 (11.2)
< 17	54 (29.7)	81 (30.3)

Source: Company reports

Exhibit 35. Masitinib 4.5mg Was Found to Generate a Greater Effect in Patients with Mild Impairment vs. Moderate Impairment on ADAS-Cog

Mean (+/SEM) Plot of ADAS-COG - Masitinib 4.5 versus Pooled Placebo (FAS))



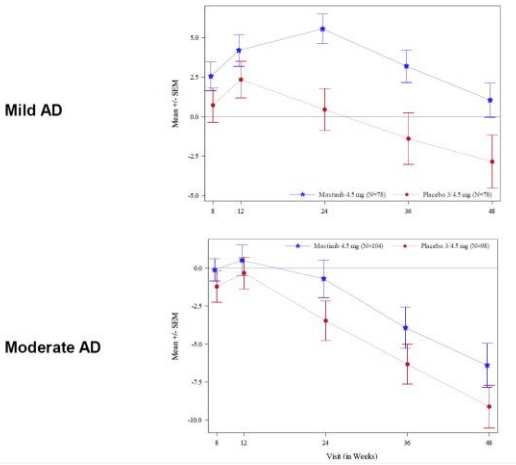
Week 24	n	LS Mean (95% CI)	LS Mean Diff. (96% CI)	p-value
Mild patients				
M + SoC	78	-3.23 (0.807)	-2.62 (-4.45, -0.78)	0.0015
P + SoC	78	-0.61 (0.812)		
Moderate patients				
M + SoC	104	-0.55 (0.655)	-1.80 (-3.66, 0.06)	0.0300
P + SoC	98	1.25 (0.721)		

M = Masitinib 4.5 mg/kg/day ; P Masitinib placebo 3 & 4.5 ; SOC = memantine and anticholinesterase

Source: Company reports

Exhibit 36. Masitinib 4.5mg Was Found to Generate a Greater Effect in Patients with Mild Impairment vs. Moderate Impairment on Composite iARDS

Mean (+/SEM) Plot of iARDS - Masitinib 4.5 versus Pooled Placebo (FAS))



Week 24	n	LS Mean (95% CI)	LS Mean Diff. (96% CI)	p-value
Mild patients				
M + SoC	78	5.02 (1.490)	4.76 (1.37, 8.15)	0.0018
P + SoC	78	0.26 (1.497)		
Moderate patients				
M + SoC	104	0.33 (1.343)	2.41 (-1.23, 6.06)	0.1359
P + SoC	98	-2.08 (1.413)		

M = Masitinib 4.5 mg/kg/day ; P Masitinib placebo 3 & 4.5 ; SOC = memantine and anticholinesterase

Source: Company reports

Phase 3 Trial Optimized for Success

AB Science is making multiple adjustments to the Phase 3 trial to maximize the likelihood of success. The Phase 3 trial will enroll only mild AD patients (MMSE: 20–25), as masitinib has been found to have a greater effect in earlier-stage patients. The Phase 3 will evaluate ADAS-Cog as the primary endpoint and iARDS as a secondary endpoint. If the study is successful only on the primary endpoint, AB Science will pursue an indication for symptomatic treatment of memory loss. If both ADAS-Cog and iARDS are stat-sig, AB Science will pursue an indication for disease-modifying treatment of AD. The primary endpoint will be at 24 weeks. The study will include an open-label extension period out to 48 weeks, with a repeat analysis at week 48.

Study design. The Phase 3 study design has already been discussed with the FDA and EMA. The Phase 3 trial will be a multicenter (91 sites), double-blind, randomized, placebo-controlled trial enrolling 600 patients. Patients will be randomized 1:1 to masitinib 4.5mg or placebo. The study will be 24 weeks.

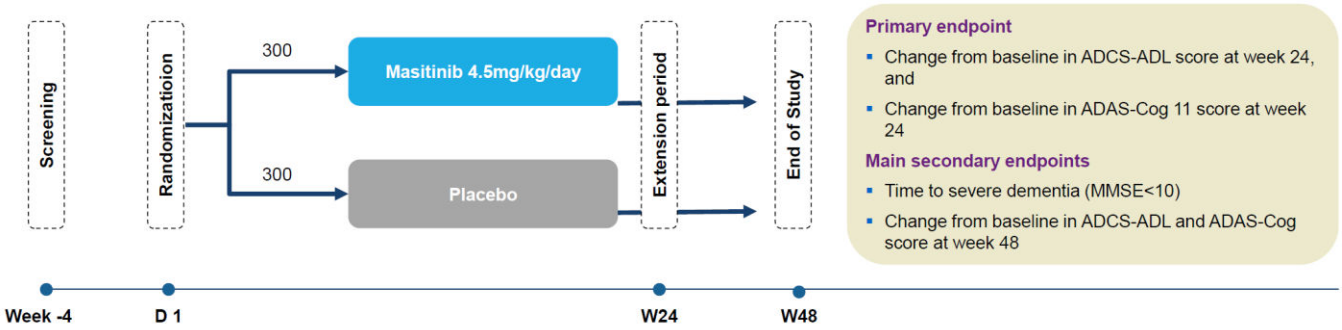
The co-primary endpoints are:

1) Change from baseline in ADCS-ADL score at week 24
2) Change from baseline in ADAS-Cog 11 score at week 24

The secondary endpoints are:

1) Time to severe dementia (MMSE<10)
2) Change from baseline in ADCS-ADL and ADAS-Cog scores at week 48

Exhibit 37. Phase 3 Trial Design



Main inclusion criteria

- Patients with clinical diagnosis of Alzheimer's disease based on cognitive impairment and daily functional dependency at screening visit
- Patients with ADCS-ADL score at screening visit and baseline visit < 73
- Patients with MMSE ≥ 14 and ≤ 25 at screening visit and baseline visit

Study Status

Confirmatory phase 3 study design has been discussed with FDA and EMA

Approved countries (91 sites)

- Belgium, Bulgaria, Czech Republic, France, Germany, Greece, Hungary, Ireland, Italy, Poland, Portugal, Romania, Slovakia, Spain, Sweden, United Kingdom
- USA

Source: Company reports

Potential combination programs. AB Science is also evaluating combination therapies with masitinib and amyloid-clearing agents. The company plans to evaluate masitinib + amyloid-clearing agents in treatment-naïve patients, as well as masitinib as an adjunctive therapy in pre-treated patients and treatment failures.

Exhibit 38. Combination Potential in Alzheimer's Disease

Combined with amyloid-targeting drugs

- Phase 1/2
- Mild AD, not previously treated (or only with lecanemab or donanemab), ApoE ε4 non-carriers or heterozygotes, with positive amyloid PET
- Masitinib + registered anti-amyloid therapy versus anti-amyloid therapy
- 72 weeks
- Change in ADAS-Cog
- Change in iARDS

Adjuvant to amyloid-targeting drugs

- Phase 2
- Mild & Moderate AD, achieving negative positive amyloid PET following treatment with registered amyloid-targeting drugs
- Masitinib versus placebo
- 48 weeks
- Change in ADAS-Cog
- Change in iARDS

Source: Company reports

Multiple Sclerosis

Multiple sclerosis (MS) is an autoimmune disorder that impacts the central nervous system and leads to immune-mediated destruction of the brain, optic nerves, and spinal cord. In MS, the immune system attacks the myelin sheath, the protective covering of the axons in neurons. This leads to neuronal damage and death, leading to impaired motor function, cognitive deficits, visual impairment, and other neurological issues. The damaged areas develop scar tissue, creating multiple areas of scarring (sclerosis).

Pathology. MS is characterized by the presence of multifocal lesions or MS plaques. During the acute or active phase, mononuclear cells (lymphocytes, microglia, and macrophages) destroy myelin and, to some extent, oligodendrocytes. Eventually, gliosis develops and demyelinated axons traverse glial scar tissue. Oligodendrocytes attempt remyelination at this stage, but the process is limited. In more advanced lesions, remyelination is prevented because gliosis creates a barrier between the myelin-producing cells and their targets. In many cases, the inflammation subsides only to occur at another location, or the lesions expand peripherally.[12]

Patients are usually diagnosed via MRI, with active plaques showing gadolinium enhancement. Gadolinium enhancement is associated with inflammation and increased vascular permeability. Active plaques may resolve with treatment or time. MRI may also reveal inactive plaques around the lateral ventricles, and advanced MS may exhibit brain atrophy.

Patients with MS have also been found to have bioenergetic deficits in their brains, as measured by 31-phosphorus magnetic resonance spectroscopy (31P-MRS). Oligodendrocyte precursor cells near MS lesions show impaired mitochondrial activity, and bioenergetic deficits have been observed in autopsied brains of MS patients.

MS typically develops in patients between the ages of 20–40 and is the leading cause of non-traumatic disability in young adults. The prevalence is ~3x higher in women than in Men, except in primary progressive MS. MS is the most common inflammatory demyelinating disease. The cause of MS is not well understood, but scientists believe it likely involves a combination of genetic factors, immune system abnormalities, and environmental influences. Patients typically develop one of four courses (phenotypes) of MS, clinically isolated syndrome (CIS), relapsing-remitting MS (RRMS), primary progressive MS (PPMS), and secondary progressive MS (SPMS).[13]

MS Phenotypes

- **Clinically Isolated Syndrome (CIS).** CIS is the first episode of neurologic symptoms involving inflammation and demyelination. CIS lasts at least 24 hours but generally does not meet the diagnostic criteria for MS, as not all patients will develop MS. Patients with CIS may develop lesions in the brain similar to those seen in MS and have a high likelihood of a second neurologic episode. When CIS occurs without lesions in the brain, the risk of developing MS is lower. CIS patients may be treated with DMD therapies for the indication.
- **Relapsing-remitting MS (RRMS).** RRMS is the most common disease course, affecting ~85% of MS patients at diagnosis. RRMS presents as clearly defined attacks (relapses) or new symptoms. The relapses are followed by periods of partial or complete recovery (remission). Symptoms may fully resolve or persist and become permanent. There is no disease progression during remission. Patients with RRMS can be categorized as active (relapsing and/or with new MRI activity), not active, worsening (increased disability), or not worsening.
- **Secondary progressive MS (SPMS).** Some patients with RRMS transition to SPMS, characterized by progressive worsening of neurologic function and accumulation of disability over time. SPMS patients can be described as active, not active, with progression (disability accumulation), or without progression.
- **Primary progressive MS (PPMS).** PPMS patients have worsening function and accumulation of disability from symptom onset, without early relapses or remissions. PPMS patients can be described as active, not active, with progression, or without progression.

Prevalence in the U.S. It is estimated that there are 852,000–914,000 individuals living in the U.S. with MS.[14] A prior study found differing prevalence rates between men and women.[15] A study by Wallin et al. reported a prevalence rate of ~309.2 per 100,000 men, with a prevalence rate of 450.1 per 100,000 for women and 159.7 per 100,000, suggesting women are almost ~3x more likely to develop MS than men. The prevalence rate was found to be highest in the 55–64-year age group. It is estimated that there are ~2.5M patients globally, and the global immunomodulator market is valued at ~$23B.

Currently, there are limited options for progressive MS, with no FDA-approved therapies for non-active secondary progressive MS and only one approved therapy for primary progressive MS. Multiple disease-modifying therapies (DMT) are approved to treat MS, but these therapies primarily manage symptoms or reduce the magnitude of inflammation. Nearly all approved therapies target relapsing MS. Their mechanisms of action generally involve immunosuppression or immunomodulation to limit autoimmune attacks on myelin. Immunomodulatory DMTs not only reduce the risk of inflammatory attacks but also slow the development of disabilities, thereby delaying progression from relapsing MS to secondary progressive multiple sclerosis (SPMS). While some DMTs substantially reduce autoimmune attacks, none address the ongoing loss of function in non-active MS patients (those no longer experiencing attacks). Additionally, none of the approved therapies have demonstrated an ability to improve remyelination of damaged and remyelinated axons or protect neurons. Ocrevus (ocrelizumab) is currently the

[12] https://neuropathology-web.org/chapter6/chapter6aMs.html
[13] https://www.nationalmssociety.org/What-is-MS/Types-of-MS
[14] https://www.nationalmssociety.org/About-the-Society/News/Landmark-Study-Estimates-Nearly%C2%A01-Million-in-the-U
[15] https://n.neurology.org/content/92/10/e1029

only therapy approved for primary progressive MS. This leaves a significant market opportunity for AB Science to develop masitinib for primary progressive MS and non-active secondary progressive MS.

Exhibit 39. Significant Unmet Need in Progressive Multiple Sclerosis

	Manufacturer	Label				First approved
		PPMS	Non-active SPMS*	Active SPMS	RRMS	
Distribution of patients *(Estimated Nbr of patients Europe + USA)*		**15%** *(~ 150 000)*	**35%** *(~ 350 000)*	**10%** *(~ 90 000)*	**40%** *(~ 400 000)*	
Total number of drugs registered		**1**	**0**	**15**	**16**	
Mayzent (siponimod)	Novartis			X	X	2019
Vumerity (diroximel fumarate)	Alkermes / Biogen			X	X	2019
Ocrevus (ocrelizumab)	Roche / Genentech	X		X	X	2017
Mavenclad (cladribine)	EMD Serono / Merck			X	X	2017
Plegridy (peginterferon beta-1a)	Biogen			X	X	2014
Tecfidera (dimethyl fumarate)	Biogen	Masitinib positioning		X	X	2013
Aubagio (Teriflunomide)	Sanofi-Aventis			X	X	2012
Gilenya (fingolimod)	Novartis			X	X	2010
Extavia (interferon beta-1b)	Novartis			X	X	2008
Tysabri (natalizumab)	Biogen			X	X	2004
Lemtrada (alemtuzumab)	Sanofi / Genzyme			X	X	2001
Rebif (interferon beta-1b)	Serono			X	X	1998
Avonex (interferon beta-1a)	Biogen			X	X	1996
Copaxone (glatiramer acetate)	Teva Pharms			X	X	1996
Betaferon / Betaseron (interferon beta-1b)	Bayer Healthcare			X	X	1993

Source: Company reports

Masitinib MOA may be effective in treating progressive MS. It is understood that progressive forms of MS (PPMS and non-active SPMS) are primarily driven by self-perpetuating innate immunity-related inflammation within the central nervous system (CNS). Microglia and mast cells have been found to be strongly associated with the pathophysiology of MS. Masitinib's MOA, which modulates mast cells and activated macrophages/microglia, may be effective at slowing or preventing worsening of disability in progressive MS.

Masitinib has demonstrated directionally positive data in treating progressive MS in a Phase 2 study

Masitinib was initially evaluated in a proof-of-concept study in patients with primary progressive MS (PPMS) or relapse-free secondary progressive MS (rfSPMS) patients. The study was a multicenter, randomized, placebo-controlled, exploratory pilot trial that enrolled 35 patients. Patients were randomized 3:1 to masitinib or placebo (27 to masitinib, eight to placebo). In the active arm, there were nine PPMS and 15 rfSPMS patients; in the placebo group, there were three PPMS and three rfSPMS. The patients were administered masitinib or placebo orally at a dose ranging from 3mg/kg/day to 6mg/kg/day for at least 12 months. The primary endpoint was the change relative to baseline in the Multiple Sclerosis Functional Composite (MSFC) score. A clinical response was defined as an increase in MSFC score relative to baseline of >100%.[16]

Positive trends seen in data. Although the primary endpoint was not met, masitinib demonstrated directionally positive effects on MS-related impairment. Masitinib improved MSFC scores relative to baseline, compared with worsening MSFC scores in patients receiving placebo (+103% ± 189 versus -60% ± 190 at month 12, respectively). This effect was observed in both PPMS and rfSPMS patients and appeared as early as month 3, sustained through month 18. A total of 32% of assessable masitinib patients were classified as "clinical responders" (defined as an increase in MSFC score greater than 100%) after 12 months vs. none in the placebo group. The Expanded Disability Status Scale (EDSS) score remained stable for both treatment groups over the study period.

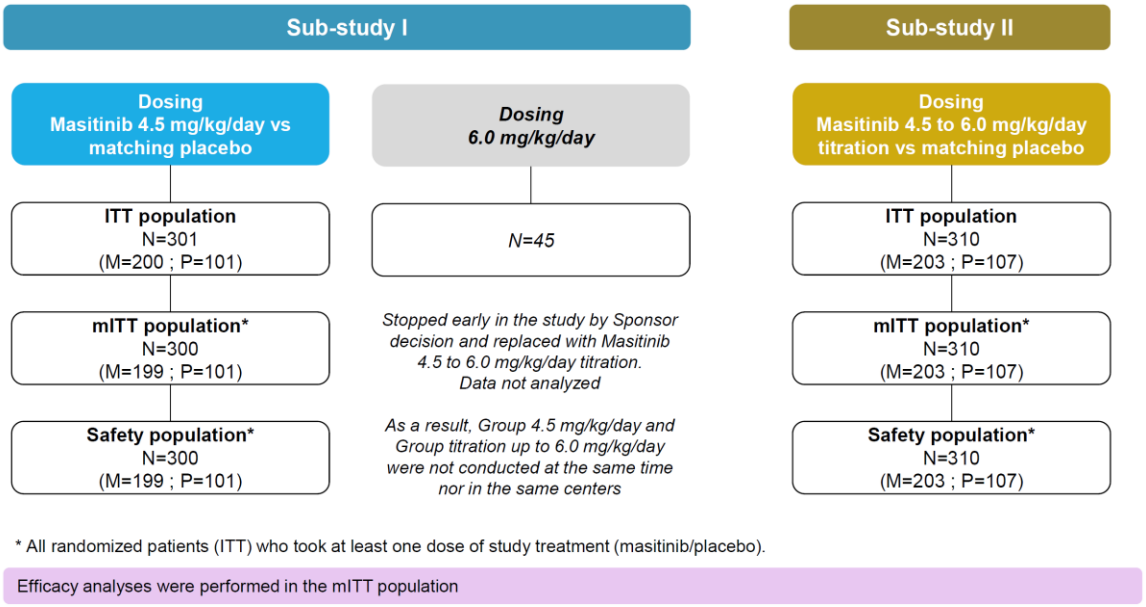
Well tolerated. Masitinib was relatively well tolerated. The most common adverse events (AEs) were asthenia, rash, nausea, edema, and diarrhea. The overall frequency of AEs was similar to placebo; however, a higher incidence of severe and serious events was associated with masitinib treatment.

Following the initial Phase 2 study, AB Science evaluated masitinib in a larger Phase 2b/3 study

Study design. The Phase 2b/3 study was a multicenter, randomized, double-blind, two parallel-group, placebo-controlled trial. The study enrolled 611 patients and evaluated two dose levels of masitinib vs. equivalent placebo. Patients were randomized 2:1 to masitinib or placebo. The trial treated patients with primary progressive MS (PPMS) or non-active secondary progressive MS (nSPMS) without relapse for ≥two years, aged 18–75 years, with a baseline Expanded Disability Status Scale (EDSS) score of 2.0–6.0, regardless of time from onset. The patients were treated for 96 weeks. The primary endpoint was change in EDSS from baseline, assessed using repeated measures with a generalized estimating equation (timeframe W12–W96, measured every 12 weeks). Positive values indicated disease progression.

[16] Vermersch, P., Benrabah, R., Schmidt, N., Zéphir, H., Clavelou, P., Vongsouthi, C., Dubreuil, P., Moussy, A., & Hermine, O. (2012). MASITINIB treatment in patients with progressive multiple sclerosis: A randomized pilot study. *BMC Neurology*, *12*(1). https://doi.org/10.1186/1471-2377-12-36

Exhibit 40. Phase 2b/3 Study Design

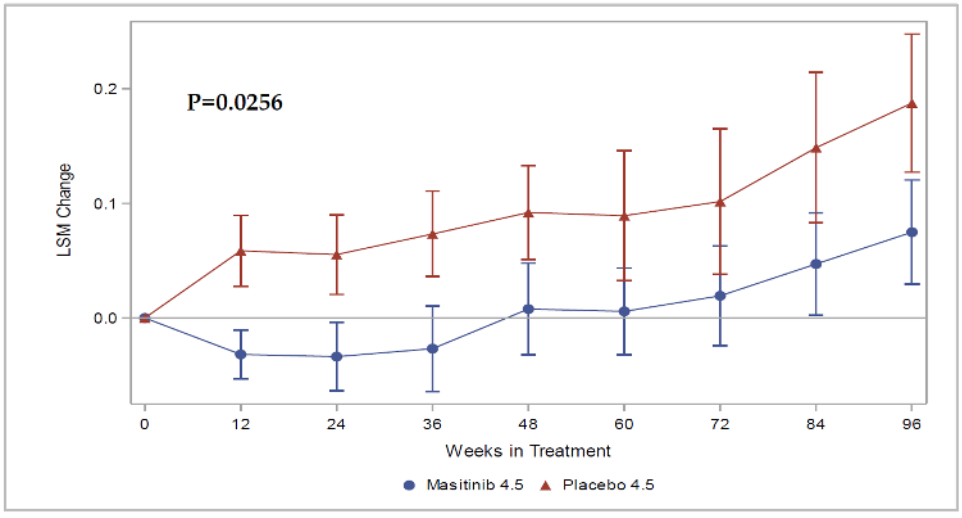


Source: Company reports

Statistically significant results seen with masitinib 4.5mg in slowing disease progression. The study demonstrated a statistically significant benefit for the lower dose of masitinib (4.5mg/kg/day) vs. placebo in slowing disability progression (0.001 vs. 0.098, with a between-group difference of −0.097 (97% CI −0.192 to −0.002); p = 0.0256). Data from masitinib 6.0mg/kg/day were inconclusive. The safety profile was consistent with masitinib's known profile (diarrhea, nausea, rash, and hematologic events), with no elevated risk of infection.

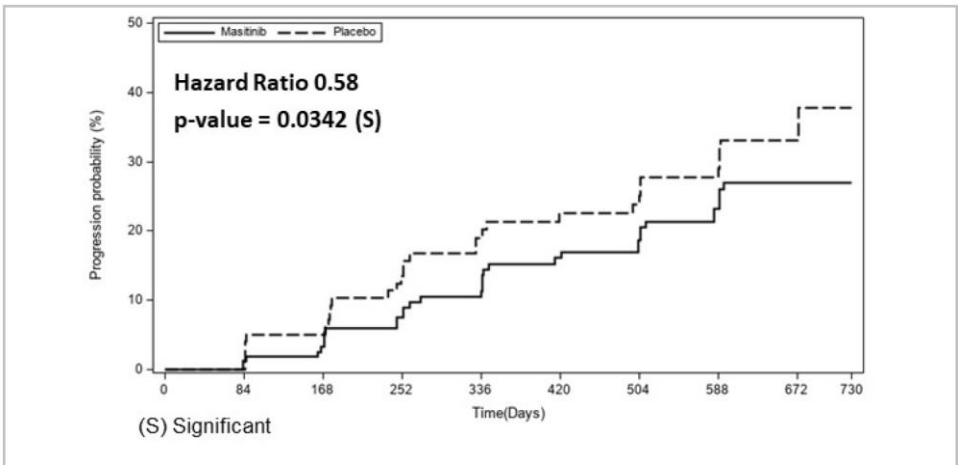
The Phase 2b/3 study found that masitinib 4.5mg statistically significantly reduced MS disease progression on EDSS. We would note the study enrolled advanced MS patients. The median age of patients was 50 years. The median duration of the first MS symptom was 12.4 years vs. 12.2 years for placebo. The Median EDSS score was 5.5 in both the masitinib and placebo groups. The percentage of patients with an EDSS score of six was 49% in the masitinib group and 47.5% in the placebo group.

Exhibit 41. Masitinib Demonstrated Statistically Significant Reduction in Disease Progression on EDSS



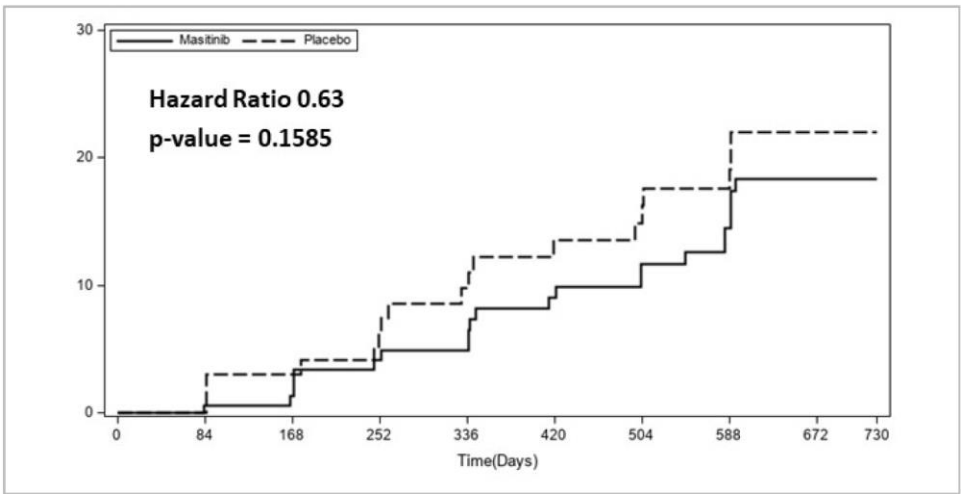
Source: Company reports

Exhibit 42. Masitinib Demonstrated a 42% Reduction in Time to Disability



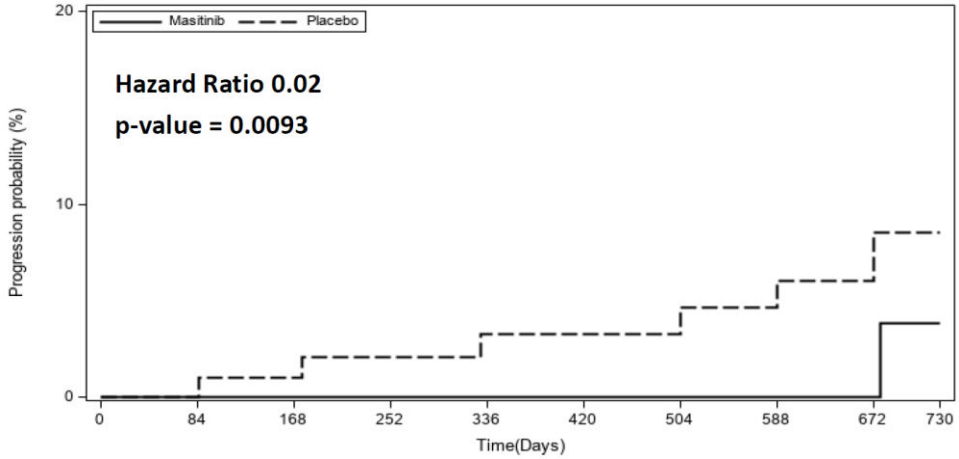
Source: Company reports

Exhibit 43. Masitinib 4.5mg Demonstrated a 37% Risk Reductio in Time to Confirmed Disability Progression (12 Weeks)



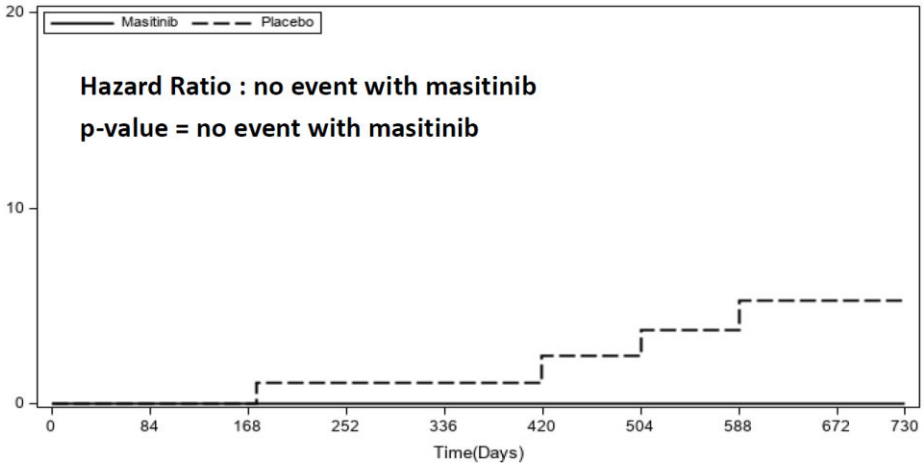
Source: Company reports

Exhibit 44. Kaplan Meier plot of Analysis of time to First EDSS score of 7.0



Source: Company reports

Exhibit 45. Kaplan Meier plot of Analysis of time to Confirmed EDSS score of 7.0



Source: Company reports

Exhibit 46. The study did not find a significant benefit when dose-escalating patients from 4.5mg to 6.0mg. Numerically, masitinib 6.0mg was similar to masitinib 4.5mg. That being said, the placebo groups in the 4.5mg to 6.0mg evaluation cohorts experienced unexpected improvement, and thus mathematical statistical significance was not achieved. The placebo group in the 4.5mg-only analysis experienced worsening, as was expected, resulting in statistical significance in that analysis.

Analysis of change from baseline in EDSS Score (Primary Analysis)

Treatment	N	LS Means	Estimate (CI)	p-Value
M4.5 to 6.0 mg/kg/day	203	0.009	0.014 (-0.111, 0.1399)	0.8019
Placebo	**107**	**-0.005**		
M4.5 mg/kg/day	199	0.001	-0.097 (-0.192, -0.002)	0.0256
Placebo	**101**	**0.098**		

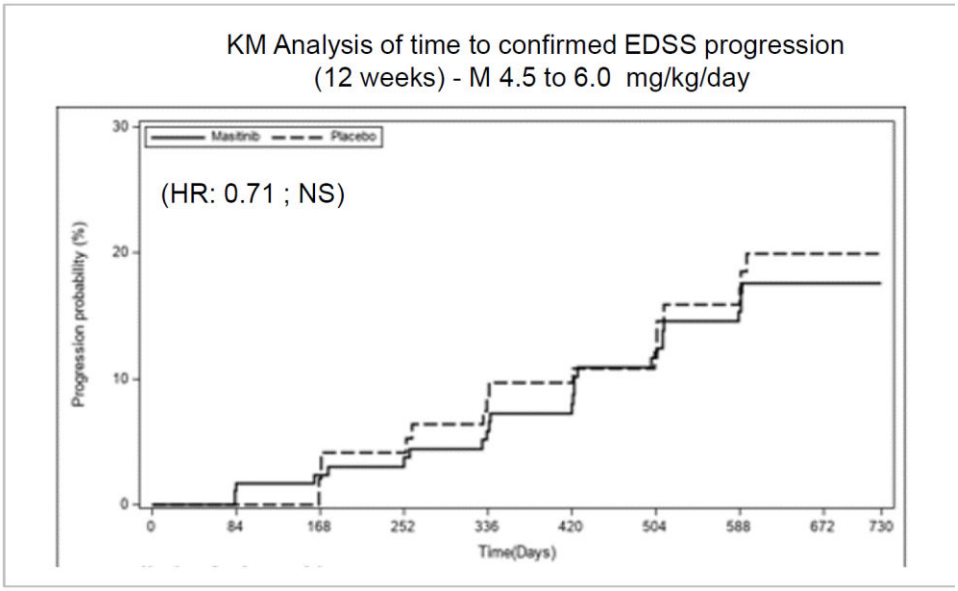
Similar treatment effect of Masitinib

Unexpected improvement

Expected worsening

Source: Company reports

Exhibit 47. Dose-escalating patients from masitinib 4.5mg to 6.0mg demonstrated a 29% risk reduction in time to confirmed disability progression (12 Weeks) (not statistically significant).



Source: Company reports

AE profile consistent with prior masitinib studies. The AE profile of masitinib was consistent with prior studies, with no new safety signals. The most common AEs were diarrhea, maculopapular rash, nausea/vomiting, peripheral edema, pruritus, and various laboratory assessments There were no fatal SAEs occurring in ≥2 patients over 96 weeks at the masitinib 4.5mg dose.

Masitinib's long-term safety profile has been established across multiple indications. The majority of AEs have been found to be mild to moderate in severity. The most common AEs were periorbital edema, anemia, diarrhea, nausea, vomiting, and rash. The AEs were primarily seen in the first three months and were manageable with dose adjustment. Future masitinib studies will include a dose-escalation protocol going from 3mg to 4.5mg to reduce AE incidence at the beginning of a treatment cycle. Notably, since masitinib is not an immunosuppressive therapy, it can be used chronically.

Exhibit 48. MS Phase 2b/3 Safety Results (Masitinib and Placebo)

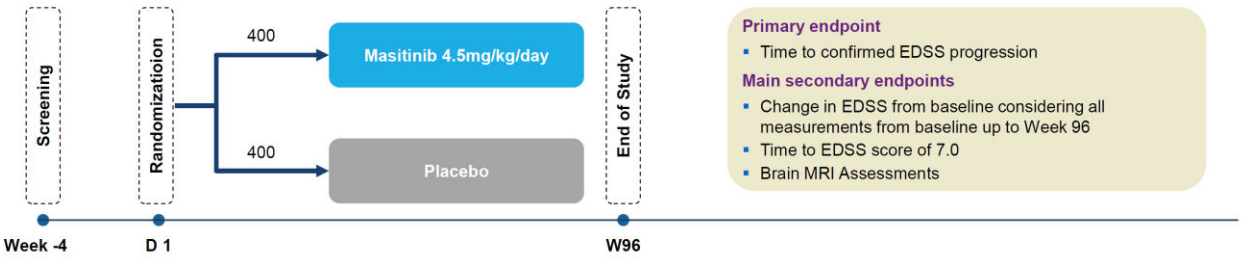
Patients with ≥1 event	MAS (n=199)	PBO (n=101)	Δ[M–P] (%)
Rash Maculo-Papular	1.5% (3)	0% (0)	1.5%
Erythema Multiforme	1.0% (2)	0% (0)	1.0%
GGT Increased	1.0% (2)	0% (0)	1.0%
Neutropenia	1.0% (2)	0% (0)	1.0%
PP Erythrodysesthesia	1.0% (2)	0% (0)	1.0%
Urinary Tract Infection	1.0% (2)	1.0% (1)	0%
MS Relapse	2.0% (4)	3.0% (3)	-1.0%

Source: Company reports

Proposed Masitinib Phase 3 Trial Design in Multiple Sclerosis. The Phase 3 study will be a multicenter (67 sites), double-blind, randomized, placebo-controlled trial enrolling 800 patients. The trial will evaluate masitinib 4.5mg against placebo. Patients will be randomized 1:1 to masitinib or placebo. The trial will be 96 weeks long. The primary endpoint will be time to confirmed EDSS progression. The secondary endpoints will include: 1) change in EDSS from baseline, considering all measurements from baseline up to week 96; 2) time to EDSS score of 7.0; and 3) brain MRI assessments.

The study will enroll primary progressive MS patients and exclusively secondary progressive MS patients without superimposed inflammation documented by MRI at baseline in order to exclude inflammatory brain lesions at inclusion. Patients will be required to have onset of symptoms at least five years prior to inclusion. The study design has been confirmed by the FDA and EMA.

Exhibit 49. Phase 3 Study Design



Main inclusion criteria:

The study will enroll PPMS and exclusively SPMS patients without superimposed inflammation documented by MRI at baseline in order to exclude inflammatory brain lesions at inclusion

- PPMS and nSPMS, stratified
- Onset of symptoms at least five years before inclusion
- No relapse diagnosed at least two years before inclusion (according to the 2017 revised McDonald's criteria)
- EDSS score progression ≥1 point with no improvement during 2 years before screening
- **Absence of T1 Gadolinium-enhancing brain lesions as measured by MRI**
- EDSS score of [3.0 to 6.0] inclusive

Study Status

Confirmatory phase 3 study design has been discussed with FDA and EMA

Approved countries

- Bulgaria, France, Germany, Greece, Italy, Netherlands, Poland, Norway, Portugal, Spain, Sweden, United Kingdom
- USA

Approved sites: 67

Source: Company reports

Planned interim analysis. The Phase 3 study will include an interim analysis to evaluate whether the sample size needs to be expanded to 1,200 patients.

The interim analysis could occur at the earlier of:

1. 50% of patients randomized who have reached week 96, or
2. 50% information fraction of the planned number of 12-week CDP events.

The sample size will be increased only if the study is expected to succeed with the SSR (option 3 and 4) below.

1. If Conditional Power (CP) ≤20% with no SSR and <40% with SSR, futility will be considered.
2. If CP ≥80%, there will be no SSR.
3. If CP <80% and CP ≥80% with SSR, then SSR will be implemented with a maximum 100% increase in sample size to reach a CP of 80%.
4. If CP <80% and maximum SSR (=100%) gives ≥60% and <80%, maximum SSR (+100%) will be implemented.
5. No SSR in all other scenarios.

Masitinib for Mast Cell Diseases

Due to masitinib's MOA, AB Science is also evaluating the development of masitinib for treating mast cell diseases. We believe that, due to the current capital situation and priorities, the development of masitinib for neurodegenerative disease is the priority, and we consider any development and data in mast cell disease to be upside.

Mast Cell Activation Syndrome. Mast cell activation syndrome (MCAS) is a condition in which mast cells over-release chemical mediators. Patients with MCAS experience severe symptoms, including low blood pressure, nasal congestion, abdominal pain, fainting, swelling, diarrhea, vomiting, flushing, hives, and itching. In some cases, MCAS can cause anaphylaxis. Unlike allergies that occur due to a specific exposure, MCAS episodes can occur without a clear trigger. For a condition to be considered MCAS, ≥2 body systems must be affected, which include: 1) the skin, 2) respiratory system, 3) cardiovascular system, and 4) gastrointestinal tract. The current treatment paradigm is to treat the symptoms of MCAS instead of MCAS itself. There are multiple therapies used to treat allergic symptoms, pain, and anaphylactic episodes.[17] [18]

While mast cells produce many mediators, only a few mediators or their stable metabolites are reliably found in laboratory tests. Patients have been found with increases in serum mast cell tryptase and in urine levels of N-methylhistamine, 11B -prostaglandin F2α (11B-PGF2α) and/or leukotriene E4 (LTE4), which can be used to diagnose MCAS.

Ongoing Phase 3 trial. AB Science has initiated a Phase 3 trial for masitinib for treating MCAS. The company has completed the first study but has paused the trial for the second study. AB Science is currently prioritizing capital and resources for the development of masitinib for treating ALS and neurodegenerative diseases and is thus not currently focused on MCAS. When considering masitinib's MOA, we believe there is a chance for success in this study. The data generated in neurodegenerative disease also support masitinib's MOA and potential efficacy in treating mast cell-associated diseases. That being said, we consider this program upside.

Indolent Systemic Mastocytosis (ISM). AB Science is also evaluating the development of masitinib for treating indolent systemic mastocytosis (ISM), since it is a mast cell disorder. ISM is a rare disease that leads to the production and accumulation of abnormal mast cells. Accumulation of abnormal mast cells can lead to symptoms similar to severe allergic reactions. In ~95% of patients, ISM is believed to be caused by a gene mutation called KIT D816V.The most common symptoms of ISM include skin lesions and rash, severe allergic reactions, including anaphylaxis and gastrointestinal symptoms such as nausea, vomiting, and diarrhea. Systemic mastocytosis (SM) is estimated to have a prevalence of around one in 10,000 people. The most common form of SM is ISM, which accounts for around 75%–90% of cases of SM. ISM can progress to advanced systemic mastocytosis (AdvSM), which occurs in approximately 3%–4% of patients.[19] Similar to MCAS development, we believe this program is upside to AB Science and do not factor it into our valuation. Considering masitinib's MOA, we believe there is a positive probability of success in this program.

[17] https://my.clevelandclinic.org/health/diseases/mast-cell-activation-syndrome
[18] https://www.aaaai.org/conditions-treatments/related-conditions/mcas
[19] https://ayvakit.com/indolent-systemic-mastocytosis/about-ism/?gad_source=1&gad_campaignid=21464391777&gbraid=0AAAAAoMfFAtMMBEqkgEllLekgLJyzAnRX&gclid=EAIaIQobChMIj-v3_KCLkQMVOkf_AR3yXSF1EAAYASAAEgIqHPD_BwE&gclsrc=aw.ds

Sickle Cell Disease

AB Science is also evaluating the development of masitinib for treating sickle cell disease (SCD). We believe that, due to the current capital situation and priorities, the development of masitinib for neurodegenerative disease is the priority, and we consider any development and data in sickle cell disease to be upside.

Sickle cell disease (SCD), also known as sickle cell anemia, is a group of inherited red blood cell disorders that affect hemoglobin, the major protein that carries oxygen in red blood cells. Normal red blood cells are typically disc-shaped, flexible, and can move easily through blood vessels to carry oxygen throughout the body. In SCD, due to a problem with hemoglobin, the cells become stiff and take on a crescent or "sickle" shape. These sickled cells are not flexible, cannot change shape easily, and tend to stick to blood vessel walls, causing blockages. This slows or stops blood flow, which prevents oxygen from reaching tissues. Sickle cells also break apart and die quickly (10–20 days instead of the normal 90–120 days), leading to a shortage of red blood cells.[20]

SCD is associated with significant serious symptoms. SCD is associated with vaso-occlusive crises (VOCs), which are periodic episodes of sudden, severe pain that occur when sickled cells block blood flow to the chest, abdomen, joints, or other body parts. SCD can cause anemia (shortage of red blood cells), leading to fatigue, dizziness, and sometimes a yellowish color of the skin or eyes (jaundice); swelling of the hands and feet (dactylitis) due to blocked circulation; and damage to the spleen, which can lead to frequent infections, increasing the risk of serious bacterial infections like pneumonia. SCD is also associated with the risk of acute chest syndrome, which is a life-threatening complication that involves chest pain, fever, and difficulty breathing, often caused by sickling in the lung blood vessels. In addition, patients are at increased risk of stroke if sickled cells block blood flow to the brain, requiring immediate care.

Epidemiology. SCD affects > 100,000 people in the U.S. and 8M people worldwide. In the U.S., 9 out of 10 people who have sickle cell disease are of African ancestry or identify as Black. About 1 in 13 Black babies are born with the sickle cell trait. About 1 in every 365 Black babies is born with sickle cell disease.

Current treatment paradigm. Casgevy (exagamglogene autotemcel) is an autologous, one-time, CRISPR/Cas9-edited cellular gene therapy available to treat SCD patients with severe VOCs. Additional therapies are used to manage the symptoms of SCD. Hydroxyurea is an oral therapy that can reduce the sickling of red blood cells and help prevent pain crises and acute chest syndrome. It is often prescribed for infants as young as nine months old. L-glutamine (Endari) is an oral powder approved for people ages five and older and is used to reduce the frequency of pain crises. Adakveo (crizanlizumab) is an intravenous (IV) therapy for people ages 16 and older that helps prevent blood cells from sticking to vessel walls, reducing pain crises. SCD patients may receive penicillin to lower the risk of life-threatening infections. SCD patients may also receive analgesics, including OTC analgesics, to manage acute and chronic pain.

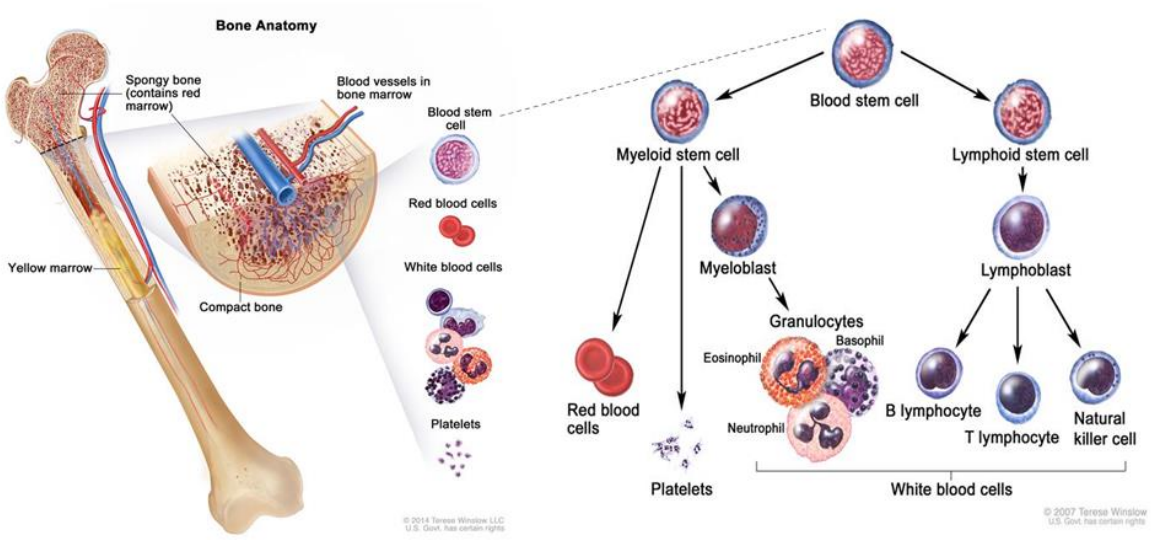
SCD patients may require blood transfusions to treat and prevent complications such as stroke and severe anemia by increasing the number of healthy red blood cells in the body. SCD patients may also receive bone marrow/stem cell transplants. This procedure can potentially cure SCD by replacing the affected bone marrow with healthy bone marrow from a matched donor (usually a sibling). This is typically reserved for children with severe symptoms due to the high risks.

[20] https://www.nhlbi.nih.gov/health/sickle-cell-disease

AB8939 (Microtubule Destabilizer and ALDH1/2 Inhibitor) for Acute Myeloid Leukemia (AML)

Acute myeloid leukemia (AML). AML is a rapidly progressing hematologic cancer. Specifically, AML is a heterogeneous disease characterized by malignancy of the myeloid lineage (precursors to other blood cells). Myeloblasts, which cease to differentiate appropriately, accumulate in the bone marrow, interfering with normal hematopoiesis (blood cell production). Immature, nonfunctional cells accumulate in the bone marrow, while normal blood cells (i.e., red blood cells and platelets) are displaced. Coincidentally, the marrow may also stop producing normal levels of platelets, red blood cells, and white blood cells. Anemia, a deficiency of red blood cells, develops in virtually anyone with leukemia. At the same time, the body's ability to fight infection becomes impaired due to the lack of normal white blood cells. In addition, the shortage of platelets results in bruising and bleeding. Although it is considered a rare disease, AML is the most common form of leukemia among adults, representing 90% of adult leukemia cases. SEER and the American Cancer Society estimate that ~22,000 individuals will be diagnosed with AML in 2025, accounting for 1.8% of all cancer deaths in the United States.[21]

AML: cancer of the myeloid lineage. AML is a very aggressive cancer of the blood and bone marrow. Normally, the bone marrow produces blood stem cells that can develop into either myeloid stem cells or lymphoid stem cells. In AML, the myeloid stem cells cannot differentiate appropriately and remain as immature white blood cells called myeloblasts (or myeloid blasts). Consequently, these myeloblasts, or leukemia cells, can accumulate in the blood and bone marrow, leaving less room for healthy white blood cells, red blood cells, and platelets, leading to infection, anemia, or bleeding.



Source: www.cancer.gov/types/leukemia.

The median age at diagnosis is 68 years, with ~75% of new AML cases occurring in patients aged 55 and older. The survival rate at five years is <10%–25 %, depending on age, and declines further with increasing age. The five-year survival rates by age group are: 62% (15–44), 42% (45–54), 25% (55–64), 10% (65–74), and 2% (75+).[22] AML is a highly heterogeneous disease that requires individualized cytogenetic and molecular characterization. From a broad perspective, the disease can be categorized into favorable, intermediate, or high-risk groups. At the time of diagnosis, standard karyotype analysis can be used in many patients to determine important and distinct chromosomal translocations, while large chromosome gains or losses can also be detected. Sequencing and other molecular approaches can be used to determine the presence of recurrent gene mutations that are undetectable by cytogenetic analysis. Many of these mutations have prognostic implications, independently and/or in the presence of other co-occurring driver mutations.[23] Mutation groups for which there have been successes in terms of targeted therapies gaining approval (at least in the relapsed/refractory setting, subsets of AML) include FLT3 and IDH1/IDH2, which belong to broader AML mutation categories of signaling/kinase pathways and epigenetic modifiers, respectively.

Treatment landscape and standard of care. Once an AML diagnosis is made, 1L therapeutic options need to be determined, with patients classified as either "fit" or "unfit" for intensive induction chemotherapy. Although age should not be the sole determinant of a patient's fitness for intensive therapy, it is widely used; in general, "fit" patients are typically <60 years of age.[24] However, multiple factors must be considered, and if an older patient is deemed medically fit, intensive chemotherapy is still recommended as a frontline approach to AML management. The

[21] *SEER Cancer Stat Facts: Acute Myeloid Leukemia. National Cancer Institute.*
[22] *American Cancer Society*
[23] *DiNardo and Cortes. Hematology. 2016;(1):348-355*
[24] *Percival & Estey. Clin Adv Hematol Oncol. 2017; 15(8);632-642*

standard of care for fit patients is induction chemotherapy with the 7+3 regimen of cytarabine and an anthracycline. The complete remission (CR) rates range from 50% to 75% in this setting. Induction therapy is followed by consolidation therapy to manage residual disease and prolong time to recurrence.[25,26] For unfit patients, the frontline standard of care in induction therapy is lower-intensity therapy, typically the azacitidine + venetoclax doublet. Venetoclax (Ven, a BCL-2 inhibitor) was first approved in November 2018 as a combination with azacitidine (Aza) in newly diagnosed AML. In the registration-enabling study, patients were over age 75 or had comorbidities that made them ineligible for intensive chemotherapy. The CR rate was 37%, and the composite CR + CR with incomplete hematological recovery (CRh) was 66%. In another study, the Ven + Aza doublet demonstrated improved overall survival vs. Aza alone in unfit patients.[27] However, although overall survival improved and CR rates were higher with Ven/Aza, it remains <15 months, and patients develop resistance.

Mutations and targeted therapies. Beyond traditional cytotoxic chemotherapy, therapies targeted to specific genetic lesions (e.g., FLT3, IDH1, or IDH2) have more recently gained regulatory approval, although they yield moderate overall responses and low complete response rates. Even with the treatment landscape shifting, therapy options remain limited for relapsed/refractory AML. The heterogeneity of AML suggests that single-target agents are unlikely to achieve cure rates for large numbers of patients. Therefore, combinations of novel agents and traditional chemotherapy are likely required to achieve durable responses. Several targeted therapies have been approved, as detailed below.

Mutation-specific targeted therapies

- **FLT3 inhibitors.** FLT3, or FMS-like tyrosine kinase 3, is mutated in ~30% of AML patients, resulting in constitutive activation and downstream signaling of the RAS/RAF/MEK growth and proliferation pathways, as well as the PI3K/AKT pro-survival pathways. The degree of mutational burden in FLT3 is also important. The activating mutations in the FLT3 gene lead to AML blasts expressing both high levels of the mutated protein and the WT FLT3 receptor, which are associated with poor clinical prognosis. There are three approved FLT3 inhibitors: midostaurin (Rydapt, 2017), gilteritinib (Xospata, 2018), and quizartinib (Vanflyta, 2023). Midostaurin was the first AML drug approved since 2000 and was cleared for FLT3-mutated AML in the newly diagnosed setting. Gilteritinib followed in 2018 for relapsed/refractory (r/r) AML. Quizartinib received a complete response letter (CRL) in 2020 for an indication in r/r AML but was approved in mid-2023 in combination with chemotherapy (cytarabine + anthracycline) in newly diagnosed AML as part of consolidation therapy or maintenance after consolidation therapy.[28,29,30]
- **IDH inhibitors.** Isocitrate dehydrogenase, or IDH1 and IDH2, are mutated in 8% and 12% of AML patients, respectively. These homodimeric enzymes catalyze the conversion of isocitrate to alpha-ketoglutarate, a key step in the tricarboxylic acid (TCA) cycle, also known as the Krebs cycle. The TCA/Krebs cycle is a central metabolic pathway that supports cellular bioenergetics. Mutations in IDH1 and IDH2 are associated with loss of function of the Krebs cycle and instead trigger a reverse reaction that generates the "oncometabolite" 2-hydroxyglutarate (2-HG), which competitively inhibits alpha-ketoglutarate-dependent enzymes. The key consequence is that this partially stops the normal maturation of blood cells. There are three approved IDH inhibitors: enasidenib (Idhifa, 2017), ivosidenib (Tibsovo, 2018), and olutasidenib (Rezlidhia, 2022). These agents are used in the r/r AML setting, though they can be used in 1L treatment in patients with IDH mutations who are unfit for intensive induction chemotherapy.

Non-mutation-specific targeted therapies

- **Myelotarg (gemtuzumab ozogamicin).** This is a monoclonal antibody targeting CD33, conjugated to a cytotoxic agent. It was first approved in 2017 for the treatment of newly diagnosed CD33-positive AML, as well as for relapsed/refractory (r/r) CD33-positive AML. Myelotarg can be used as monotherapy or in combination with other therapies.
- **Daurismo (hedgehog pathway inhibitor).** This hedgehog inhibitor was first approved in 2018 for the combination treatment of adults over the age of 75 with newly diagnosed AML. The drug works by inhibiting the transmembrane protein "smoothened" (Smo) in the hedgehog signaling pathway. This inhibition has been shown to impair cancer stem cell development and survival.
- **Venclexta (venetoclax).** This oral BCL-2 inhibitor (B-cell lymphoma 2) is indicated for several blood cancers. For AML, it is approved in combination with azacitidine or decitabine (hypomethylating agents, Aza or Dec), or low-dose cytarabine, for the treatment of newly diagnosed AML in adults aged 75 or older, or those who have comorbidities that preclude the use of intensive induction chemotherapy. The Ven + Aza combo is the standard of care for "unfit" patients, a population representing 50%–70% of AML patients. Venetoclax was granted accelerated approval in AML in 2018 and full approval in 2020.

AB8939 – Next-Generation Synthetic Microtubule Destabilizer

AB Science is developing AB8939 to treat AML. AB8939 is a novel, small-molecule, synthetic microtubule destabilizer and targeted stem cell ALDH1/2 inhibitor. AB8939 can circumvent P-glycoprotein (Pgp) and myeloperoxidase (MPO)-mediated resistance. Microtubules are important in cell division, and microtubule-targeting chemotherapies are gold standards in many cancers. Resistance is commonly seen with currently approved tubulin inhibitors, which are degraded by myeloperoxidase (produced by cancer cells) in AML.

[25] *Cortez & Mehta. Am J Hematol. 2021; 96(4):493-507*
[26] *StatPearls, Acute Myeloid Leukemia*
[27] *Zeidan et al. Blood. 2021; 138:277-279*
[28] *Cortez J. J Hematol Oncol. 2024; 111(17):OA*
[29] *Negotei et al. J Clin Med. 2023; 12(20):6429*
[30] *FDA.gov/drugs*

ALDH is also important to cancer stem cells, as it has been found to contribute to therapy resistance. MDS1 and EVI1 complex locus (MECOM) rearrangements in AML have been found to have an over-expression of ALDH. By inhibiting ALDH1, AB8939 can help repopulate the bone marrow with normal progenitors. AB8939 can also potentially treat refractory/relapsed AML and has been found to have low hematological toxicity.

For patients who cannot receive intensive salvage chemotherapy, treatment options are limited, with complete response rates around 20% and a median overall survival of ~eight months. AB8939 has been found to be >100x more potent than doxorubicin (Adriamycin). In addition, AB8939 is not deactivated by the myeloperoxidase enzyme, which is commonly seen in currently available vinca alkaloids (vincristine or vinblastine). AB Science is developing AB8939 for AML patients receiving 2L or 3L treatments who are unfit to receive intensive chemotherapy. AB8939 has received orphan drug designation (ODD) from the FDA.

Exhibit 50. Current Treatment Landscape

		Relapsed / refractory AML (R/R AML) *	
	Line 1	Line 2	Line 3
Patients eligible to high dose chemotherapy	Anthracyclines + cytarabine + targeted therapies (IDH/FLT3)	High dose chemotherapy or Low dose chemotherapy	No approved drug Low dose chemotherapy or Best supportive care
Patients ineligible to high dose chemotherapy	Hypomethylating agents (azacitidine / venetoclax) + targeted therapies (IDH/FLT3)	No approved drug Low dose chemotherapy or Best supportive care	No approved drug Best supportive care

AB8939 current positioning in AML

* One Menin inhibitors recently registered for R/R AML patients with KMT2Ar rearrangement or NPM1 mutation

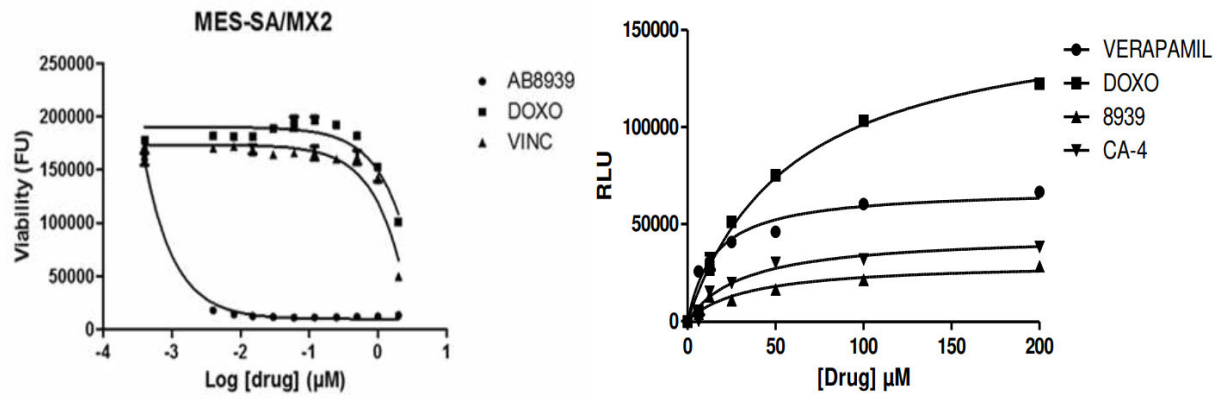
Anthracyclines = Daunorubicin or Idarubicin
Cytarabine = Ara C
Azacitidine = Vidaza
Venetoclax = Venclyxto

Source: Company reports

Positive Data Generated Preclinically

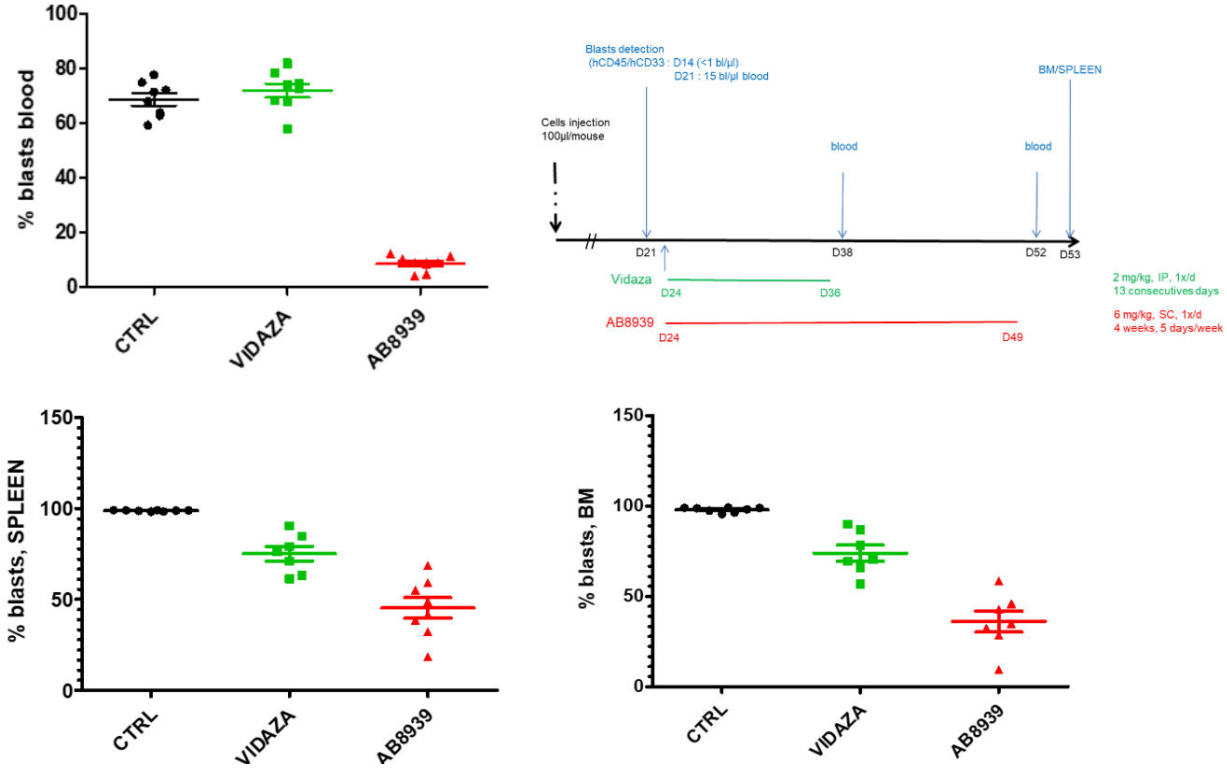
AB8939 was evaluated in cytarabine (Ara-C)-resistant and azacitidine (Vidaza)-resistant patient-derived xenograft (PDX) models. Ara-C is considered the most clinically relevant cytotoxic agent for AML, and azacitidine is also widely used in AML. AB8939 was found to substantially decrease the concentration of blasts in blood (38 days post-graft), bone marrow (BM), and spleen (52 days post-graft) vs. azacitidine.

Exhibit 51. AB8939 was found to overcome multidrug resistance. AB8939 blocked proliferation of the Pgp-overexpressing, drug-resistant MES-SA/MX2 cell line in a six-day proliferation/survival assay.



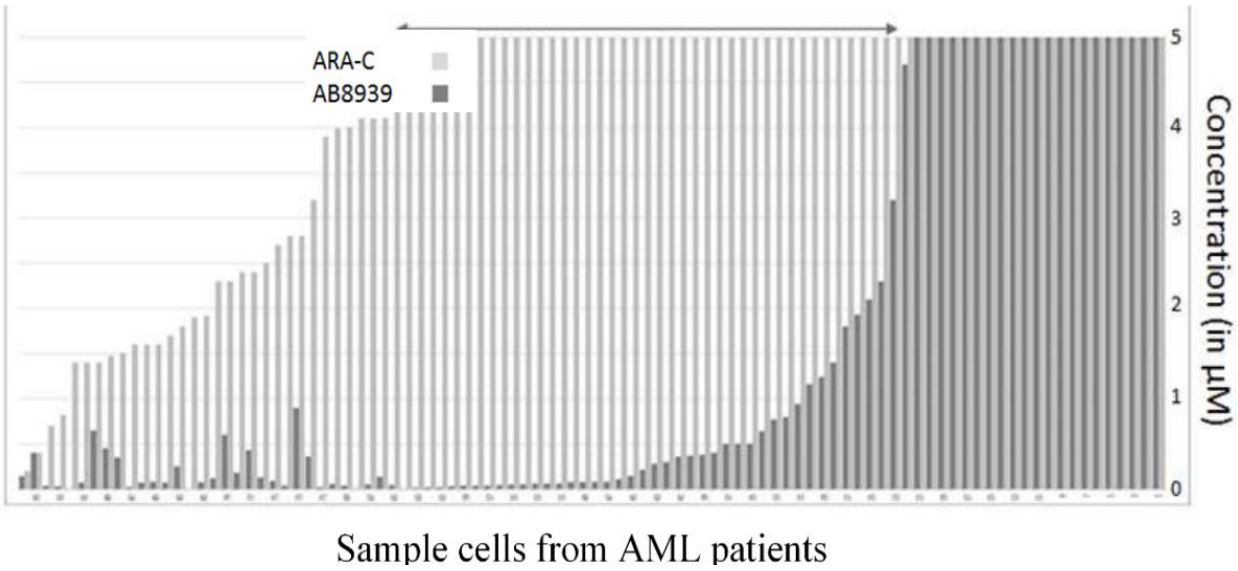
Source: Company reports

Exhibit 52. AB8939 substantially decreased the concentration of blasts in blood (38 days post-graft), bone marrow (BM), and spleen (52 days post-graft) relative to azacitidine.



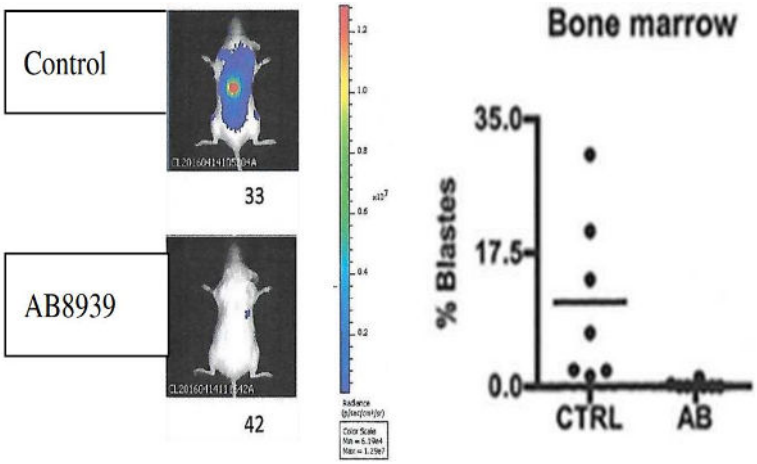
Source: Company reports

Exhibit 53. AB8939 was found to overcome Ara-C resistance. Sixty-six percent of Ara-C-resistant blasts were sensitive to AB8939, and 69% of blasts exhibited nanomolar sensitivity (IC50 ≤ 500nM). Forty-four percent of blasts were very sensitive (IC50 ≤ 100nM). AB8939 also demonstrated broad anti-proliferative activity across AML subtypes.



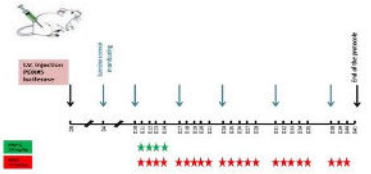
Source: Company reports

Exhibit 54. AB8939 found to eliminate blasts from the marrow in an AMKL26 PDX model. AB8939 was also well tolerated, with no toxicity-related deaths and no effects on weight or behavior.

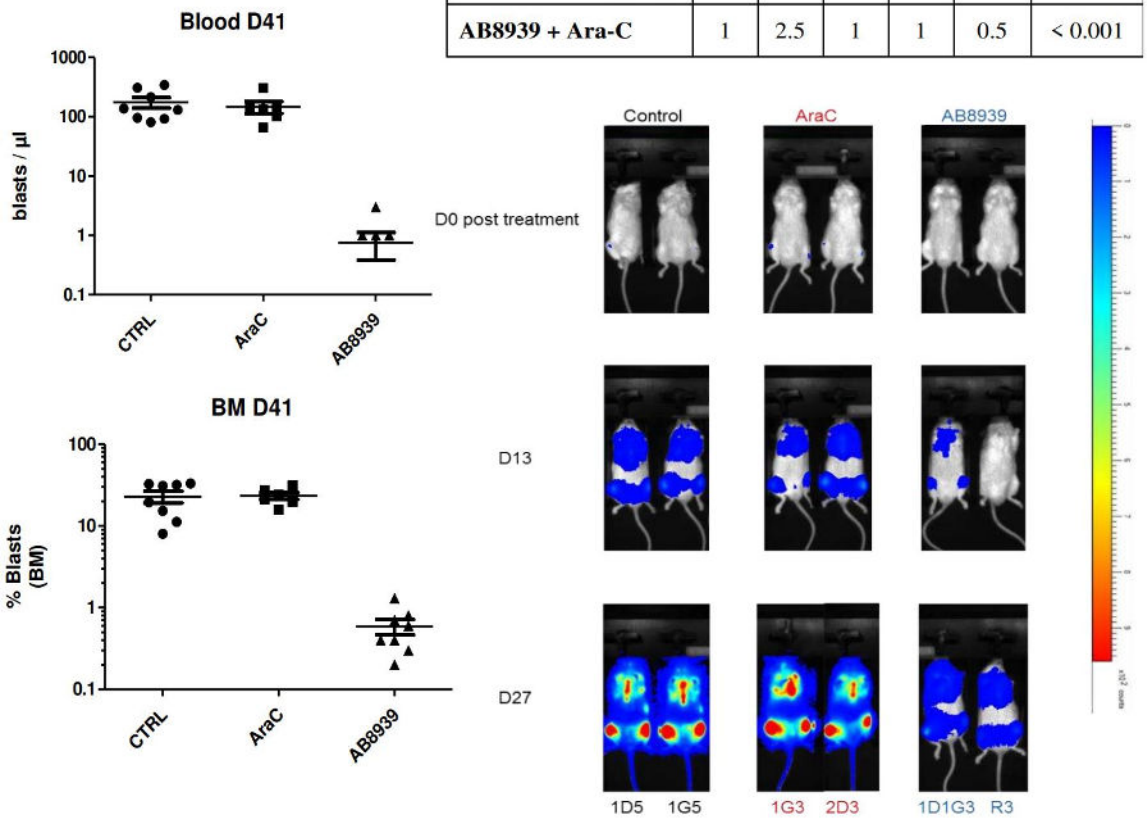


Source: Company reports

Exhibit 55. AB8939 was found to significantly decrease tumor growth and the concentration of blasts in blood and bone marrow.



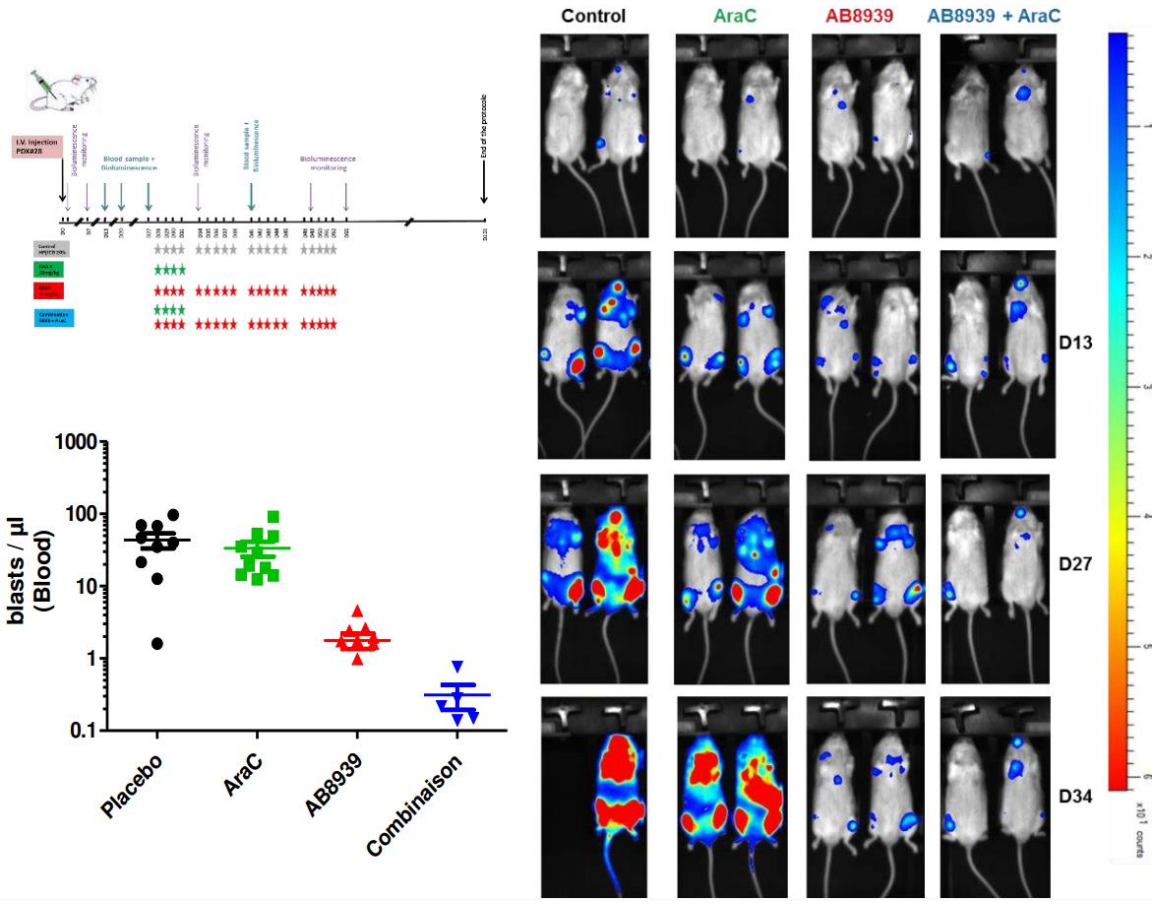
Bioluminescence (days post graft)	D27	D34	D41	D49	D55	P value*
Control	1	9	44	113	133.5	-
Ara-C 10 mg/kg (bid)	1	5	17	73	64	NS
AB8939 6 mg/kg/day	1	3	3.5	6	6	< 0.001
AB8939 + Ara-C	1	2.5	1	1	0.5	< 0.001



Source: Company reports

Exhibit 56. AB8939 was found to significantly decrease disease burden (day 55 post-graft; day 27 treatment)

Bioluminescence (days post graft)	D10	D17	D24	D31	D38	P value*
Control	1	10.8	66.6	373.9	2191.6	-
Ara-C 10 mg/kg (bid)	1	11.6	49.6	659.5	2144.6	NS
AB8939 6 mg/kg/day	1	10.6	19.8	50.6	151.3	< 0.001



Source: Company reports

Positive Responses Seen in MECOM-Rearranged and Non-MECOM AML Patients in Phase 1 Study

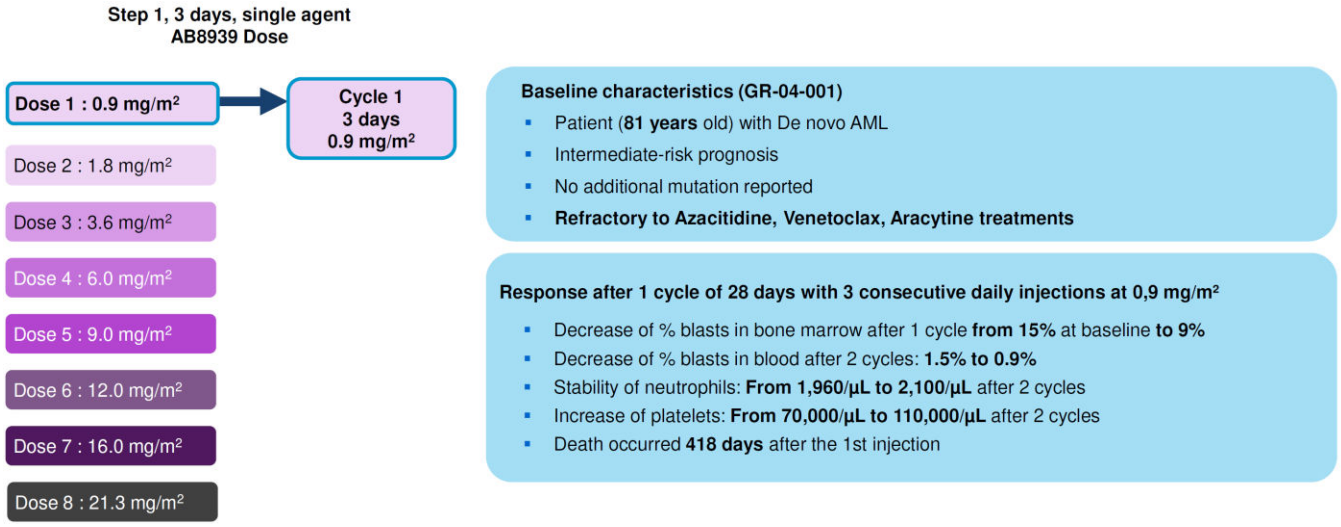
AB8939 was evaluated in a Phase 1 study to determine the maximum tolerated dose (MTD) over three different cycles. The Phase 1 trial enrolled 28 patients. Patients initially received one of nine doses for three days. The first step of the study determined the maximally tolerated dose (MTD), which was 21.3mg/m^2. The second part of the study evaluated two doses for 14 days: 16.0mg/m^2 and 21.3mg/m^2. The 16.0mg/m^2 cohort enrolled seven patients, with one dose-limiting toxicity (DLT) observed. Following the second part of Phase 1, AB Science plans to evaluate combinations of: 1) AB8939 + Venetoclax or Azacitidine, and 2) AB8939 + venetoclax + azacitidine (all three combined).

Positive responses seen in two non-MECOM patients. In the lowest-dose cohort, a non-MECOM refractory AML patient with intermediate-risk prognosis showed stable disease after at least 108 days. The patient was refractory to azacitidine, venetoclax, and aracytine.

Following treatment, the patient showed several positive responses and stability in their hematological profile. The percentage of blasts in the bone marrow decreased significantly, dropping from 15% at baseline to 9% after one treatment cycle. A similar trend was observed in the peripheral blood, where the percentage of blasts decreased from 1.5% to 0.9% after two cycles. Additionally, stability was maintained in the

neutrophil count, which slightly increased from 1,960/uL at baseline to 2,100/uL after two cycles. Platelet counts also improved, increasing from 70,000/uL to 110,000/uL after two cycles. The patient died 418 days after the first injection.

Exhibit 57. Non-MECOM Patient Responded to Monotherapy

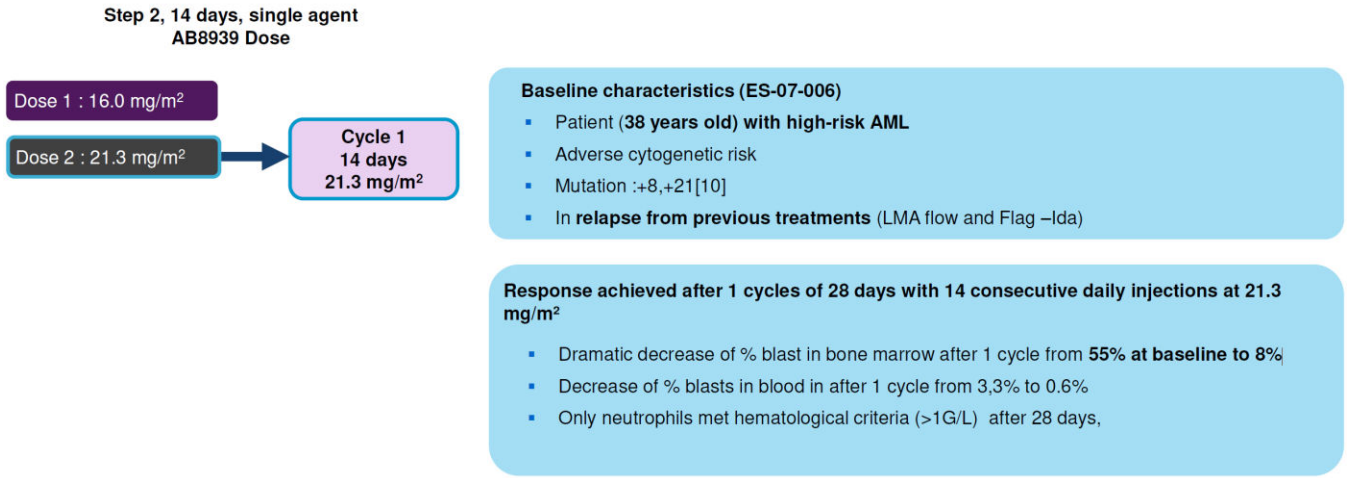


Source: Company reports

A second non-MECOM patient demonstrated a response at the 21.3mg/m^2 dose. The patient was a 38-year-old with high-risk AML and in relapse from previous treatments (LMA flow and Flag-IDA).

The patient achieved a response after one 28-day cycle, consisting of 14 consecutive daily injections at 21.3mg/m². The patient experienced a significant decrease in the percentage of blasts in the bone marrow, falling from 55% at baseline to 8%. Similarly, the percentage of blasts in the blood decreased significantly, from 3.3% to 0.6%. However, only the neutrophils met the required hematological criteria, defined as >1G/L, after the 28-day period.

Exhibit 58. Second Non-MECOM Patient Responded to Monotherapy

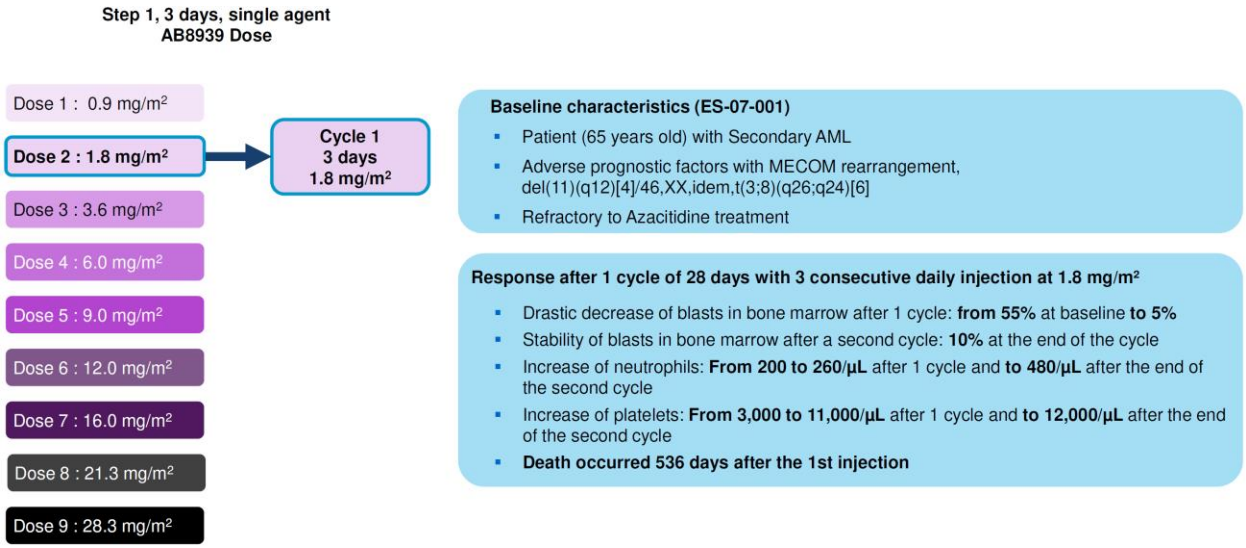


Source: Company reports

AB8939 also demonstrated positive benefits in MECOM patients

AB8939 at 1.8mg/m^2 demonstrated a response in a refractory AML patient with MECOM rearrangement. The patient was 65 years old, with secondary AML, and was refractory to azacitidine treatment. The patient showed a response after one 28-day cycle, consisting of three consecutive daily injections. The blasts in the bone marrow decreased from 55% at baseline to 5%. Following the second cycle, the bone marrow blast percentage remained relatively stable at 10%. Concurrently, the neutrophil count improved, increasing from 200/µL at baseline to 260/µL after the first cycle and reaching 480/µL after the second cycle. Platelets also increased significantly, rising from 3,000/µL at baseline to 11,000/µL after the first cycle and reaching 12,000/µL after the second cycle. The patient died 536 days after the first injection.

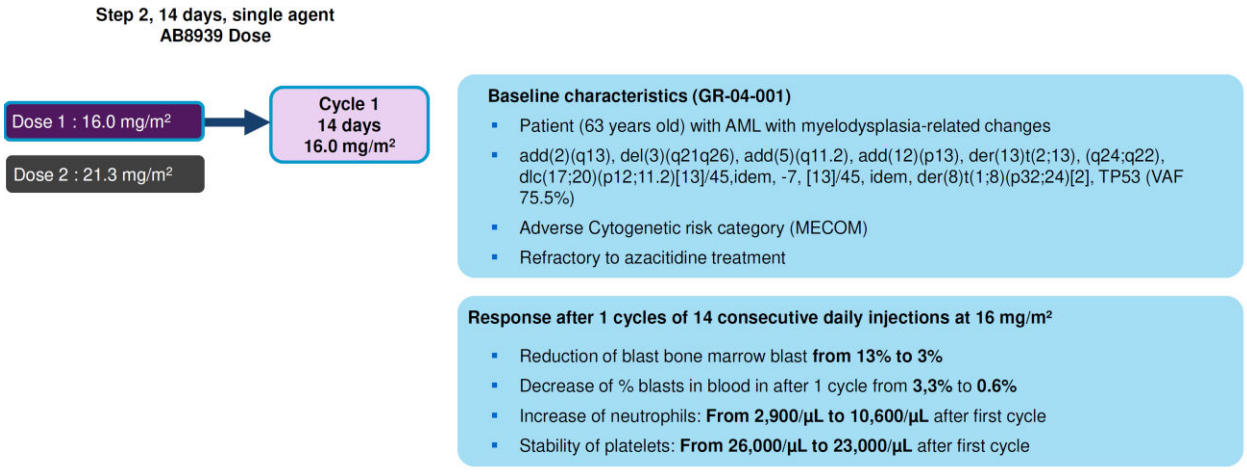
Exhibit 59. First MECOM Patient Response



Source: Company reports

AB8939 16.0mg/m^2 demonstrated a response in an AML patient with myelodysplasia-related changes and MECOM rearrangement after 14 days. The patient was 63 years old and refractory to azacitidine treatment. The patient responded after one cycle of 14 consecutive daily injections at 16mg/m². The blasts in the bone marrow decreased from 13% at baseline to 3%. The percentage of blasts in the blood also decreased after one cycle, from 3.3% to 0.6%. Concurrently, the neutrophil count improved, increasing from 2,900/µL at baseline to 10,600/µL after one cycle. Platelet counts also increased, rising from 23,000/µL at baseline to 26,000/µL after the first cycle.

Exhibit 60. Second MECOM Patient Response



Source: Company reports

Exhibit 61. AB8939 has overall shown a positive trend in MECOM-rearranged tumors in both in vitro and in-human studies with two out of four patients responding in Phase 1.

Non clinical *in vitro* evidence

- **50% response rate in *in-vitro* tests**

In-vitro, AB8939 was effective (IC50 of 50nM and 13nM) against 2 out of 4 patient blasts with MECOM rearrangement

Drug sensitivity (IC50 µM) in MECOM Karyotype				
Patient ID	AML type	AraC	AB8939	Azacitidine
1135	M0	>20	>2	49,90
1156	M0	>20	>5	>50
C1005	M1 refractory	4,1	0,05	NT
C1012	M4 refractory	7,9	0,013	9,7

Clinical evidence in MECOM

- **50% response rate in early phase 1**

2 out of 4 patients with MECOM after 1 cycle of 3 days or 14 days AB8939 treatment below the MTD

Patient ID	AB8939	Best Response
ES-12-001	0,9 mg/m^2, 3 days	Early discontinuation
ES-07-001	1.8 mg/m^2, 3 days,	Response (BM blast from 55% to 5%)
ES-07-002	16 mg/m^2, 14 days	Stable disease
GR-04-001	16 mg/m^2,14 days	Response (BM blast from 13% to 3%)

Source: Company reports

Update as of October 16, 2025. AB Science has completed Stages 1 and 2 of the Phase 1 program. Stage 1 evaluated 28 patients, and Stage 2 evaluated 13 patients. Thus far, in MECOM-rearranged patients, AB8939 has shown a disease control rate of 100% (3/3) and the partial response rate is 100% (3/3), including one patient in complete remission. These results were after the first cycle of treatment (14 days of treatment) in patients receiving third- or fourth-line treatment, 2 of whom had previously progressed on venetoclax in combination with other chemotherapies.

AB Science has initiated Stage 3 of the Phase 1 study, which will treat patients with a combination treatment of AB8939 + venetoclax. The full dataset from the Phase 1 study is expected in mid-2026. Following Stage 3, AB Science may initiate a Stage 4 extension trial with 15 patients, in which it treats patients with an AB8939 + venetoclax + azacitidine triple therapy.

Phase 2 Study Design. AB Science is evaluating the development of AB8939 for the treatment of MECOM AML patients. The company could potentially seek accelerated approval through a well-designed Phase 2b/3 trial. The Phase 2b/3 could be a multicenter, single-arm trial that enrolls fewer than 60 patients. The study would evaluate AB8939 as a monotherapy or in combination with venetoclax and/or azacitidine. AB Science is targeting a response rate of 14% based on established literature for MECOM patients. AB Science may also potentially evaluate relapsed or refractory AML as a monotherapy or combination therapy, with a target response rate of 17.5%. AB Science may also evaluate 1L AML patients in combination with venetoclax and/or azacitidine, targeting a 37% response rate. The EMA requires AB Science to start with 2L or 3L patients, but the Stage 4 extension trial from the Phase 1 may provide some insight into 1L treatment.

The three pathways for pivotal studies, while not mutually exclusive, include:

- AB8939 + venetoclax in first line of treatment, with aged patients and/or adverse genetics (tp53mut + nras + kras + complex k + monosomy 5/7 + mecom)
- AB8939 + venetoclax in second- or third-line of treatment, all patients or adverse genetics
- AB8939 in MECOM in second- or third-line of treatment

MECOM-rearranged AML patients account for 5% of AML patients and are estimated to represent a >€100M market. Relapsed/refractory MECOM-rearranged patients have an incidence rate of 90,200 annually.

Exhibit 62. MECOM AML Patients Represent a ≥€100M Opportunity

Region	Incidence Case (1)	Market Size (per in in Mio EUR)
R/R AML	90,200	2 200 000
AML with MECOM gene rearrangement		5%
		≈ 100

Source: Company reports

Prior developments support a small-study regulatory pathway. Revumenib, which is indicated for treating AML with KMT2Ar rearrangements or NPM1 mutations, was approved based on only a Phase 1/2 study that evaluated 95 adult patients, of whom only 57 were evaluable. The study showed a complete or near-complete response rate of 23%. Ziftomenib is another menin inhibitor in development and has completed a Phase 1 study with a small number of patients.

MECOM-rearranged AML patients account for 5% of AML patients and are estimated to represent a >€100M market. Relapsed/refractory MECOM-rearranged patients have an incidence rate of 90,200 annually.

Masivet

AB Science commercializes Masivet in Europe. Masivet is a veterinary product that targets mast cell tumors (MCTs), the most common cutaneous malignancies in dogs. Dogs with MCT have a poor prognosis. Masivet was initially approved in 2008 by the EMA to treat non-resectable MCTs (grade 2 or 3) in dogs with a mutated form of the receptor protein c-Kit. The product is not a strategic priority and generates <€2M annually. It is profitable, helping cover some expenses and mitigating AB Science's cash needs. AB Science is evaluating whether to develop the product in the U.S. The study would cost <$2M, and the drug could potentially be launched within three years from study onset. It is already known the product works; therefore, the study is expected to be successful. A study can be conducted with <100 dogs. An approval in the U.S. could generate $5M–$7M annually. This pathway is being evaluated, but the strategic priority remains masitinib for ALS.

Investment Risks

- **Investment risk:** AB Science's pipeline products are not yet approved. Additionally, the company may not generate sufficient revenue to fund operations even after product approvals.
- **Revenue risk:** While none of AB Science's pipeline products are currently approved, even if they are approved and commercialized, future revenue may be immaterial or could decline due to various factors. Insufficient revenue could impair AB Science's ability to fund its operations.
- **Development risk:** AB Science's potential pipeline products may fail in clinical studies or progress more slowly than expected. The company may also divest or cancel pipeline programs due to evolving development or commercial risks. Additionally, the company may fail to expand its pipeline in a manner satisfactory to investors.
- **Regulatory risk:** AB Science's clinical-stage products may not be successful in clinical trials and may not meet the requirements for regulatory approval(s). Regulatory agencies may determine that trial results are insufficient for approval, even if statistically significant, due to safety concerns or lack of clinical meaningfulness.
- **Commercial risk:** AB Science's pipeline products are not approved or commercialized, and if/when they become commercially available, they may not achieve meaningful market share. The company may also fail to obtain sufficient reimbursement from payers, limiting market adoption.
- **Reimbursement risk:** The company may fail to obtain sufficient reimbursement from managed care agencies to pay for new products, and reimbursement may decline for existing products. Managed care companies may impose restrictions on reimbursement that could cause healthcare practitioners to be less willing to prescribe products, thereby impacting market adoption and sales.
- **Patent risk:** AB Science may eventually lose patent protection for its products. Loss of patents may lead to generic entrants, resulting in market share erosion, lower sales, and pricing pressure.
- **Pricing risk:** Pricing pressure from generics and new market entrants may force AB Science to reduce net prices or offer higher rebates, adversely impacting profitability.
- **Competitive technology risk:** While AB Science has innovative products, competitors may develop products using newer technologies that may gain patient, physician, and payer support, displacing AB Science's products.
- **Manufacturing risk:** AB Science is dependent on third-party contract manufacturing organizations. These partners may fail to deliver sufficient quantities, meet quality standards, or do so at acceptable costs, which could impact company revenue or margins.
- **Supply chain risk:** Domestic or global supply chains may negatively impact AB Science's ability to source materials necessary for manufacturing.
- **Financial risk:** The company may experience market share loss and declining sales, which could impair its ability to cover operating expenses.
- **Foreign exchange risk:** While AB Science is domiciled in France and reports results in euros (€), the company's clinical trials and operations are conducted internationally, exposing it to currency fluctuations. Ineffective or insufficient hedging could reduce revenue, increase costs, and adversely affect the company's operations.
- **Dilution risk:** The company may require additional capital raises to fund operations, and equity-based offerings may dilute existing investors.
- **Credit Risk:** AB Science has debt on its balance sheet. The company may be unable or unwilling to meet interest or principal payments, which could result in default, asset seizure, downgraded ratings, reduced borrowing capacity, or bankruptcy.

Valuation

We forecast masitinib launching in 2030 for the treatment of amyotrophic lateral sclerosis (ALS). We do not model the rest of the pipeline or other indications and assume them to be upside. We apply a 60% sales risk adjustment to masitinib for ALS based on the stage of development, regulatory risk, and commercial risks. A 30% discount is applied to the free cash flow, discounted EPS, and sum-of-the-parts models, which are equally weighted to derive a 12-month price target of €4.

Exhibit 63.Free Cash Flow Model

DCF Valuation Using FCF (mln):

	2024	2025	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035
units ('000)	2024A	2025E	2026E	2027E	2028E	2029E	2030E	2031E	2032E	2033E	2034E	2035E
EBIT	(6,083)	(5,595)	(6,327)	(7,367)	(14,598)	(11,694)	38,037	101,054	181,897	269,372	363,931	466,054
Tax Rate	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	15%	15%
EBIT(1-t)	(6,083)	(5,595)	(6,327)	(7,367)	(14,598)	(11,694)	38,037	101,054	181,897	269,372	311,161	398,476
CapEx	(155)	(139)	-	-	-	-	-	-	-	-	-	-
Depreciation	667	326	-	-	-	-	-	-	-	-	-	-
Change in NWC												
FCF	(5,571)	(5,408)	(6,327)	(7,367)	(14,598)	(11,694)	38,037	101,054	181,897	269,372	311,161	398,476
PV of FCF	(9,343)	(7,003)	(6,327)	(5,689)	(8,705)	(5,384)	13,525	27,746	38,566	44,103	39,339	38,902
Discount Rate	30%											
Long Term Growth Rate	1%											
Terminal Cash Flow	1,443,817											
Terminal Value YE2035	140,956											
NPV	317,033											
NPV-Debt	15,800											
Shares out (thousands)	83,355	2035e										
NPV Per Share (EUR)	€4											

Source: Maxim Group estimates

Exhibit 64.Discounted-EPS Model

Current Year	2025
Year of EPS	2035
Earnings Multiple	17
Discount Factor	30%
Selected Year EPS	€3.37
NPV (EUR)	€4

Source: Maxim Group estimates

	Discount Rate and Earnings Multiple Varies, Year is Constant					
	2035 EPS					
Earnings Multiple	5%	10%	15%	20%	25%	30%
5	€10	€6	€4	€3	€2	€1
10	€21	€13	€8	€5	€4	€2
15	€31	€19	€12	€8	€5	€4
20	€41	€26	€17	€11	€7	€5
25	€52	€32	€21	€14	€9	€6
30	€62	€39	€25	€16	€11	€7
35	€72	€45	€29	€19	€13	€9
40	€83	€52	€33	€22	€14	€10

Exhibit 65.Sum-of-the-Parts Model

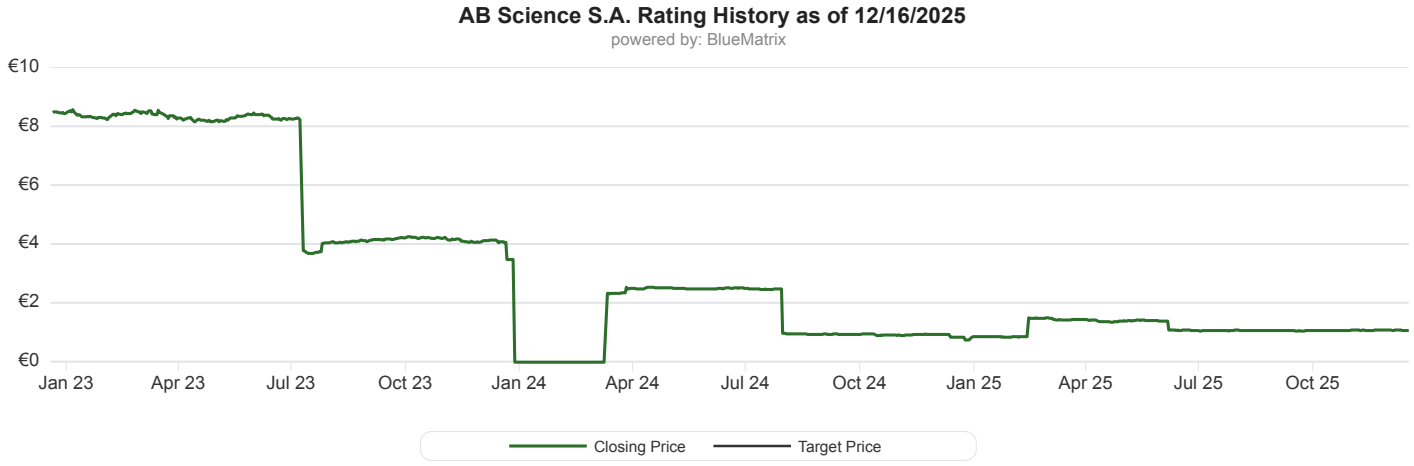
AB Science	LT Gr	Discount Rate	Yrs. to Mkt	% Success	Peak Sales MM's	Term Val
Masitinib for Amyotrophic Lateral Sclerosis (ALS)	1%	30%	4	40%	€532	€1,899
NPV						€2.82
Platform Value + Potential Milestones	1%	30%	4	40%	€50	€179
NPV						€0.26
Net Margin						75%
MM Shrs OS (2035E)						83
Total (€EUR)						€3

Source: Maxim Group estimates

ABS.PA: Income Statement (€000) EUR	2023	Jun-24	Dec-24	2024	Jun-25	Dec-25	2025	Jun-26	Dec-26	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035
ABS: YE December	**2023A**	**1H24A**	**2H24A**	**2024A**	**1H25A**	**2H25E**	**2025E**	**1H26E**	**2H26E**	**2026E**	**2027E**	**2028E**	**2029E**	**2030E**	**2031E**	**2032E**	**2033E**	**2034E**	**2035E**
Masitinib for Amyotrophic Lateral Sclerosis (ALS)	0	0	0	0	0	0	0	0	0	0	0	0	0	53,459	123,815	214,207	311,994	417,682	531,804
Masitinib for Progressive Multiple Sclerosis (MS)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Masitinib for Alzheimer's Disease (AD)	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Rx Product Sales	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**-**	**53,459**	**123,815**	**214,207**	**311,994**	**417,682**	**531,804**
Non-Rx Product Operating Income:																			
Non Rx Product Revenue	0			0			0			0	0	0	0	0	0	0	0	0	0
Upfronts, Milestones and Royalty	0			0			0	0	0	0	0		0	0	0	0	0	0	0
Other	970	560	512	1,072	515	500	1,015	500	500	1,000	1,000	1,000	1,000	1,000	1,000	1,000	1,000	1,000	1,000
Total Non-Product Operating Income	**970**	**560**	**512**	**1,072**	**515**	**500**	**1,015**	**500**	**500**	**1,000**	**1,000**	**1,000**	**1,000**	**1,000**	**1,000**	**1,000**	**1,000**	**1,000**	**1,000**
Total Operating Revenue	**970**	**560**	**512**	**1,072**	**515**	**500**	**1,015**	**500**	**500**	**1,000**	**1,000**	**1,000**	**1,000**	**54,459**	**124,815**	**215,207**	**312,994**	**418,682**	**532,804**
Cost of Goods Sold	383	93	(269)	(176)	364	200	564	200	200	400	400	400	400	5,346	12,382	21,421	31,199	41,768	53,180
Gross Profit	**587**	**467**	**781**	**1,248**	**151**	**300**	**451**	**300**	**300**	**600**	**600**	**600**	**600**	**49,113**	**112,434**	**193,786**	**281,795**	**376,914**	**479,624**
Operating Expenses																			
Research and Development	10,477	2,564	1,372	3,936	1,836	2,020	3,856	2,221	2,406	4,627	5,552	6,662	3,331	1,666	1,499	1,514	1,529	1,544	1,560
Administrative expenses	3,017	1,295	1,784	3,079	893	982	1,875	965	1,004	1,969	2,068	8,171	8,579	9,008	9,459	9,932	10,428	10,950	11,497
Marketing expenses	522	190	126	316	150	165	315	162	169	331	347	365	383	402	422	443	465	489	513
Other				0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Total Operating expenses	**14,016**	**4,049**	**3,282**	**7,331**	**2,879**	**3,167**	**6,046**	**3,348**	**3,579**	**6,927**	**7,967**	**15,198**	**12,294**	**11,076**	**11,380**	**11,889**	**12,423**	**12,983**	**13,570**
Operating income (Loss)	(13,429)	(3,582)	(2,501)	(6,083)	(2,728)	(2,867)	(5,595)	(3,048)	(3,279)	(6,327)	(7,367)	(14,598)	(11,694)	38,037	101,054	181,897	269,372	363,931	466,054
Interest expense	-			-			-			-	-	-	-	-	-	-	-	-	-
Interest Income	-			-			-			-	-	-	-	-	-	-	-	-	-
Financial income	4,993	322	356	678	212	233	445	229	238	467	491	515	541	568	597	626	658	691	725
Financial expenses, net	(3,549)	(1,210)	(1,217)	(2,427)	(2,661)	(2,794)	(5,455)	(2,700)	(2,700)	(5,400)	(5,400)	(5,400)	(5,400)	0	0	0	0	0	0
Other Income	-			-															
Total other income	**1,444**	**(888)**	**(861)**	**(1,749)**	**(2,449)**	**(2,561)**	**(5,010)**	**(2,471)**	**(2,462)**	**(4,933)**	**(4,909)**	**(4,885)**	**(4,859)**	**568**	**597**	**626**	**658**	**691**	**725**
Pretax Income	**(11,985)**	**(4,470)**	**(3,362)**	**(7,832)**	**(5,177)**	**(5,428)**	**(10,605)**	**(5,519)**	**(5,740)**	**(11,259)**	**(12,276)**	**(19,483)**	**(16,552)**	**38,605**	**101,650**	**182,523**	**270,030**	**364,621**	**466,779**
Income Tax Expense (Benefit)	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	52,870	67,683
Tax Rate		0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	15%	15%
Fx Effects	0			0			0			0	0	0	0	0	0	0	0	0	0
Net Income (loss)	**(11,985)**	**(4,470)**	**(3,362)**	**(7,832)**	**(5,177)**	**(5,428)**	**(10,605)**	**(5,519)**	**(5,740)**	**(11,259)**	**(12,276)**	**(19,483)**	**(16,552)**	**38,605**	**101,650**	**182,523**	**270,030**	**311,751**	**399,096**
EPS	**(0.24)**	**(0.09)**	**(0.06)**	**(0.15)**	**(0.09)**	**(0.08)**	**(0.17)**	**(0.08)**	**(0.08)**	**(0.17)**	**(0.18)**	**(0.27)**	**(0.22)**	**0.51**	**1.32**	**2.32**	**3.37**	**3.82**	**4.79**
GAAP EPS (dill)	(0.24)	(0.09)	(0.06)	(0.15)	(0.09)	(0.08)	(0.17)	(0.08)	(0.08)	(0.17)	(0.18)	(0.27)	(0.22)	0.51	1.32	2.32	3.37	3.82	4.79
Waged Avg Suhrs (Bas) - '000s	49,907	49,366	52,118	50,742	57,277	66,000	61,638	67,320	68,666	67,993	69,700	71,455	73,612	75,460	76,977	78,524	80,102	81,713	83,355
Waged Avg Suhrs (Dill) - '000s	49,907	49,366	52,118	50,742	57,277	66,000	61,638	67,320	68,666	67,993	69,700	71,455	73,612	75,460	76,977	78,524	80,102	81,713	83,355

Source: Company reports and Maxim

DISCLOSURES



Maxim Group LLC Ratings Distribution			As of: 12/17/25
		% of Coverage Universe with Rating	% of Rating for which Firm Provided Banking Services in the Last 12 months
Buy	Fundamental metrics and/or identifiable catalysts exist such that we expect the stock to outperform its relevant index over the next 12 months.	**84%**	**49%**
Hold	Fundamental metrics are currently at, or approaching, industry averages. Therefore, we expect this stock to neither outperform nor underperform its relevant index over the next 12 months.	**16%**	**55%**
Sell	Fundamental metrics and/or identifiable catalysts exist such that we expect the stock to underperform its relevant index over the next 12 months.	**0%**	**0%**
	**See valuation section for company specific relevant indices*		

I, **Naz Rahman, CFA**, attest that the views expressed in this research report accurately reflect my personal views about the subject security and issuer. Furthermore, no part of my compensation was, is, or will be directly or indirectly related to the specific recommendation or views expressed in this research report.

The research analyst(s) primarily responsible for the preparation of this research report have received compensation based upon various factors, including the firm's total revenues, a portion of which is generated by investment banking activities.

Maxim Group makes a market in AB Science S.A.

Maxim Group managed/co-managed/acted as placement agent for an offering of the securities for AB Science S.A. in the past 12 months.

Maxim Group received compensation for investment banking services from AB Science S.A. in the past 12 months.

Maxim Group expects to receive or intends to seek compensation for investment banking services from AB Science S.A. in the next 3 months.

ABS.PA: For AB Science S.A., we use the BTK (NYSE Biotechnology Index) as the relevant index.

Valuation Methods
ABS.PA: We forecast masitinib launching for the treatment of amyotrophic lateral sclerosis (ALS). We do not model the rest of the pipeline or other indications and assume them to be upside. We apply a sales risk adjustment to masitinib for ALS based on the stage of development, regulatory risk, and commercial risks. A discount rate is then applied to the free cash flow, discounted EPS, and sum-of-the-parts models, which are equally weighted to derive a 12-month price target.

Price Target and Investment Risks
ABS.PA: Aside from general market and other economic risks, risks particular to our price target and rating for AB Science S.A. include: 1) Investment risk: AB Science's pipeline products are not yet approved. Additionally, the company may not generate sufficient revenue to fund operations even after product approvals. 2) Revenue risk: While none of AB Science's pipeline products are currently approved, even if they are approved and commercialized, future revenue may be immaterial or could decline due to various factors. Insufficient revenue could impair AB Science's ability to fund its operations. 3) Development risk: AB Science's potential pipeline products may fail in clinical studies or progress more slowly than expected. The company may also divest or cancel pipeline programs due to evolving development or commercial risks. Additionally, the company may fail to expand its pipeline in a manner satisfactory to investors. 4) Regulatory risk: AB Science's clinical-stage products may not be successful in clinical trials and may not meet the requirements for regulatory approval(s). Regulatory agencies may determine that trial results are insufficient for approval, even if statistically significant, due to safety concerns or lack of clinical meaningfulness. 5) Commercial risk: AB Science's pipeline products are not approved or commercialized, and if/when they become commercially available, they may not achieve meaningful market share. The company may also fail to obtain sufficient reimbursement from payers, limiting market adoption. 6) Reimbursement risk: The company may fail to obtain sufficient reimbursement from managed care agencies to pay for new products, and reimbursement may decline for existing products. Managed care companies may impose restrictions on reimbursement that could cause healthcare practitioners to be less willing to prescribe products, thereby impacting market adoption and sales. 7) Patent risk: AB Science may eventually lose patent protection for its products. Loss of patents may lead to generic entrants, resulting in market share erosion, lower sales, and pricing pressure. 8) Pricing risk: Pricing pressure from generics and new market entrants may force AB Science to reduce net prices or offer higher rebates, adversely impacting profitability. 9) Competitive technology risk: While AB Science has innovative products, competitors may develop products using newer technologies that may gain patient, physician, and payer support, displacing AB Science's products. 10) Manufacturing risk: AB Science is dependent on third-party contract manufacturing organizations. These partners may fail to deliver sufficient quantities, meet quality standards, or do so at acceptable costs, which could impact company revenue or margins. 11) Supply chain risk: Domestic or global supply chains may negatively impact AB Science's ability to source materials necessary for manufacturing. 12) Financial risk: The company may experience market share loss and declining sales, which could impair its ability to cover operating expenses. 13) Foreign exchange risk: While AB Science is domiciled in France and reports results in euros (€), the company's clinical trials and operations are conducted internationally, exposing it to currency fluctuations. Ineffective or insufficient hedging could reduce revenue, increase costs, and adversely affect the company's operations. 15) Dilution risk: The company may require additional capital raises to fund operations, and equity-based offerings may dilute existing investors. 16) Credit Risk: AB Science has debt on its balance sheet. The company may be unable or unwilling to meet interest or principal payments, which could result in default, asset seizure, downgraded ratings, reduced borrowing capacity, or bankruptcy.

RISK RATINGS

Risk ratings take into account both fundamental criteria and price volatility.

Speculative – Fundamental Criteria: This is a risk rating assigned to early-stage companies with minimal to no revenues, lack of earnings, balance sheet concerns, and/or a short operating history. Accordingly, fundamental risk is expected to be significantly above the industry. Price Volatility: Because of the inherent fundamental criteria of the companies falling within this risk category, the price volatility is expected to be significant with the possibility that the investment could eventually be worthless. Speculative stocks may not be suitable for a significant class of individual investors.

High – Fundamental Criteria: This is a risk rating assigned to companies having below-average revenue and earnings visibility, negative cash flow, and low market cap or public float. Accordingly, fundamental risk is expected to be above the industry. Price Volatility: The price volatility of companies falling within this category is expected to be above the industry. High-risk stocks may not be suitable for a significant class of individual investors.

Medium – Fundamental Criteria: This is a risk rating assigned to companies that may have average revenue and earnings visibility, positive cash flow, and is fairly liquid. Accordingly, both price volatility and fundamental risk are expected to approximate the industry average.

Low – Fundamental Criteria: This is a risk rating assigned to companies that may have above-average revenue and earnings visibility, positive cash flow, and is fairly liquid. Accordingly, both price volatility and fundamental risk are expected to be below the industry.

DISCLAIMERS

Some companies that Maxim Group LLC follows are emerging growth companies whose securities typically involve a higher degree of risk and more volatility than the securities of more established companies. The securities discussed in Maxim Group LLC research reports may not be suitable for some investors. Investors must make their own determination as to the appropriateness of an investment in any securities referred to herein, based on their specific investment objectives, financial status and risk tolerance.

This communication is neither an offer to sell nor a solicitation of an offer to buy any securities mentioned herein. This publication is confidential for the information of the addressee only and may not be reproduced in whole or in part, copies circulated, or disclosed to another party, without the prior written consent of Maxim Group, LLC ("Maxim").

Information and opinions presented in this report have been obtained or derived from sources believed by Maxim to be reliable, but Maxim makes no representation as to their accuracy or completeness. The aforementioned sentence does not apply to the disclosures required by FINRA Rule 2241. Maxim accepts no liability for loss arising from the use of the material presented in this report, except that this exclusion of liability does not apply to the extent that such liability arises under specific statutes or

regulations applicable to Maxim. This report is not to be relied upon in substitution for the exercise of independent judgment. Maxim may have issued, and may in the future issue, other reports that are inconsistent with, and reach different conclusions from, the information presented in this report. Those reports reflect the different assumptions, views and analytical methods of the analysts who prepared them and Maxim is under no obligation to ensure that such other reports are brought to the attention of any recipient of this report.

Past performance should not be taken as an indication or guarantee of future performance, and no representation or warranty, express or implied, is made regarding future performance. Information, opinions and estimates contained in this report reflect a judgment at its original date of publication by Maxim and are subject to change without notice. The price, value of and income from any of the securities mentioned in this report can fall as well as rise. The value of securities is subject to exchange rate fluctuation that may have a positive or adverse effect on the price or income of such securities. Investors in securities such as ADRs, the values of which are influenced by currency volatility, effectively assume this risk. Securities recommended, offered or sold by Maxim: (1) are not insured by the Federal Deposit Insurance Company; (2) are not deposits or other obligations of any insured depository institution; and (3) are subject to investment risks, including the possible loss of principal invested. Indeed, in the case of some investments, the potential losses may exceed the amount of initial investment and, in such circumstances, you may be required to pay more money to support these losses.

ADDITIONAL INFORMATION IS AVAILABLE UPON REQUEST

MAXIM GROUP

Corporate Headquarters

New York City
300 Park Ave., 16TH Floor
New York, NY 10022
Tel: 212-895-3500

Capital Markets/Syndicate
212-895-3695

Corporate Services
212-895-3818

Equity/Options Trading
212-895-3796

Equity Research
212-895-3736

Fixed Income Trading
212-895-3875

South Florida Hub
555 Washington Ave., Suite 320
Miami Beach, FL 33139
Tel: 786-864-0880

Global Equity Trading
212-895-3623

Institutional Sales/Sales Trading
212-895-3873

Prime Brokerage
212-895-3668

Wealth Management
212-895-3540

Stamford, Connecticut
700 Canal Street
Stamford, CT 06902

Red Bank, New Jersey
68 White Street, 2nd Floor
Red Bank, NJ 07701
Tel: 732-784-1900

Fort Lauderdale, Florida
1 East Broward Blvd, Suite 1430
Fort Lauderdale, FL 33301

Woodbury, New York
100 Crossways Park Dr West, Suite 207
Woodbury, NY 11797
Tel: 516-393-8300