AB Science Highlights

January 2026

Forward-looking statements and disclaimer 1/2

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AB Science management is experienced, stable, and committed to deliver these milestones

ALAIN MOUSSY, Co-founder and CEO

Former Head of Carrefour Corporate Development

President of AFIRMM, association of mastocytosis patients

CHRISTIAN FASSOTTE, MD, **Chief Medical Officer**

Medical Doctor. 30 years of experience, including executive position at Sanofi for Medical, Regulatory Affairs and R&D

OLIVIER HERMINE, MD, PhD , **Co-founder and Chairman of Scientific Committee**

Member of the French Académie des Sciences and author of 900 international publications

LAURENT GUY, Chief Financial Officer

Former positions in the banking industry (Société Générale and Paribas) and strategy consulting (Accenture).

AB Science is a European clinical development stage company with ALS and AML as key indications

Proprietary Drug Portfolio

Platform	Drug / *Target*	Therapeutic area	Indication	Development Stage
Tyrosine Kinase Inhibitor	Masitinib (Veterinary)	Oncology	Canine Mast Cell Tumor	Registered in the EU (>1M€ annual sales)
Tyrosine Kinase Inhibitor	Masitinib (Oral)	Neuro-degenerative Diseases (NDD)	Amyotrophic Lateral Sclerosis	Phase 3 *Authorized**
			Progressive Forms of Multiple Sclerosis	Phase 3 *Authorized**
			Alzheimer's Disease	Phase 3 *Authorized**
		Blood diseases	Sickle Cell Disease	Phase 2 *To be authorized*
ALDH / Microtubule	AB8939 (IV)	Hematology	Acute Myeloid Leukemia (AML)	Phase 1 *Initiated in Europe*
Tyrosine Kinase Inhibitor		Neuro-degenerative Diseases (NDD)		Drug Discovery

* EMA and FDA

Key Considerations

- **AML Phase 1 and expansion phase are fully financed**
- **SCD phase 2 is fully financed through 9M€ grant**
- **AB Science has cash runway above twelve months with these programs on-going**
- **AB Science is seeking funding of phase 3 in ALS**
- **AB Science is seeking to partner MS and AD phase 3 programs**
- **IP rights are owned by AB Science**

MASITINIB IN AMYOTROPHIC LATERAL SCLEROSIS

Masitinib is an orally-administered kinase inhibitor selectively targeting mast cells and macrophages, uniquely positioned to realize its therapeutic potential in neurodegenerative diseases (NDDs)

Masitinib targets mast cells

- Masitinib is designed to be a potent and selective inhibitor of c-Kit, Lyn, and Fyn kinases. These kinases play critical roles in the activation of mast cells

Masitinib targets macrophages/microglia

- Masitinib is designed to be a potent and selective inhibitor of MCSFR-1

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
Macrophages / Microglia	MCSFR-1	90	0.0076

Dubreuil 2009, PLoSONE.4(9):e7258; AB Science. Davis 2011, Nat Biotechnol; 29(11):1046

Microglia and mast cells are at the cutting edge of research regarding NDDs, with a consensus that drugs aimed at these targets will have strong therapeutic potential

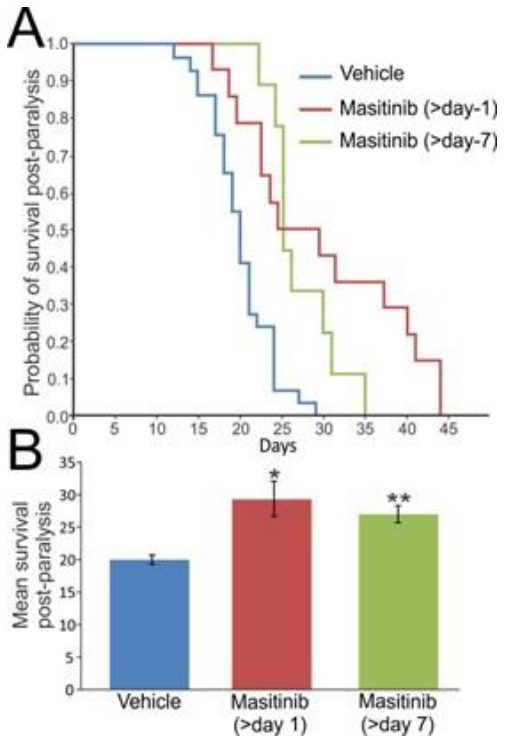
[1] Mado H, et al. Int J Mol Sci. 2023;24(3):1861. [2] Sandhu JK, et al. Int J Mol Sci. 2021;22(3):1093. [3] Muzio L, et al. Front Neurosci. 2021;15:742065. [4] Hagan N, et al. Cell Death Dis. 2020;11(10):904. [5] Jones MK, et al. Front Cell Neurosci. 2019;13:171. [6] Long JM, et al. Cell. 2019;179(2):312-339. [7] Skaper SD, et al. Front Cell Neurosci. 2018 Mar 21;12:72.

Masitinib clinical trial data shows neuroprotective benefits in ALS, MS, and AD, showing that targeting microglia and mast cells is a valid strategy

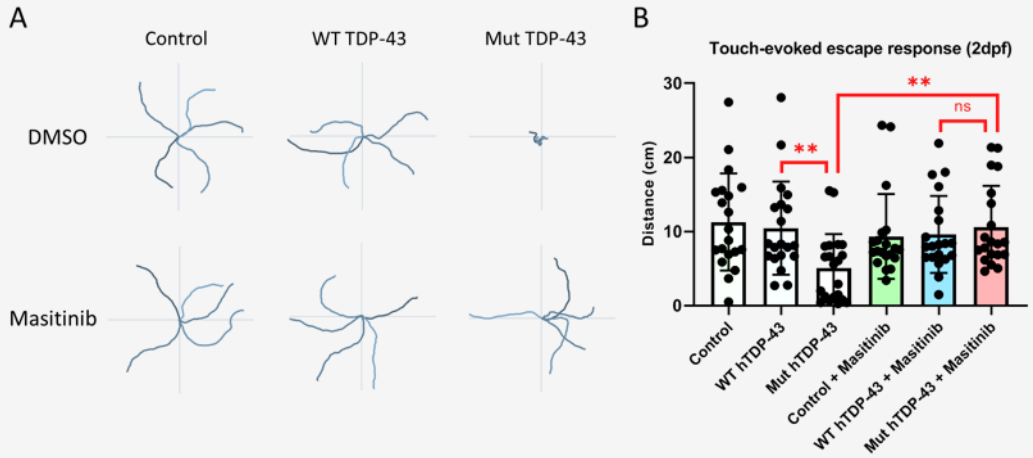
[8] Ketabforoush AHME, et al. Biomed Pharmacother. 2023;160:114378. [9] Kovacs M, et al. Acta Neuropathol Commun. 2021;9(1):136. [10] Trias E, et al. Glia. 2020;68(6):1165-1181. [11] Harrison JM, et al. Neurobiol Dis. 2020;145:105052. [12] Trias E, et al. JCI Insight. 2018;3(19):e123249. [13] Trias E, et al. JCI Insight. 2017;2(20):e95934. [14] Trias E, et al. J Neuroinflammation. 2016;13(1):177. [15] Lin CJ, et al. Cell Rep. 2023;42(9):113141. [16] Harcha PA, et al. Int J Mol Sci. 2021;22(4):1924. [17] Leng F et al. Nat Rev Neurol. 2021;17(3):157-172. [18] Li T, et al. J Alzheimers Dis. 2020;76(4):1339-1345. [19] Schwabe T, et al. Neurobiol Dis. 2020;143:104962. [20] Folch J, et al. Expert Rev Neurother. 2015 May 11:1-10. [21] Kamma E, et al. J Neuroinflammation. 2022;19(1):45. [22] Mahmood A, et al. Curr Opin Pharmacol. 2022;63:102188. [23] Pinke KH, et al. Neural Regen Res. 2020;15(11):1995-2007.. [24] Vermersch P, et al. BMC Neurol. 2012;12:36.

Masitinib has a proven mechanism of action in animal models of ALS, published in several peer-reviewed journals

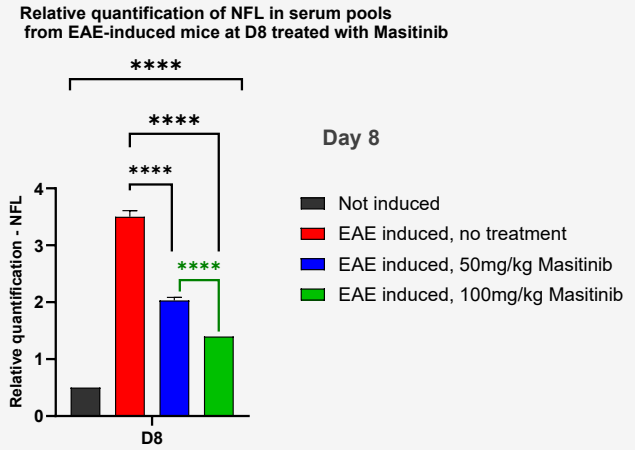
Masitinib treatment initiated 7 days after paralysis onset prolonged survival by 40 % in SOD1 model



Masitinib restored motor function in a zebrafish model of mutant TDP-43 overexpression



Masitinib lowered blood levels of neurofilament light (NfL) in a neurodegenerative disease model*



*EAE model

Díaz-Amarilla P., et al., Proc Natl Acad Sci U S A. 2011 Nov 1;108(44) ; Trias, E., et al., JCI Insight, 2017. **2**(20) ; Trias, E., et al. J Neuroinflammation, 2016. **13**(1): p. 177 ; Trias, E., et al.. JCI Insight. 2018. **3**(19).

Phase 2B enrolled 394 patients with broad inclusion criteria and evaluated Masitinib after 48 weeks of treatment. The primary analysis was preplanned in the population defined as “Normal” progressors, following an early protocol amendment

Design

Design:

Double blind, placebo controlled, 2-parallel groups

Standard of care: Riluzole

394 patients enrolled

- Masitinib 4.5 mg/kg/day + riluzole : 130 patients
- Masitinib 3.0 mg/kg/day + riluzole : 131 patients
- Placebo + riluzole : 133 patients

Primary endpoint:

- Change in the ALSFRS-R score at 48 weeks (ΔALSFRS-R)

Duration: 48 weeks

Main inclusion criteria

- Probable, or definite ALS, sporadic or familial ALS, without restriction on baseline ALSFRS-R score
- Stable dose of riluzole for at least 30 days prior to screening
- Patients with disease duration ≤ 36 months
- Patients with FVC ≥ 60%

Statistical analysis

Two distinct populations were differentiated:

- ‘Normal Progressors’: rate <1.1 points/month — **84% of trial patients**
- ‘Fast Progressors’: rate ≥ 1.1 points/month — **16% of trial patients**

- Rate of ALSFRS-R progression from first symptom to randomization (points/month):

Efficacy analyses were conducted in a stepwise manner

- Fixed sequence method, to control the global family-wise error rate at the 0.05 level for the primary analysis for each dose.

	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

Study AB10015 generated consistent results on primary and secondary endpoints

ALSFRS-R (Normal Progressors ; M4.5 vs P)

Analysis (M4.5 vs placebo)	**Difference**	**p-value**
mLOCF Method (PRIMARY)	3.39	0.0157
CIR Method (SENSITIVITY)	2.67	0.462

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

Analysis (M4.5 vs placebo)	**Difference**	**p-value**
mLOCF Method	-7.76	0.0078

FVC (Normal Progressors ; M4.5 vs P)

Analysis (M4.5 vs placebo)	**Difference**	**p-value**
mLOCF Method	7.54	0.0296

Progression Free Survival analysis (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]	

Event Free Survival analysis (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]	

OS analysis (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]	

- Primary analysis : 27% difference in slope of deterioration
- Copy Increment in Reference (CIR) assumes progressive return to reference (placebo effect) for patients who discontinue masitinib treatment
- There was an observed benefit in quality of life
- There was an observed benefit in respiratory function
- 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
- There was an Event Free Survival (EFS) benefit (+3 months) with a p=0.0162
- Event being the earlier event between progression, tracheotomy, ventilation or gastrostomy
- Trend of survival benefit based on long-term follow-up

A large proportion of patients in phase 2B/3 treated with masitinib 4.5 mg/kg/day had a survival duration of more than 5 years from onset

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%)

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

Masitinib holds the promise of a significantly improved survival, potentially doubling life expectancy in the target population

In its benefit-risk assessment, EMA considered the safety of Masitinib acceptable for further trials in ALS

Masitinib Safety Profile based on a Large Database

Number of Patients Randomized in Masitinib Treatment Arms
Safety Population

Safety population		Patients exposed to Masitinib				
	All	≤ 3 months	More than 3 months	More than 6 months	More than 1 year	More than 2 years
All	**4,318**	**1,674**	**2,644**	**1,924**	**1,255**	**560**
Healthy Volunteers	96	96	0	0	0	0
Non-Oncology subjects	**2,184**	**565**	**1,619**	**1,307**	**958**	**453**
Oncology subjects	2,038	1,013	1,025	617	297	107
ICH Topic E1 requirements for non-orphan drugs	*1,500*			*300-600*	*100*	

Masitinib Safety is Deemed Acceptable in ALS

CHMP Latest Assessment Report

Conclusion on clinical safety

"Having considered the data from masitinib studies, the safety profile of mentioned medicinal product is considered acceptable" [1]

1 Extract from EMA communication

There was 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)

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

A strong hypothesis was generated from this phase 2B/3 for confirmatory phase 3 with the identification of an optimal population

		AB10015 - Primary Analysis Population (Normal Progressors)	AB10015 – Subgroup Analysis
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**
Quality of Life (ALSAQ-40) (CIR)	Diff. of mean	-6.04	**-6.22**
	p-value	0.0305	**0.044**
Forced Vital Capacity (FVC) (CIR)	Diff. of mean	5.85	**7.59**
	p-value	0.0931	**0.0384**
Median Progression Free Survival	Gain	+ 4 months	**+ 9 months**
	Median [95% CI]	20 vs 16	25 vs 16
	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 vs 40	53 vs 41
	p-value log rank	0.0761	**0.0192**

Subgroup identified for Confirmatory Phase 3:

ALS patients <u>prior to any loss of function</u>

- Subgroup with ALSFRS-R > 0 on all items
- Results consistent with Mechanism of Action (neuroprotection but no regrowth of destroyed motorneurones)

All key parameters are aligned to maximize risks of success of the phase 3 study

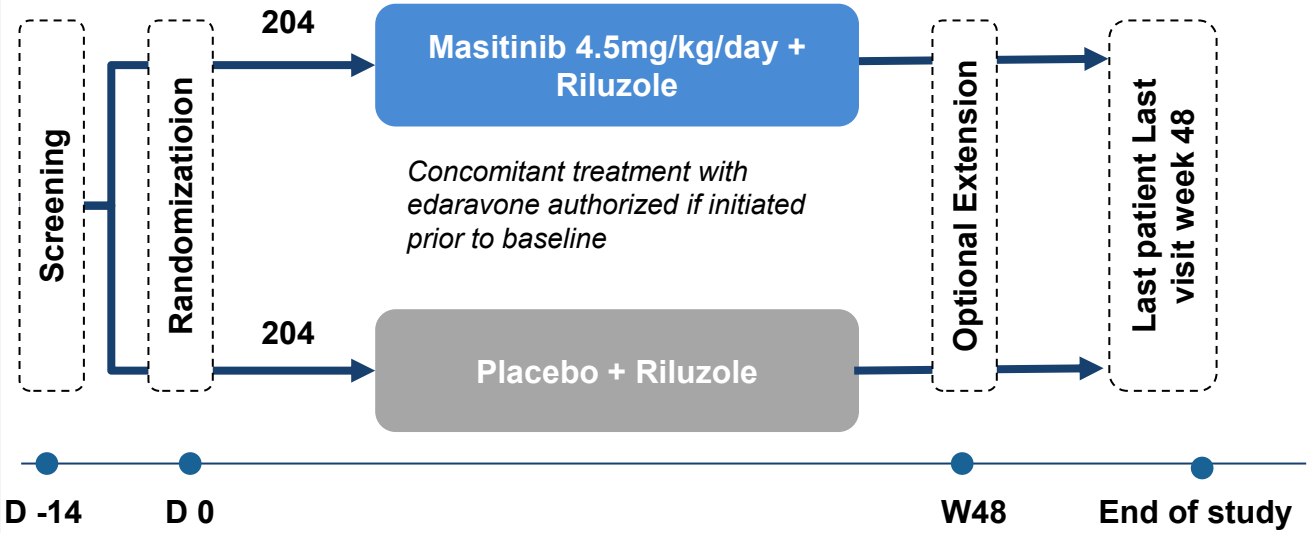
- Having validated masitinib MoA, modulating microglia and inhibiting mast cells
- Reducing heterogeneity of the study population through identification of an optimal population : patients prior to loss of function
- Having the same long term time point week 48 in phase 2b/3 and confirmatory phase 3
- Doubling necessary sample size from ~90 patients per arm to ~200 patients per arm
- Benefiting from a large safety database of more than 4,000 patients with safety considered acceptable by agencies
- Having identified a Biomarker in the plasma of pathological microglia and introducing it in phase 3 or launching a dedicated phase 1 in 2026 in parallel
- Choosing a global CRO to reduce risk of execution and protecting integrity of data

The objections raised by EMA in the context of a conditional marketing authorization procedure have been mitigated and solutions integrated into the next confirmatory phase 3 study authorized now both by EMA and FDA

EMA Assessment		Mitigation
Primary analysis population	EMA disagreed with the exclusion of fast progressors in the primary analysis after study started even if the amendment was early	▪ Exclusion of Fast progressors and the definition of fast progressors have been validated by EMA's Scientific Advisory Group Neurology ▪ Exclusion of fast progressors is prespecified in the confirmatory phase 3
Imputation of data for discontinued patients	Missing data should be treated with a penalty in case of discontinuation.	▪ Integrated in the statistical part of the confirmatory study ▪ The sample size has been enlarged to detect a significant treatment effect with two conservative imputation methods, Copy Increment in Reference (CIR) and Jump to Reference (JTR)
Compliance	EMA inspection highlighted protocol deviations	▪ For Phase 3, a global CRO will ensure data quality and integrity.

The confirmatory phase 3 has been authorized by FDA and EMA and is endorsed by prominent opinion leaders in ALS

Authorized Phase 3 design



Steering Committee Support from prominent opinion leaders in ALS

Institution	Expert
University of Ulm – Germany	Dr. A. Ludolph
Hospital Universitario San Rafael, Madrid - Spain	Dr. J. Mora
Mass General Brigham Neuroscience Institute, Boston - USA	Dr. M. Cudkowicz
Lewis Katz School of Medicine at Temple University, Philadelphia - USA	Dr. T. Heiman
CHU Limoges - France	Dr. P. Couratier

Duration :
- 48 weeks, Open label treatment after week 48

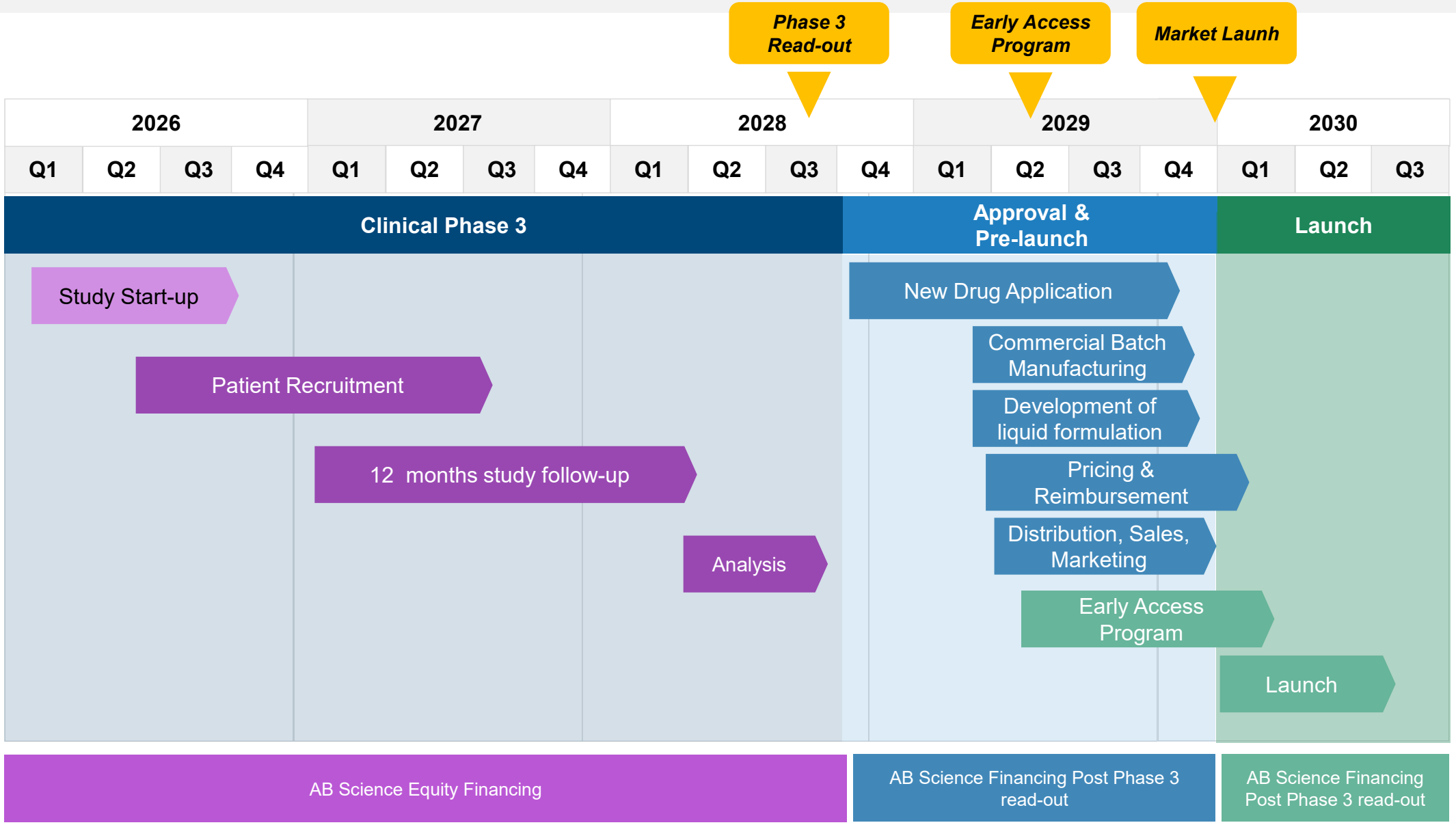
Sample size
- 408 patients, Masitinib 4.5 mg/kg/day vs placebo, Randomization 1:1

Primary endpoint
- CAFS (FDA), Change in ALSFRS-R (EMA)

Main secondary endpoints
- PFS, Quality of life, OS

Surrogate endpoint
- NfL, Biomarkers

A clear plan is laid out to read-out phase 3 study by Q4 2028, initiate early access program in Q2 2029 and launch end 2029, assuming study initiating in Q1 2026



Masitinib has a blockbuster potential in ALS, with projected peak sales of 2B€, if approved, based on strategic consulting survey

	EUROPE	US
Global disease Prevalence	6 per 100.000	6 per 100.000
Number of ALS patients	30,000	20,000
% of patients targeted in the disease*	75%	75%
Expected market share in the label**	75%	75%
% of patients covered by insurance	90%	90%
Annual treatment price***	80K€	140K$
Potential annual peak sales	~1bn €	~1bn $

** : Excluding fast progressors patients and excluding patients prior to complete loss of function*
*** : In ALS, which is a fatal disease, patients regularly use all available treatments*
**** : Based on 12 months treatment duration, as per study data*

AB8939 IN ACUTE MYELOID LEUKEMIA

AB8939 targets proliferating leukemia blasts and leukemia stem cells

Problem in AML is recurrence of tumor

This problem may be solved by the dual Moa of AB8939

CANCER CELLS ARE RESISTANT TO CHEMOTHERAPY

BLOCKING PROLIFERATING LEUKEMIA CELLS THROUGH MICROTUBULES DISRUPTION

- Destabilizes microtubules a well known MoA in cancer but that suffers for known drugs (taxol, taxotere, vincristine, vinblastine) from multi drug resistance and rapid metabolization
- AB8939 is not subjected to multidrug resistance (no PgP binding)
- AB8939 is not degraded by myeloperoxidase

RELAPSING DUE TO PERSISTENCE OF CANCER STEM CELLS

TARGETING OF LEUKEMIA CANCER STEM CELLS THOUGH ALDHS INHIBITION

- ALDH plays critical role in cancer stem cells
- AML cells with high ALDH activity are more resistant to chemotherapeutic agents
- ALDH is over-expressed in MECOM

THIS DUAL EFFECT IS DESIGNED TO DECREASE OF TUMOR RECURRENCE

Phase 1 in monotherapy has been completed and phase 1 in combination has begun

Step 1

MTD* 3 DAYS
AB8939 alone

28 patients randomized
(some patients received several cycles at increased dose following observed clinical benefit)

Step 2

MTD 14 DAYS
AB8939 alone

13 patients randomized

Step 3

MTD 14 DAYS
AB8939 Combination

- **AB8939 + Venetoclax**
- **AB8939 + Venetoclax + Azacitidine**

#	Dose	Patients	DLT	
1	0.9 mg/m^2	3	0	
2	1.8 mg/m^2	3	0	
3	3.6 mg/m^2	3	0	
4	6.0 mg/m^2	3	0	
5	9.0 mg/m^2	3	0	
6	12.0 mg/m^2	3	0	
7	16.0 mg/m^2	3	0	
8	**21.3 mg/m^2**	**4**	**1**	**MTD 3D**
9	28.3 mg/m^2	3	2	

#	Dose	Patients	DLT	
1	16.0 mg/m^2	7	1	
2	**21.3 mg/m^2**	**6**	**1**	**MTD 14D**

AB8939 + Venetoclax

#	Dose	Patients	DLT
1	16.0 mg/m^2	3	0
2	21.3 mg/m^2	1	0

THERE IS A STRONG RATIONALE TO COMBINE AB8939 WITH VENETOCLAX

1. **BOTH MOLECULES HAVE LOW HEMATOLOGIC TOXICITY**
2. **BOTH MOLECULES HAVE DIFFERENT AND COMPLEMENTARY TARGETS IN CANCER CELLS**

In combination, AB8939 + Venetoclax remarkably generated responses in patients in Line 3 or 4 with high risk adverse profile, complex karyotypes, TP53, Mecom and NRAS mutation. There was no toxicity, no DLT

Outcome of the first four patients receiving AB8939 + Venetoclax in Step 3 of phase 2 study

Patient ID	Treatment Line	Prognostic Risk	Response
#1 (16 mg/m2 plus venetoclax)	3rd Line	NRAS mutant	Complete remission with incomplete hematologic recovery (CRi)
#2 (16 mg/m2 plus venetoclax)	4th Line	Complex karyotype , mecom	Partial remission at D45 with no circulating blasts
#3 (16 mg/m2 plus venetoclax)	4th Line	Complex karyotype and TP53 mutation	Partial remission (PR)
#4 (21 mg/m2 plus venetoclax)	3rd Line	Monosomy of chromosome 5 and TP53 mutation	Partial remission (PR)

The objective is to position AB8939 combination treatment to be the standard of care in AML with a high risk profile adverse genetic, which represents a market size potential above EUR 2 billions per annum

- Adverse genetics regroup TP53 mutant, NRAS/KRAS mutant, complex karyotype, monosomy 5 and 7, Mecom
- It represents around 50% of patients.
- These patients are refractory to or relapsing the current standard of care. Median survival is less than 12 months

Region	Incidence Case (1)	% high risk profile (2)	% Insured Patients (3)	Drug Price (€)	Market Size (per in in Mio EUR)
USA / CANADA	23,700	50%	90%	100,000(5)	1 000 000
EUROPE	27,600		90%	60,000	770 000
APAC	27,800		30%	60,000	250 000
INDIA	11,000		30%	60,000	100,000
LATAM	7,200		30%	60,000	65 000
MENA	3,900		30%	60,000	35 000
TOTAL	**90,200**				**2 200 000**

(1) Zhou, Y et al. Global, regional, and national burden of acute myeloid leukemia, 1990–2021: a systematic analysis for the global burden of disease study 2021. Biomark Res 12, 101 (2024).
(2) estimated
(3) Estimated
(4) Choi M. et al. Costs per patient achieving remission with venetoclax-based combinations in newly diagnosed patients with acute myeloid leukemia ineligible for intensive induction chemotherapy. Journal of Managed Care & Specialty Pharmacy Volume 28, Number 9. https://doi.org/10.18553/jmcp.2022.22021

MASITINIB IN PROGRESSIVE FORMS OF MULTIPLE SCLEROSIS AND IN MILD ALZHEIMER'S DISEASE

There is a tremendous unmet need in progressive MS, with no approved drugs for non-active secondary progressive MS and one for primary progressive MS

- Vast majority of drugs are effective only in active MS patients because these drugs stop immune attacks (active inflammation) but cannot repair myelin damage or protect nerves
- Ocrevus is indicated in specific forms of primary progressive MS (PPMS), for the treatment of adult patients with early PPMS in terms of disease duration and level of disability, and with imaging features characteristic of inflammatory activity

	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			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

Masitinib positioning

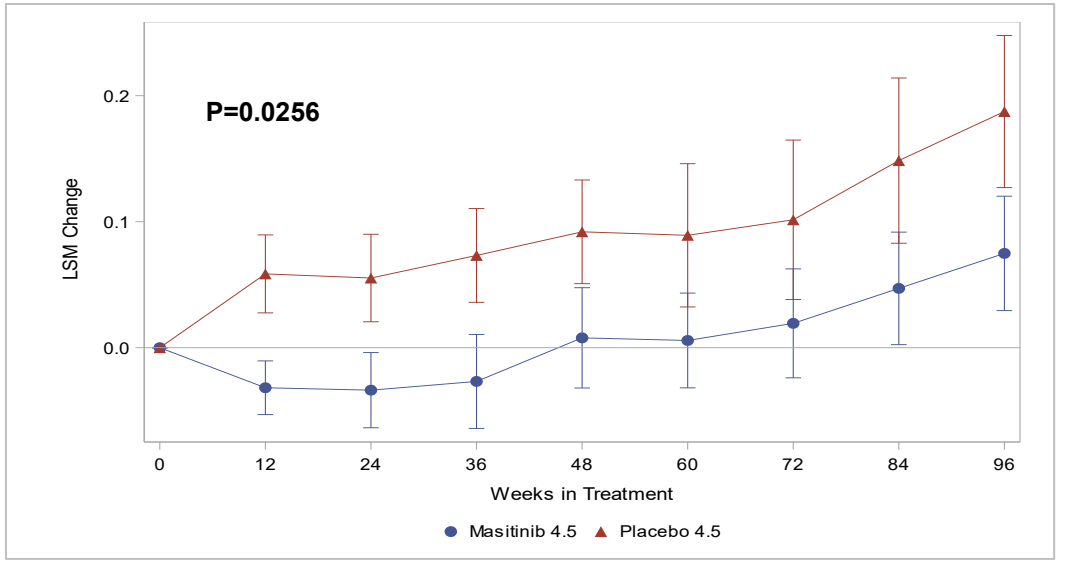
*: *Non-active SPMS is a stage of MS that follows relapsing-remitting multiple sclerosis and that is characterized with an EDSS score progression ≥ 1 point without any relapse in the last 2 years.*

Phase 2B/3 enrolled 656 MS patients tested 2 doses in two sub-studies and demonstrated a significant benefit on disability progression with masitinib 4.5 mg/kg/day in patients with advanced stage of the disease

Sub-study I	Sub-study II
Dosing **Masitinib 4.5 mg/kg/day vs matching placebo**	**Dosing** **Masitinib 4.5 to 6.0 mg/kg/day titration vs matching placebo**
ITT population N=301 (M=200 ; P=101)	**ITT population** N=310 (M=203 ; P=107)
mITT population* N=300 (M=199 ; P=101)	**mITT population*** N=310 (M=203 ; P=107)

Efficacy analyses were performed in the mITT population

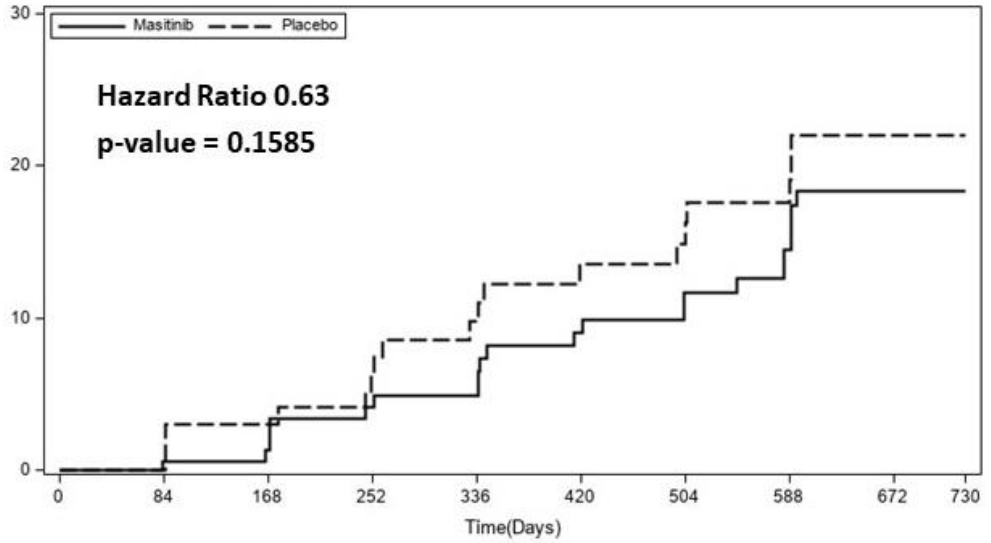
Primary endpoint
Significant reduction in progression on EDSS (Primary Endpoint*)



* Change in EDSS

Although head-to-head comparison is not possible, results with masitinib are promising in light of tolebrutinib results

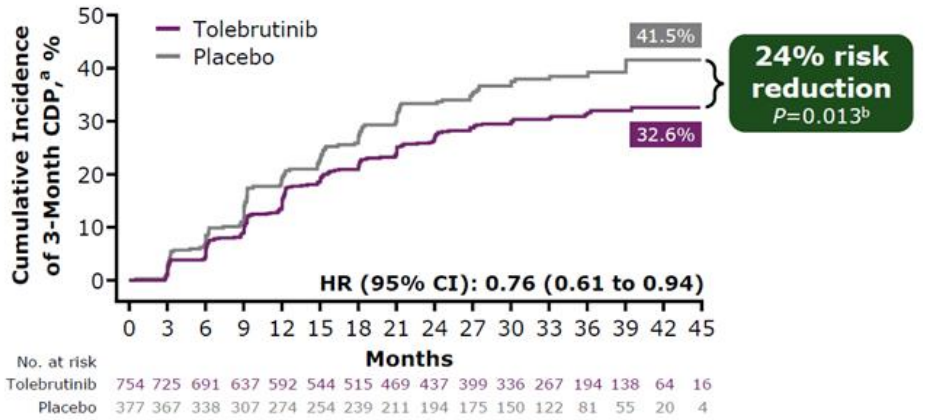
Masitinib
37% risk reduction of time to 3-months confirmed disability progression



Masitinib benefit achieved before year 2

2 years

Tolebrutinib
24% risk reduction of time to 3 months confirmed disability progression



- Tolebrutinib demonstrated a significant effect on time to 3-month CDP

Tolebrutinib benefit achieved between year 2 and year 3

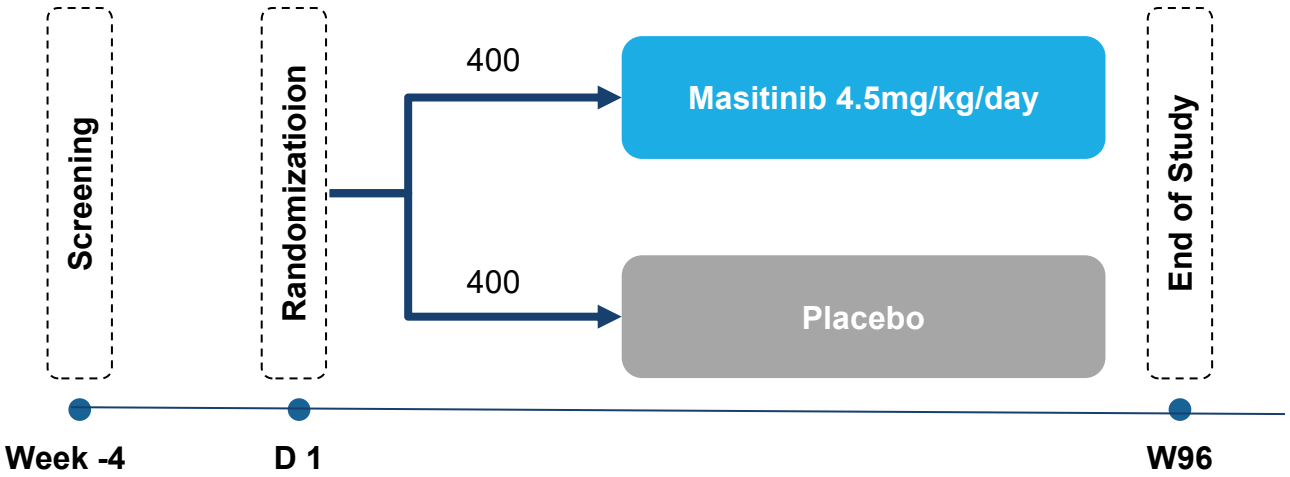
3 years

***AB07002 (NCT01433497)*

**ECTRIMS 2024 Presentation #O136*

Confirmatory phase 3 MS study is authorized by FDA and key European countries

Authorized Phase 3 design



Primary endpoint

- Time to confirmed EDSS progression

Main secondary endpoints

- Change in EDSS from baseline considering all measurements from baseline up to Week 96
- Time to EDSS score of 7.0
- Brain MRI Assessments

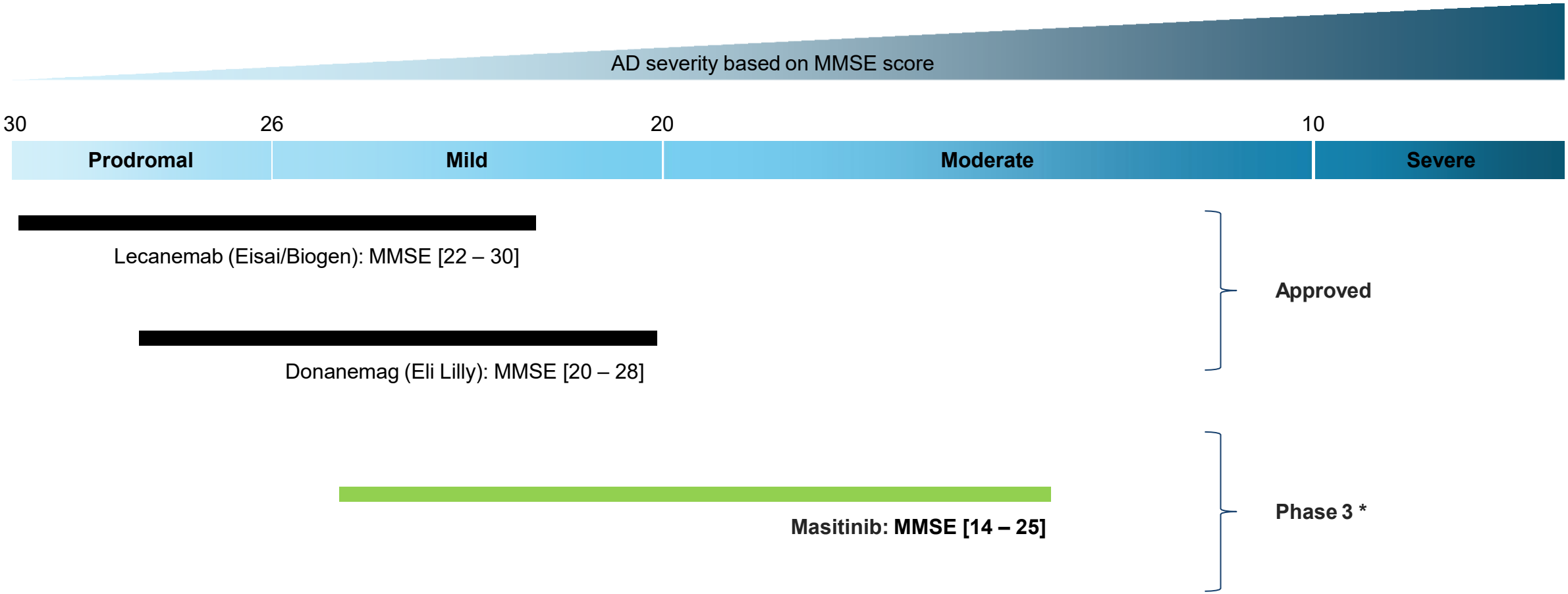
Confirmatory phase 3 study design has been discussed with FDA and EMA

Approved countries

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

Sites selected : 94

Masitinib is being developed for more advanced stages of Alzheimer's disease as compared with biologics recently approved



- Confirmatory phase 3 study is authorized by FDA and key European countries and to be started

Phase 2B/3 study enrolled 718 AD patients in two sub-studies and demonstrated a significant reduction in cognitive impairment based on ADAS-COG (p=0.0003) and improvement on daily activity based on ADCS-ADL (p=0.0381) with masitinib 4.5 mg/kg/day

Primary Analysis

Significant effect on cognitive function after 24 weeks of treatment
Change in ADAS-Cog - ANCOVA Analysis (Full Analysis Set) - M4.5 vs Placebo

Treatment	n	LS Mean (95% CI)	LS Mean Difference (97.51% CI)	p-value
Masitinib 4.5 mg/kg/day + SoC	182	**-1.46** (-2.46, -0.45)	**-2.15** (-3.48, -0.81)	**0.0003**
Placebo + SoC	176	**0.69** (-0.36, 1.75)		

Significant effect on Daily Activity after 24 weeks of treatment
Change in ADCS-ADL - ANCOVA Analysis (Full Analysis Set) - M4.5 vs Placebo

Treatment	n	LS Mean (95% CI)	LS Mean Difference (97.51% CI)	p-value
Masitinib 4.5 mg/kg/day + SoC	182	**1.01** (-0.48, 2.50)	**1.82** (-0.15, 3.79)	**0.0381**
Placebo + SoC	176	**-0.81** (-2.36, 0.74)		

Exploratory Analysis - Preferred endpoint for Phase 3

Significant improvement in combined Cognition and Daily Activities after 24 weeks of treatment
Change in iARDS - ANCOVA Analysis (Full Analysis Set) - M4.5 vs Placebo

Treatment	n	LS Mean Difference (95% CI)	P-value
Masitinib 4.5 mg/kg/day + SoC	182	**3.45** (0.90, 6.00)	**0.0025**
Placebo + SoC	176		

Clinically relevant benefit on top of standard of care (SoC)

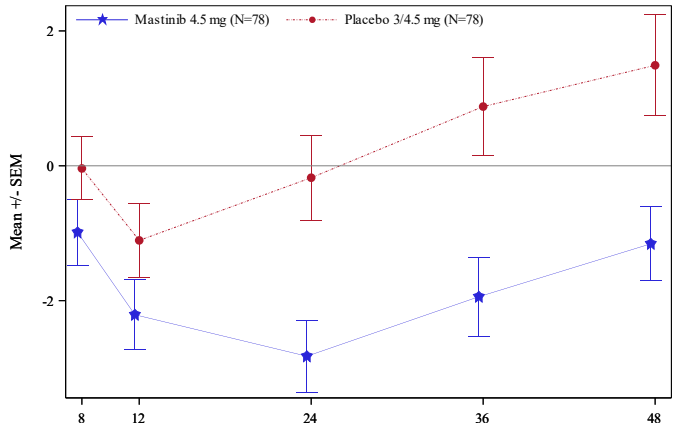
SoC = memantine and anticholinesterase

The clinical benefit on ADAS-Cog was greater in patients with mild impairment than in patients with moderate impairment

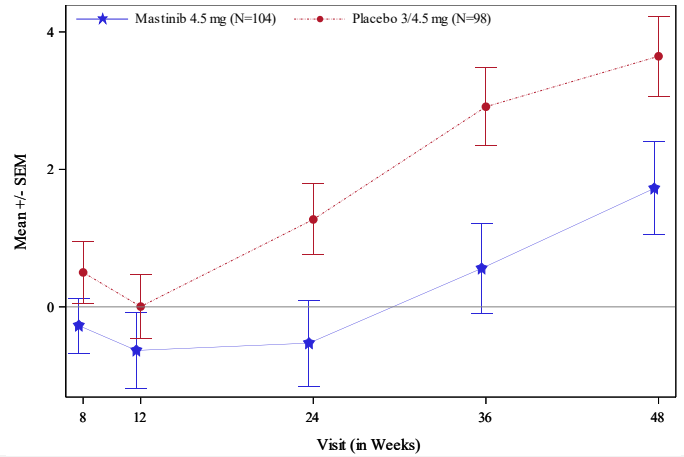
Significant effect on Composite ADAS-COG

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

Mild AD



Moderate AD

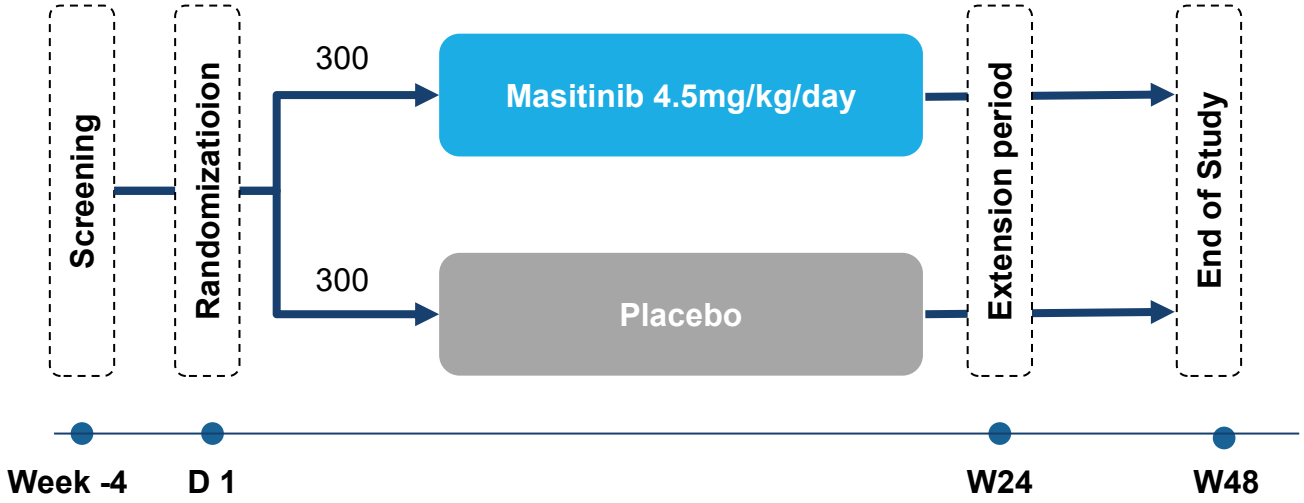


Week 24	n	LS Mean (95% CI)	LS Mean Diff. (96% CI)	p-value
Mild patients				
M + SoC	78	-3.23 (0.807)	-2.62 (-4.45, -0.78)	0.0015
P + SoC	78	-0.61 (0.812)		
Moderate patients				
M + SoC	104	-0.55 (0.655)	-1.80 (-3.66, 0.06)	0.0300
P + SoC	98	1.25 (0.721)		

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

Confirmatory phase 3 AD study is authorized by FDA and key European countries

Authorized Phase 3 design



Primary endpoint

- Change from baseline in ADCS-ADL score at week 24, and
- Change from baseline in ADAS-Cog 11 score at week 24

Main secondary endpoints

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

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

Approved countries

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

Approved sites: 91