Web-Conference

AB8939 Clinical Development Update

October 2025

Disclaimer

This presentation, together with the material set forth herein, does not constitute an offer of securities for sale nor the solicitation of an offer to purchase securities in any jurisdiction. Distribution of such presentation in certain jurisdiction may constitute a breach of applicable laws and regulation. This document is solely for your information on a confidential basis and may not be reproduced, redistributed or sent, in whole or in part, to any other person, including by email or by any other means of electronic communication. In particular, neither this document nor any copy of it may be taken, transmitted or distributed, directly or indirectly, in the United States, Canada, Japan or Australia. The distribution of this document in other jurisdictions may be restricted by law and persons into whose possession this document comes should make themselves aware of the existence of, and observe, any such restrictions. Neither the Company, nor any of its advisors and representatives may accept any responsibility for any loss or damage incurred by the use of this document or the information set forth herein. Neither the Company, nor any of its advisors and representatives takes any undertaking nor guarantees, whether explicitly or tacitly, the accuracy or the completeness of the information set forth herein. Neither this document, nor any part of it, shall form the basis of, or be relied upon in connection with, any contract or commitment whatsoever. In particular, in France, any decision to purchase such securities shall rely solely on the documents that have been reviewed by the Autorité des Marchés Financiers (the “AMF”) and/or published by the Company. This document does not constitute an offer to purchase any financial instruments in the United States. Securities mentioned in this document have not been and will not be registered under the Securities Act of 1933, as amended (the “Securities Act”) and may not be offered or sold in the United States absent registration or an exemption from the registration requirements of the Securities Act. The Company does not intend to register any offering in all or in part or to make a public offer of securities in the United States. This document contains information on the objectives of the Company along with some projections and forward-looking statements. The reader’s attention is drawn to the fact that these objectives may not be fulfilled, and the forecasts or information provided may prove erroneous, and the Company is not required to update such information. Past performance is no guide to future performance and persons needing advice should consult an independent financial adviser.

Participants

AML EXPERTS

NICHOLAS SHORT, MD, PhD
Associate Professor, Department of Leukemia, MD Anderson Cancer Center

OLIVIER HERMINE, MD, PhD
Head of Haematology Department, Paris Necker and Cochin Hospital
Chief Scientific Officer and Chaiman of Scientific Committee
Member of the French Academy of Science

CHRISTIAN AUCLAIR, PharmD, PhD
Professor Emeritus
Cofounder and former director of the PhD program in oncology at Institut Gustave Roussy, Paris-Saclay University
Former director of the biology department and CNRS UMR8113 at Ecole Normale Supérieure de Cachan/Paris-Saclay

AB SCIENCE MANAGEMENT

ALAIN MOUSSY
Co-founder and CEO

LAURENT GUY
Chief Financial Officer

Today's objective it to update the market on the second platform AB8939

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 *Sites initiated*
			Alzheimer's Disease	Phase 3 *Authorized*
		Mast Cell Diseases	Indolent Systemic Mastocytosis	Phase 3 *Initiated*
			Mast Cell Activation Syndrome	Phase 2 *Initiated*
		Blood diseases	Sickle Cell Disease	Phase 2 *To be authorized*
ALDH / Microtubule	AB8939 (IV)	Hematology	Acute Myeloid Leukemia (AML)	Phase 1 *Initiated*
	AB12319 (Oral)	Oncology	Sarcoma, Solid Tumors	Preclinical

(1) Collaborative programme with Assistance Publique - Hôpitaux de Paris (AP-HP) as sponsor, publicly funded as part of the "hospital-university health research " projects under the Future Investment Programme.

Development of AB8939, a new drug targeting

- **Tumor cells (Tubulin disruption)**

And

- **Tumor stem cells (ALDH inhibition)**

Medical Need in AML and AB8939 Mechanism of Actions

Non Clinical Data

Clinical Data - Monotherapy

Clinical Data – Combination Therapy

Next Steps

Concluding Statements from AML Experts

Market Potential and Intellectual Property

In AML, there are several registered drugs but 70% of patients relapse and die creating a persistant unmet medical need. AML remains the most lethal leukemia in humans

AML treatment

1st Line AML

Patients eligible to high dose chemotherapy (HDC)

- **Anthracyclines + cytarabine (3x7)**

Patients with specific genetic profile

- CD33+ : Gemtuzumab ozogamycin
- FLT3- ITD : Midostaurin, Quizartinib
- IDH1-mutated: Ivosidenib
- Menin inhibitors: KMT2Ar/11q23: Revumenib, Ziftomenib

Patients ineligible to high dose chemotherapy

- **Azacitidine Venetoclax**
- Decitabine
- Glasdegib

~ 30%

Complete Remission + Allogeneic stem cell transplant → **Survival long-term**

Refractory / Relapse → **2nd Line (RR/AML)**

- High dose chemotherapy or
- Low dose chemotherapy or
- Azacitidine / venetoclax

- CD33+ : Gemtuzumab ozogamycin
- FLT3- ITD : Gilteritinib
- IDH1-mutated: Ivosidenib
- IDH2-mutated: Enasidenib, Olutasidenib
- Menin inhibitors : KMT2Ar/11q23: Revumenib, Ziftomenib

- Low dose chemotherapy or Best supportive care

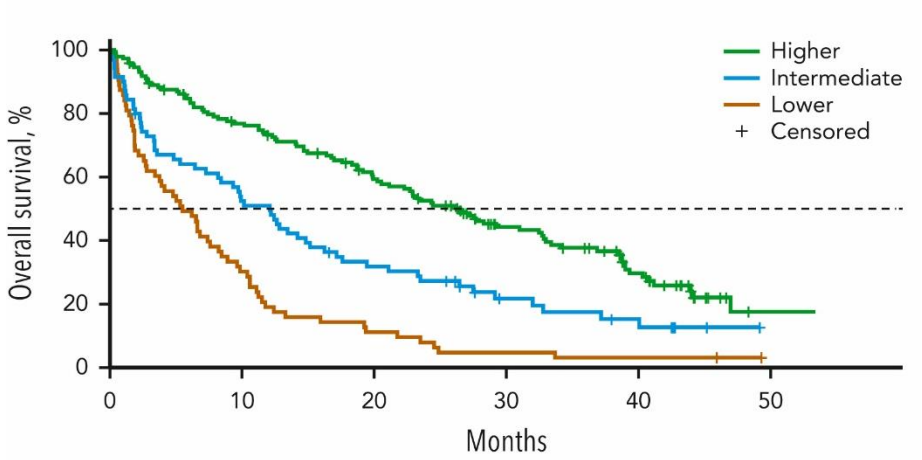
~ 70%

Relapse and death

OS depending on genetic risk profile

AML is a heterogenous disease with outcome highly dependent on genetic factors. TP53 mutation has a very poor prognosis, NRAS, KRAS mutant has a poor prognosis

Outcomes with Venetoclax + Azacitidine by Genetic Risk in treatment-naive AML patients



Patients at Risk

145	107	79	47	25	2
71	36	21	10	6	0
63	19	7	3	2	0

Ven + Aza (N = 279)	n	Events	Median OS, months (95% CI)
Higher benefit	145	96	26.5 (20.2, 32.7)
Intermediate benefit	71	57	12.1 (7.3, 15.2)
Lower benefit	63	61	5.5 (2.8, 7.6)

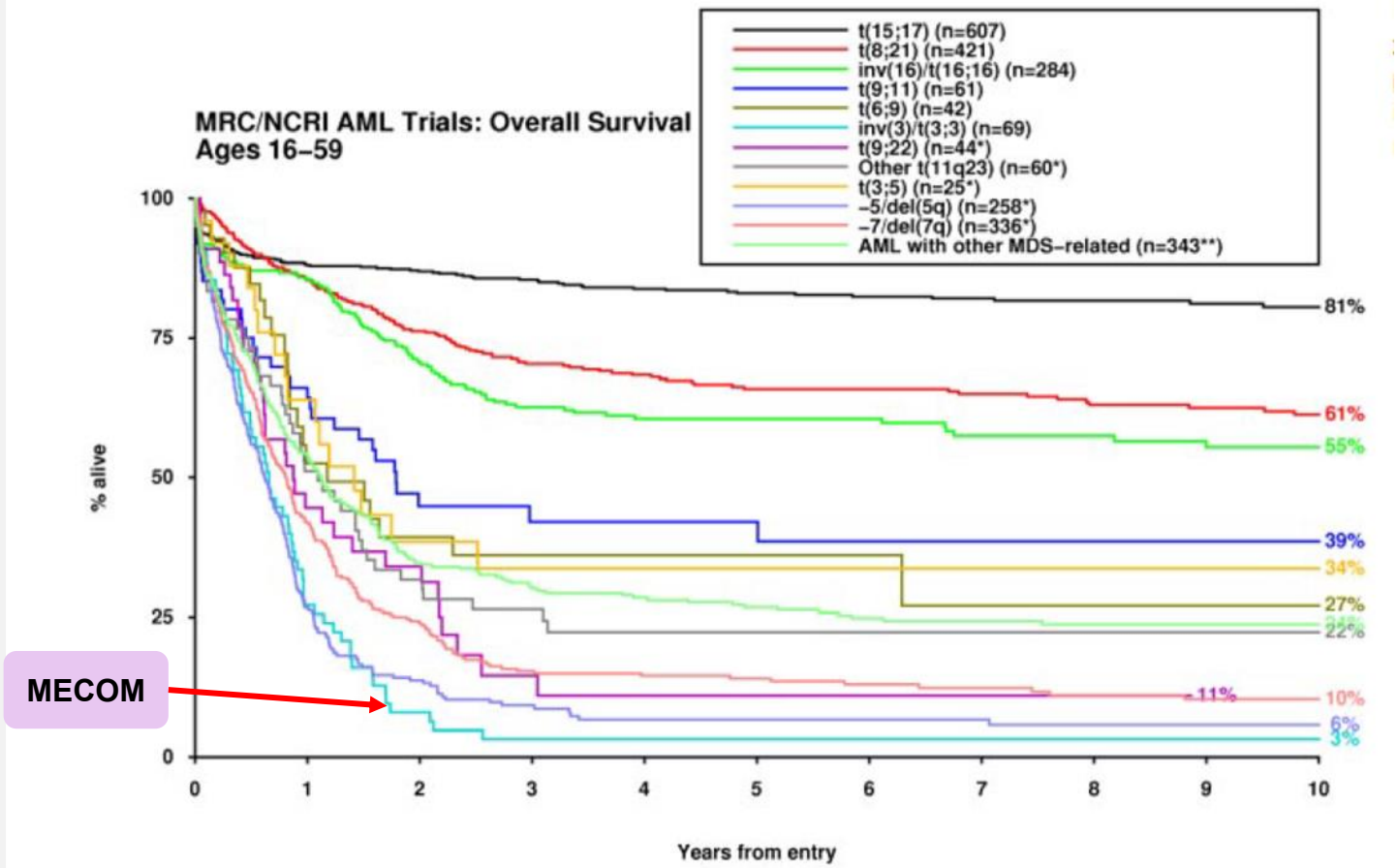
3 prognostic risk signatures, defined by the mutational status of just 4 genes

- **HIGH RISK** **TP53 MUTATION**
- INTERMEDIATE RISK KRAS, NRAS, FLT3 ITD
- LOW RISK others

Döhner et al. Genetic risk stratification and outcomes among treatment-naive patients with AML treated with venetoclax and azacitidine. Blood (2024) 144 (21): 2211–2222.

MECOM has the worst prognosis in AML, with a median OS of 5.5 months [1] in relapsed or refractory setting

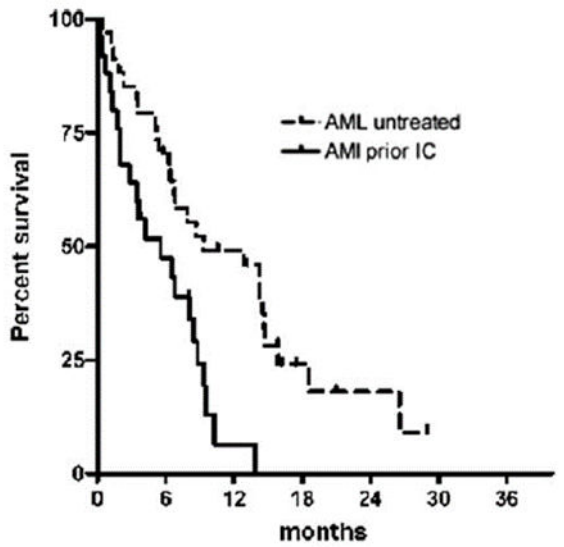
Impact of cytogenetic entities recognized in 2008 WHO classification24 on survival .



*Excluding patients with t(15;17), t(8;21), inv(16), t(9;11), t(6;9),inv(3)/t(3;3).
**Excluding patients with any other abnormalities listed previously.

Grimwade et al. Blood. 2010 Jul 22;116(3):354-65. doi: 10.1182/blood-2009-11-254441.

Survival of AML with 3q abnormality treated with AZA according to prior treatments. Survival was expressed in months and calculated using Kaplan Meier estimate.



AML: acute myeloid leukemia; IC: intensive chemotherapy.

Wanquet et al. Am. J. Hematol. 90:859–863, 2015.

(1) Wanquet A, et al. Azacitidine treatment for patients with myelodysplastic syndrome and acute myeloid leukemia with chromosome 3q abnormalities. Am J Hematol. 2015 Oct;90(10):859-63.

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 kown MoA in cancer but that suffers for known drugs (taxol, taxoter, 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 RESULTS IN THE DECREASE OF TUMOR RECURRENCE

Medical Need in AML and AB8939 Mechanism of Actions

Non Clinical Data

Clinical Data - Monotherapy

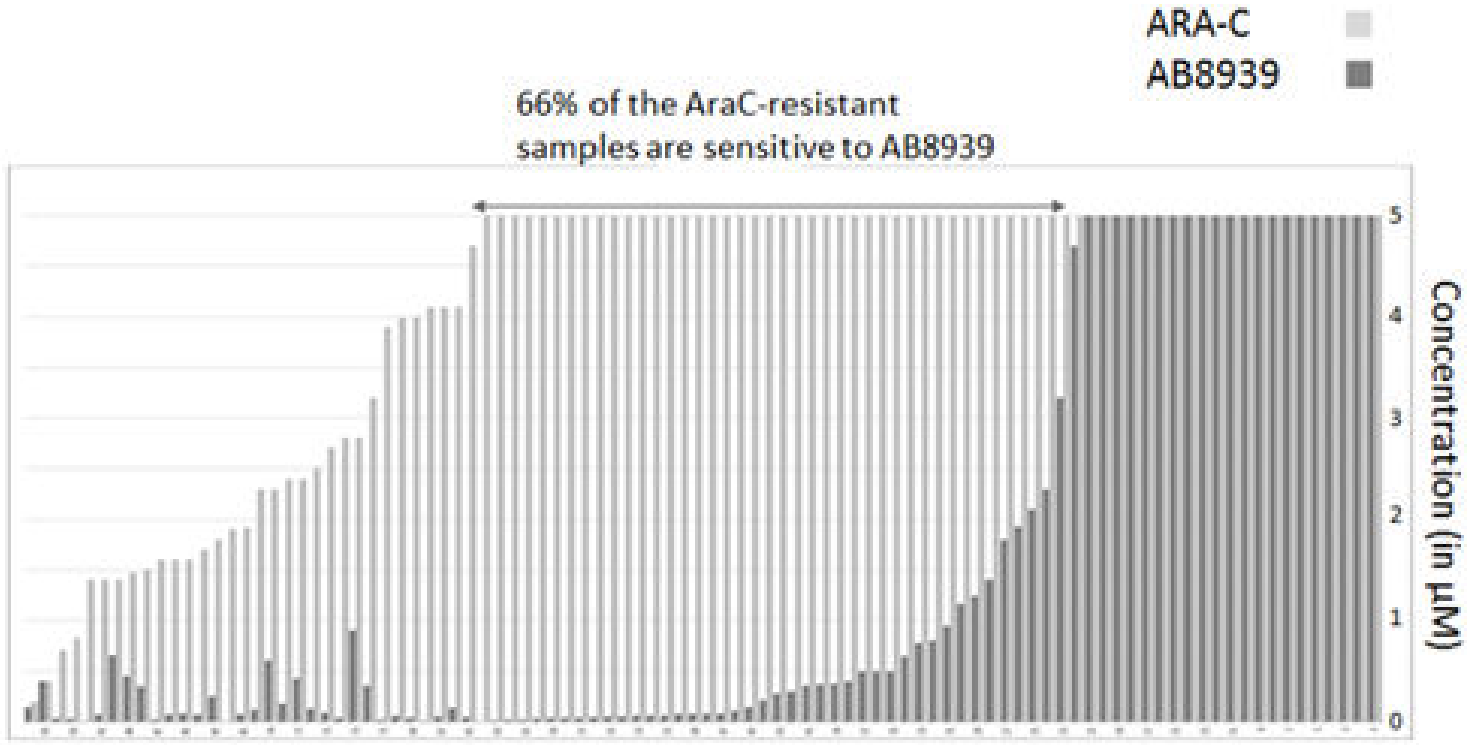
Clinical Data – Combination Therapy

Next Steps

Concluding Statements from AML Experts

Market Potential and Intellectual Property

In vitro, AB8939 has shown activity in Ara-C (cytarabine is one of the standard of care) resistant patients cell lines, including adverse genetic MECOM, TP53 mutated



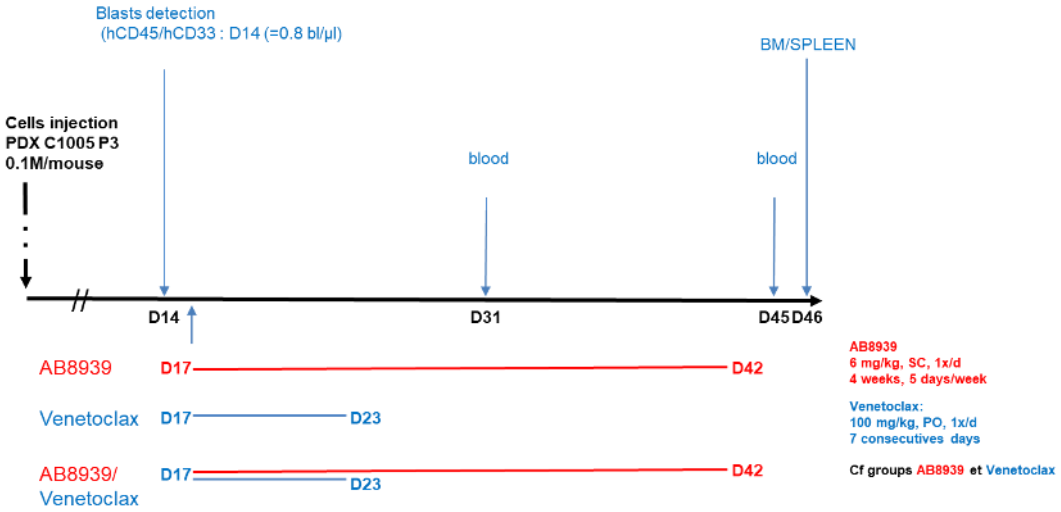
- Among the blasts isolated from a cohort of 99 AML patients, ~70% are resistant to standard AraCytine-based chemotherapy
- Among blasts isolated from this cohort that are resistant to Ara-C, 66% remain sensitive to AB8939, including with adverse genetic (MECOM, TP53 mutated)

Analysis of cell lines responsive to AB8939 shows that AB8939 is effective in cell lines with TP53 muation, MECOM and complex karyotypes, when ARAC and azacitidine is not effective

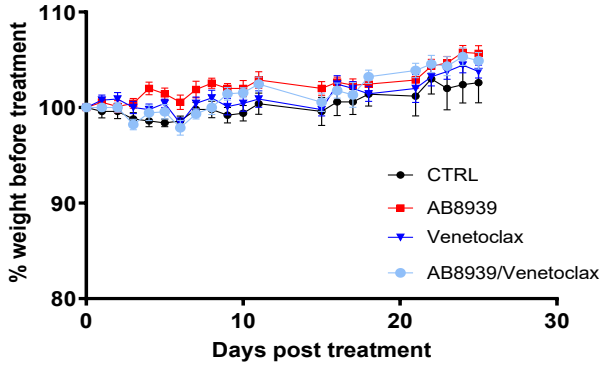
	karyotype	mutations																					Drug sensitibity (IC50 µM)		
NUM_INCLUSION	complex	TP53	FLT3	FLT3-NPM1	IDH1	IDH2	TET2	RAS	RUNx1	IKZF1	BARD1	PTPN11	DNMT3	NPM1	PHF6	NF1	JAK2	WT1	NOTCH2	ETV6	MECOM	RAD21	APC	AB8939	ARAC	AZA
C1012 P3	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0,013	7,9	9,7
C1018	0	0	0	0	0	1	0	0	0	0	0	0	1	0	1	1	0	0	0	0	0	0	0	0,04	13,1	50
C1005-P2	0	0	0	0	0	0	1	1	1	1	1	0	0	0	0	0	0	0	0	0	1	0	0	0,05	4,1	NT
C1022	0	1	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	1	0	0	0	0	0,06	>20	16,7
C1015	0	0	0	0	0	0	0	0	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0,08	>20	39,1
C1024	0	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	1	0	0	1	0	1	0	0,12	2,3	20,4
C1028	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0,38	11,5	41,7
C1021	0	0	0	1	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	0	0	0	0,94	>20	38,5

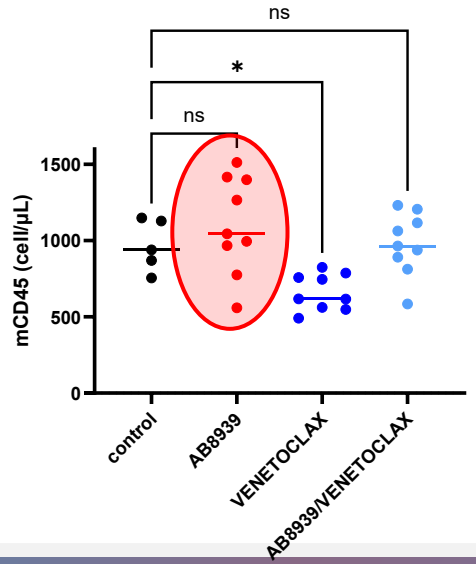
In vivo in mice, in a Mecom grafted PDX model, AB8939 increased survival and has an additive effect in combination with Venetoclax (another standard of care)

A. Study Design



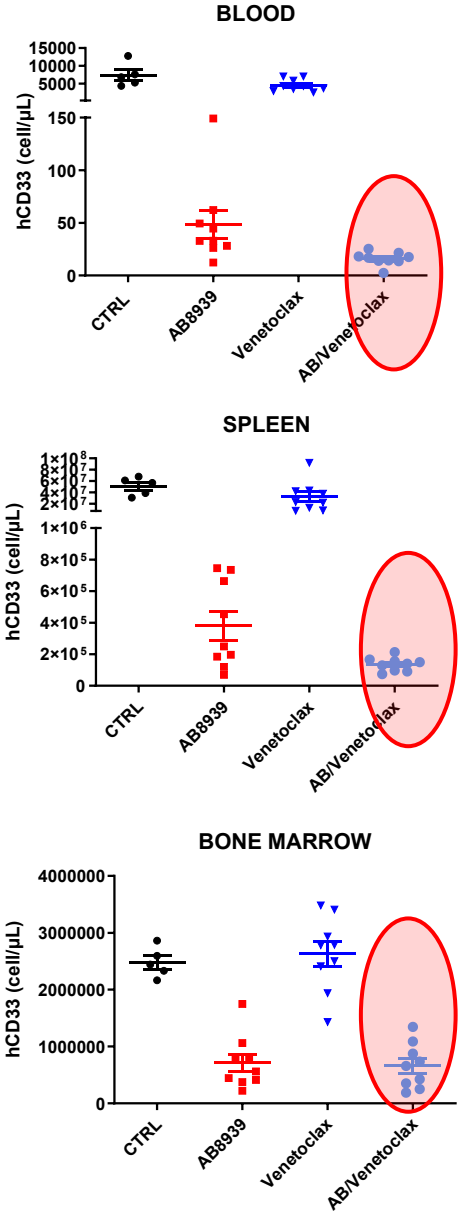
B. AB8939 monotherapy or combined with Venetoclax is well-tolerated: absence of any toxicity (left: weight curves) or hematotoxicity (right: hematopoietic progenitors mCD45)





C.

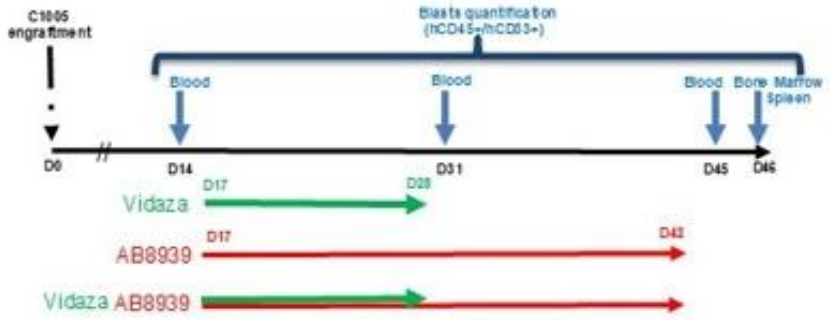
AB8939/ Venetoclax combination allows the clearing of leukemia blasts in blood, spleen and bone marrow without adding toxicities.



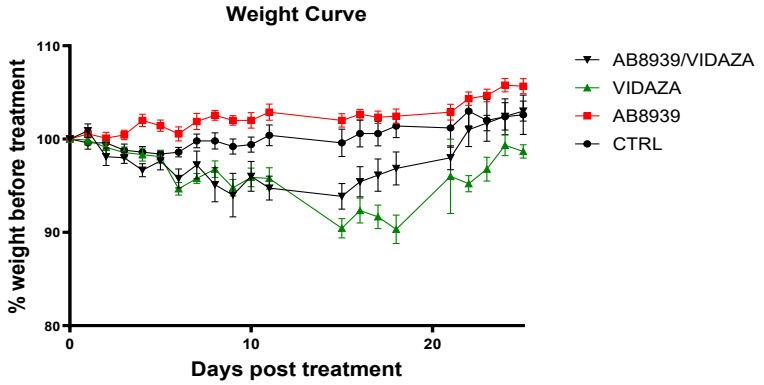
In vivo in mice, in the same Mecom PDX#C1005 model, AB8939 increased survival and has an additive effect in combination with Vidaza (another standard of care)

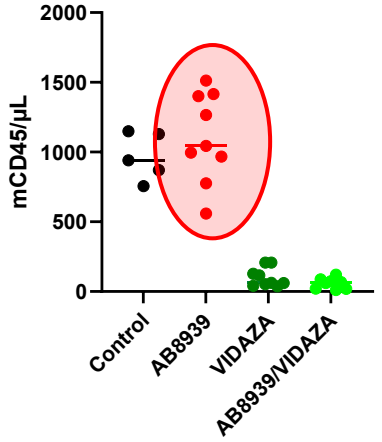
- AB8939/ vidaza combination allows the clearing of leukemia blasts in blood, spleen and bone marrow (C) without adding toxicities (B left panel weight).
- Unlike Vidaza, AB8939 does not induce any hematotoxicity (B right panel mCD45).

A. Study Design

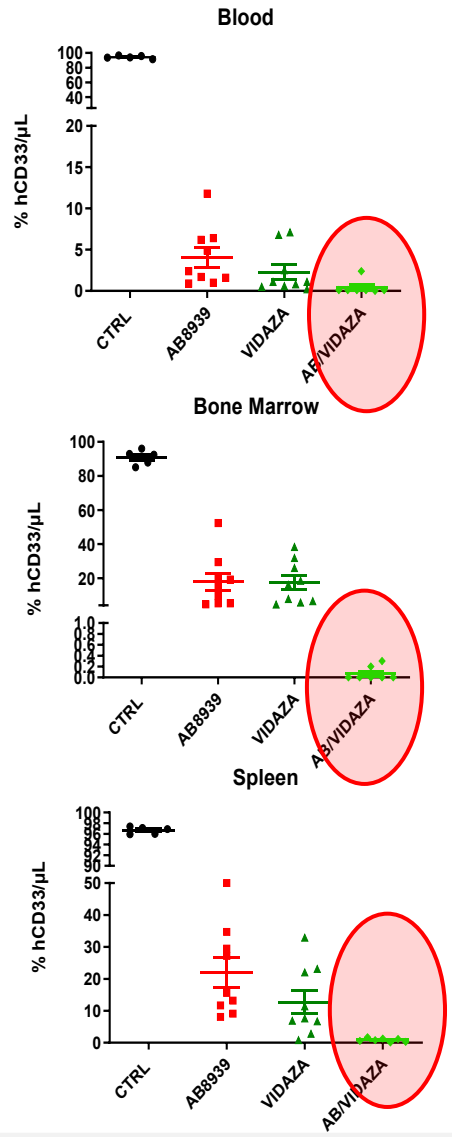


B. AB8939 monotherapy or combined with Azacytidine is well-tolerated: absence of any toxicity (left: weight curves) or hematotoxicity (right: hematopoietic progenitors mCD45)



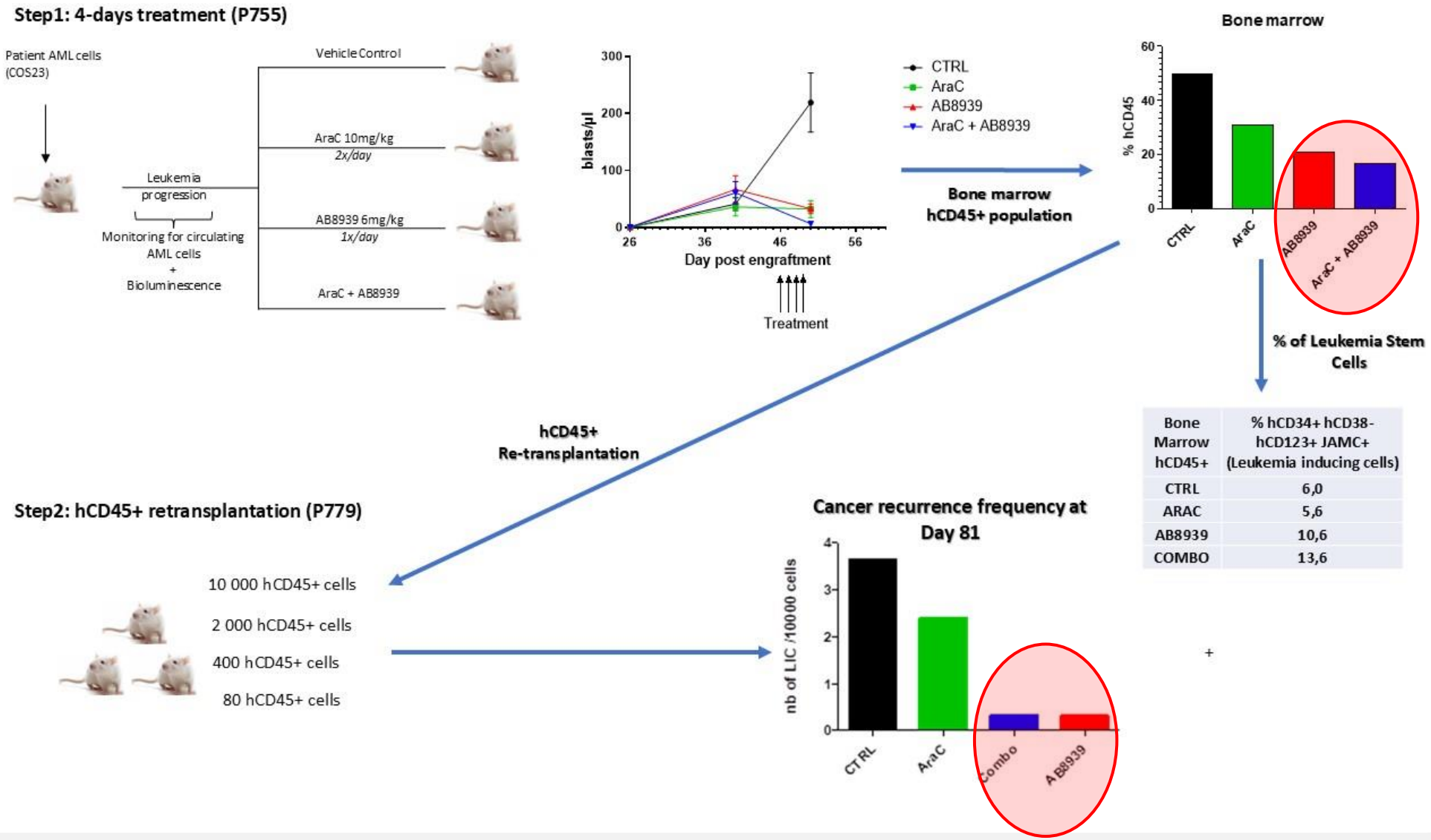


C. Treatment response



In vivo in mice, AB8939 was able to eradicate Leukemia Cancer Stem Cells in a human PDX AML animal model, which is compatible with the ALDH MoA targeting stem cells

- AB8939 reduced the re-occurrence of leukemia following re-transplanted of leukemia cells indicating that AB8939 treatment eradicated both leukemic blast and leukemia cancer stem cells(Step 2).
- AB8939 is believed to kill highly dividing blasts through microtubule disruption while it kills resting cancer Stem cells through inhibition of ALDHs.



Bone Marrow hCD45+	% hCD34+ hCD38- hCD123+ JAMC+ (Leukemia inducing cells)
CTRL	6,0
ARAC	5,6
AB8939	10,6
COMBO	13,6

Medical Need in AML and AB8939 Mechanism of Actions

Non Clinical Data

Clinical Data - Monotherapy

Clinical Data – Combination Therapy

Next Steps

Concluding Statements from AML Experts

Market Potential and Intellectual Property

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

Step 1

MTD* 3 DAYS
AB8939 alone

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

#	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	

Step 2

MTD 14 DAYS
AB8939 alone

Completed
13 patients randomized

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

Step 3

MTD 14 DAYS
AB8939 Combination

On-Going Steps

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

AB8939 in monotherapy has shown activity in MECOM, based on non-clinical data and early clinical data, with long OS benefit

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
  - Historical control shows 14% response rate(1)
- **2 patients with 18 months and 11 months OS benefit**

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%)

(1) G. Richard-Carpentier et al. Characteristics and clinical outcomes of patients with acute myeloid leukemia with inv(3)(q21q26.2) or t(3;3)(q21;q26.2). Haematologica | 108 September 2023.

Medical Need in AML and AB8939 Mechanism of Actions

Non Clinical Data

Clinical Data - Monotherapy

Clinical Data – Combination Therapy

Next Steps

Concluding Statements from AML Experts

Market Potential and Intellectual Property

There is a strong rationale to combine AB8939 with Venetoclax

1. BOTH MOLECULES HAVE LOW HEMATOLOGIC TOXICITY

- This combination is less toxic than azacitidine + venetoclax as first-line treatment for AML

2. BOTH MOLECULES HAVE DIFFERENT AND COMPLEMENTARY TARGETS IN CANCER CELLS

- There is an additive, even synergistic, efficacy potential for the combination, with 3 MoA in one treatment
  - Venetoclax MoA is to inhibit BCL2 pathway, a protein that prevents apoptosis (programmed cell death) in cancer cells. BCL2 is a key factor in AML resistance, as it allows cancer cells to survive despite treatment
  - AB8939 is pro-aopototic, destabilizing microtubule, and would benefit from BCL2 inhibition to optimize apoptosis
  - In addition, AB8939 specifically targets cancer stem cells by inhibiting ALDH, reducing resistance to treatment and limiting the risk of relapse

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

Patient ID	Treatment Line	Prognostic Risk	Response
ES-07-301	3rd Line	NRAS mutant	Complete remission with incomplete hematologic recovery (CRi)
ES-13-301	4th Line	Complex karyotype , mecom	Partial remission at D45 with no circulating blasts
ES-13-302	4th Line	Complex karyotype and TP53 mutation	Partial remission (PR)

Patient 1: AB8939 (14 days, 16 mg/m²) + Venetoclax

Baseline Characteristics

- **Age** 74
- **Prior treatments**
  1) Daunorubicine + cytarabine
  2) Azacitidine
  3) CLN-049-002
- **Cytogenetic risks category** Adverse
- **Cytogenetic, mutations details** RUNX1 with VAF del 86%. mutation RUNX1 with VAF: 9.7% mutations DNMT3A, **NRAS**, PRPF8, PTPN x 3
- **Key Mutations** NRAS

Response

- **Response evaluation D28** Complete remission with incomplete hematologic recovery (CRi)

Cycle 1 (14 days)

	Baseline	D28	D49
BM Blasts	8%	<1%	na
Blood Blasts	0%	0%	0%
Neutrophils	4,4	3,6	5.2
Platelets	59	27	47

- **Venetoclax dosing (mg) :** D1 100, D2 200 and 400 until D8 and decreased to 100mg until D14 because of antifungal treatment
- **Antifungal :** posaconazole

Patient 2: AB8939 (14 days, 16 mg/m²) + Venetoclax

Baseline Characteristics

- **Age** 73
- **Prior treatments** 1) Azacitidine
  2) **Cytarabin + venetoclax ; Refractory**
- **Cytogenetic risks category** Adverse
- **Cytogenetic details** **Complex karyotype:** 45,XX,del(1)(34), add(2)(q22-24),del(5)(q13q33), -7,add(11)(p15),add(11)(q23),der(12)add(12)(p13)del(12)(q14q21-23),-18,-20,add(21)(p11.2),+2mar[3]/46,XX[1]
- **Key Mutation** TET2, **over expression Evi1**
- **FAB Classification** M0 : Undifferentiated acute myeloblastic leukemia

Response

- **Response**
  - Stable disease (SD) at D28
  - Partial remission at D45 with no circulating blasts

Cycle 1 (14 days)

	Baseline	D28	D45
BM Blasts	18%	23%	8%
Blood Blasts	44%	12%	0%
Neutrophils	1,33	1,12	1,32
Platelets	21	15	11

- **Venetoclax dosing (mg) :** D1 50, D2 100 and 200 until D14
- **Antifungal :** Isavuconazole (ongoing during trial) + nystatin (only D1 and after D14 for mouth health care)

Patient 3: AB8939 (14 days, 16 mg/m²) + Venetoclax

Baseline Characteristics

- **Age** 75
- **Prior treatments**
  1) Idarubicin + Cytarabine : Disease progression
  2) FLAG+IDA ; Refractory
  3) **Azacitidine+ Venetoclax ; Refractory**
- **Cytogenetic risks category** Adverse
- **Cytogenetic details** **Complex Karyotype** 44,XY,del(5)(q13q33),add(12)(p11.2),-13,-16,-20,+mar1[5]/ 43,idem,add(1)(q22-24),-7,-18,-mar1,+mar2,+mar3[15
- **Key Mutation** **TP53+**
- **FAB Classification** M2 : Acute myeloblastic leukemia with maturation

Response

- **Response evaluation D28** Partial remission (PR)

Cycle 1 (14 days)

	Baseline	D28
BM Blasts	31%	14%
Blood Blasts	4%	2%
Neutrophils	1,07	0,37
Platelets	13	21

- **Venetoclax dosing (mg) :** D1 50, D2 100 and 200 until D14
- **Antifungal :** Mycostatin (after D14 for mouth health care)

AB8939 + Venetoclax could become a new standard of care in particular for AML patients with adverse genetics

AB8939 + Venetoclax : a potentially new standard of care

- Venetoclax + Vidaza is the standard of care for aged patient not eligible to high dose chemotherapy in Line 1
- Venetoclax + Vidaza is known to be poorly effective in TP53 and NRAS mutant
- AB8939 preliminary data give credit to the hypothesis that the combination AB8939+venetoclax could become a new standard of care
- AB8939+venetoclax is less toxic than other combinations, in particular on hematoxicity

Medical Need in AML and AB8939 Mechanism of Actions

Non Clinical Data

Clinical Data - Monotherapy

Clinical Data – Combination Therapy

Next Steps

Concluding Statements from AML Experts

Market Potential and Intellectual Property

The next step is to finish phase 1 in combination and to launch an expansion study to maximize the chance of success of a registrational study

Implementation of the expansion study

- 15 AML patients eligible to AB8939 + Venetoclax at the right dose

Rationale

- Generate robust preliminary evidence of efficacy in the AML label to support the clinical development plan and trigger partnership agreement

There are 3 possibilities of registrational studies, not mutually exclusive, that we have started to discuss with FDA and EMA

	AB8939 Positioning	Clinical Study Design
1	**AB8939 + VENETOCLAX IN LINE 1 AGED PATIENTS, ADVERSE GENETICS** *TP53mut+NRAS+KRAS+complex K+monosomy 5/7 + Mecom*	▪ (AB8939 + Venetoclax) Vs (Venetoclax + Vidaza) =standard of care) ▪ Primary endpoint: CR+CRi ▪ Secondary endpoint: Survival ▪ 200 patients, randomized 1:1
2	**AB8939 + VENETOCLAX IN LINE 2/3, ALL PATIENTS OR ADVERSE GENETICS** *L2+L3, all patients or adverse genetics*	▪ AB8939+venetoclax Vs best supportive care Vs AB8939 ▪ Primary Endpoint: CR+Cri ▪ Secondary endpoint: survival ▪ 240 patients, randomized 1:1:1
3	**AB8939 IN MECOM** *L2+L3*	▪ AB8939 vs best supportive care ▪ Primary Endpoint: survival ▪ Secondary endpoint: CR+Cri ▪ 80 patients, randomized 1:1

Medical Need in AML and AB8939 Mechanism of Actions

Non Clinical Data

Clinical Data - Monotherapy

Clinical Data – Combination Therapy

Next Steps

Concluding Statements from AML Experts

Market Potential and Intellectual Property

CHRISTIAN AUCLAIR, PharmD, PhD

Professor Emeritus, Cofounder and former director of the PhD program in oncology at Institut Gustave Roussy, Paris-Saclay University

Concluding Pharmacological Remarks

- The AB8939 molecule was initially selected based on two criteria:
  - i) its extreme efficacy in inhibiting proliferative tumor cells, particularly leukemic cell lines, and
  - ii) its extreme efficacy on cells expressing the multidrug resistance phenotype
- Subsequent investigations have shown that AB8939 was able to limit tumor recurrence in experimental models. This remarkable property is due to its ability to inhibit aldehyde dehydrogenase (ALDH), enzymes that provide metabolic support in tumor stem cells.
- This dual property makes AB8939 a molecule of choice for the treatment of refractory acute leukemia by reducing the frequency of relapses.
- Finally, AB8939 is a perfectly tolerated molecule characterized by a lack of bone marrow toxicity.

NICHOLAS SHORT, MD, PhD

Associate Professor, Department of Leukemia, MD Anderson Cancer Center

▪ *TP53*-mutated and *MECOM* rearranged AML are the worst subtypes of AML	• Limited response with standard therapies • Median OS ~6 months, no standard of care
▪ In older adults treated with HMA + venetoclax, *NRAS/KRAS* mutations is also associated with a poor prognosis	• Median OS ~12 months
▪ Single-agent AB8939 showed activity in MECOM	• Response in 2 of 4 patients with MECOM rearrangement • For reference, in other unmet needs (e.g. KMT2Ar leukemias), CR/CRh rate of ~20% sufficient for FDA approval in single-arm study
▪ AB8939 + venetoclax appears safe + and able to generate significant blast reductions in ultra-high-risk AML	• Significant blast reductions in ultra-high-risk AML with TP53-mutated AML after HMA + venetoclax)

- **Early data suggest that AB8939 (monotherapy and/or combination) may have significant activity in the highest risk subtypes of AML**
- **If similar responses rates and safety are observed during trial expansion, AB8939 well-positioned for development in adverse-risk AML in frontline and salvage settings**

Medical Need in AML and AB8939 Mechanism of Actions

Non Clinical Data

Clinical Data - Monotherapy

Clinical Data – Combination Therapy

Next Steps

Concluding Statements from AML Experts

Market Potential and Intellectual Property

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

AB8939 intellectual property rights in AML are secured until 2036 through a ‘composition of matter’ patent and 2041 with extension, and potentially until 2044 in AML with chromosome abnormality through a ‘second medical use’ patent.

Protection	Exclusivity period	Enforcement
Orphan drug status	7-year protection as of FDA approval 10-year protection as of EMA approval	▪ **Granted in the USA** ▪ To be filed with EMA
Composition of Matter patent	Until February 2036 + 5 years extension	▪ **Granted** (United States / Europe / China / Hong Kong / Japan / South Korea / India / Mexico / Israel / Brazil / South Africa / Russia / Australia)
Second Medical Use patent	Until February 2044 (if granted)	▪ **PCT patent application** filed for AML subpopulation with chromosome abnormality