



***CURRENT STATUS AND
PERSPECTIVE FOR 2018***

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Overview of AB Science

AB Science is a late stage development company with rich proprietary drugs pipeline

Company overview

- Founded in 2001 and has 125 employees
- Headquartered in Paris with a subsidiary in the USA (New Jersey)
- Listed since 2010 on Euronext, compartment B in Paris, France (AB.PA)
- IP is 100% owned by AB Science

Key assets

Masitinib

- It is the most selective kinase inhibitor targeting mast cells and macrophages
- Ongoing Phase 3 clinical trials in neurology, oncology and inflammatory diseases
- 2 randomized, controlled, phase 3 clinical trials with positive results in Amyotrophic lateral sclerosis (n=394 patients) and mastocytosis (n=129)
- 1 randomized, controlled, phase 3 clinical trials continued after interim analysis in Progressive forms of multiple sclerosis (n=656) with no need for sample size increase

Other

- New compound (AB8939) to be launched into phase 1/2 in 2018
- Rich pipeline in pre-clinical studies derived from a proprietary library of more than 5,000 compounds synthesized by AB Science
- Objective to launch one new compound into preclinical and clinical development every year

Leadership Team

- **Alain Moussy** (Founder and CEO)
 - Engineer (ENSTA), and MBA Wharton
 - Former strategy consultant at Booz, Allen & Hamilton and former head of Corporate Development at Carrefour.
 - President of AFIRMM, association of mastocytosis patients
- **Jean-Pierre Lehner** (Scientific Senior Vice-President)
 - Former Chief Medical Officer at Sanofi Group
 - Joined AB Science in January 2018
- **Laurent Guy** (CFO)
 - Career in the banking industry (Société Générale and Paribas) and strategy consulting (Accenture).
 - Joined AB Science in 2002
- **Jean-Pierre Kinet** (Scientific Committee)
 - Medical Doctor, Professor of Pathology at Harvard Medical School and Head of Laboratory of Immunology at Beth Israel Deaconess Medical Center (Boston – USA)
- **Olivier Hermine** (Scientific Committee)
 - Medical Doctor, Professor of Hematology at Paris V-René Descartes University
 - Chief of adults Hematology staff, Hospital Necker, Paris
 - Member of French Académie des Sciences
 - Author of 365 international peer-reviewed renown publications



GCP Compliance

New Organization

AB Science has completely reorganized its clinical development department, with the appointment of 6 new highly experienced professionals.

Creation of new position and appointment of Jean-Pierre Lehner as Scientific Senior Vice-President

- Responsibility : Reporting to CEO, in charge of overall clinical development and portfolio benefit risk assessment.
- Background : MD, 30 years of industry experience, with more than 20 years of experience at Sanofi Former Chief Medical Officer and Senior Vice President of Sanofi until 2012. Participated to the registration of 30 drugs throughout his career.

Appointment of new Director of Quality Assurance

- Responsibility : Ensuring adequacy of the Quality Management System and adherence to GCPs.
- Background: 21 years of industry experience, 7 years as CEO of a company specialized in Quality Management, 14 years of experience as of Head of Quality and COO.

Appointment of new Director of Pharmacovigilance and Clinical Safety

- Responsibility : Ensuring continuous evaluation of the safety of drugs into developing and reporting to health authorities
- Background : MD, 20 years of industry experience, 13 years of as Managing Director and Head of Medical Affairs of mid-size CRO, 7 years of experience as Head of Worldwide Corporate Clinical Safety (Serono).

Creation of new position and appointment of new Head of clinical development

- Responsibility : Overseeing the planning and operational execution of the clinical development program
- Background : MD, 20 years experience at Boehringer-Ingelheim, previously Regional Director of Clinical Operation for Asia Pacific and Japan.

Appointment of a new Head of Biometry

- Responsibility : Leading the Biostatistics and Statistical Programming functions within AB Science
- Background : 16 years of experience in quantitative research & data analysis, including 7 years with GSK and Pfizer.

Appointment of a new Director of Data Management and IT systems

- Responsibility : Leading the data-management functions within AB Science and ensuring the 21 CFR part 11 compliance of the systems.
- Background : 15 years experience in Data Management and IT systems. Previously head of R&D IT systems at Menarini.

GCP status

As a result of the preventive and corrective actions that have been implemented, AB Science expects GCP clinical hold to be lifted.

Upgrading of the Pharmacovigilance system

- Re-evaluated safety data
 - Remonitored 80% of all studies, sites, patients visits to ensure that no adverse events are left unreported
 - Recoded and medically assessed all Adverse events in the safety database (60,00 adverse events)
- Restructured the pharmacovigilance department
 - Updated all Standard Operating Procedures (SOPs)
 - Re-organized the department based on three dedicated groups including a PV group (management of safety data), a Clinical Safety Group (management of product safety) and a Compliance and Training Group (review of SOPs, compliance metrics and training)
- Outsourced part of management of new SAE to warrant the accurate collection and timely reporting of SAE:
 - Processing of new SAE (collection, documentation, notification) outsourced since December 2017
 - Medical assessment of SAE remains within AB Science

Upgrading of the Quality Management System (QMS), identified as the root cause of previous inspection findings

- Completed the definition and follow-up of a global CAPA plan by QA department
- Completed the update of SOPs and Working Instructions (WI) of the company with support of external consultants
- Implemented independent audits of AB Science' systems, clinical sites and CROs:
 - External audits of Pharmacovigilance, Clinical Operations, Data Management, Statistics, Clinical Operations and Project Management : Completed
 - External audits of clinical sites : Ongoing (78 audits performed in 13 studies as of February, 2018)
 - External audits of Clinical Research Organizations : Ongoing (6 CROs to be audited by the end of March 2018)

Implementation of corrective actions in each clinical department in order to improve clinical processes

- Completed a roadmap detailing the list of corrective actions to be implemented in each clinical department and communicated it to health authorities
- As of March 2018, most of these corrective actions have been implemented, with pending actions to be implemented by June 2018



Clinical Data

ALS –Masitinib in Amyotrophic Lateral Sclerosis

The mechanism of action of masitinib in ALS is based on the targeting of mast cells and aberrant glial cells and has been published in peer-reviewed journals.

Downloaded from <http://insight.jci.org> on March 9, 2018. <https://doi.org/10.1172/jci.insight.95934>

JCI INSIGHT

RESEARCH ARTICLE

Evidence for mast cells contributing to neuromuscular pathology in an inherited model of ALS

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Evidence indicates that neuroinflammation contributes to motor neuron degeneration in amyotrophic lateral sclerosis (ALS), a fatal neurodegenerative disease leading to progressive muscular paralysis. However, it remains elusive whether inflammatory cells can interact with degenerating distal motor axons, influencing the progressive denervation of neuromuscular junctions (NMJs). By analyzing the muscle extensor digitorum longus (EDL) following paralysis onset in the SOD1^{G93A} rat model, we have observed a massive infiltration and degranulation of mast cells, starting after paralysis onset and correlating with progressive NMJ denervation. Remarkably, mast cells accumulated around degenerating motor axons and NMJs, and were also associated with macrophages. Mast cell accumulation and degranulation in paralytic EDL muscle was prevented by systemic treatment over 15 days with masitinib, a tyrosine kinase inhibitor currently in clinical trials for ALS exhibiting pharmacological activity affecting mast cells and microglia. Masitinib-induced mast cell reduction resulted in a 35% decrease in NMJ denervation and reduced motor deficits as compared with vehicle-treated rats. Masitinib also normalized macrophage infiltration, as well as regressive changes in Schwann cells and capillary networks observed in advanced paralysis. These findings provide evidence for mast cell contribution to distal axonopathy and paralysis progression in ALS, a mechanism that can be therapeutically targeted by masitinib.

Trias et al. *Journal of Neuroinflammation* (2016) 13:177
DOI 10.1186/s12974-016-0620-9

Journal of Neuroinflammation

RESEARCH

Open Access

Post-paralysis tyrosine kinase inhibition with masitinib abrogates neuroinflammation and slows disease progression in inherited amyotrophic lateral sclerosis



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Abstract

Background: In the SOD1^{G93A} mutant rat model of amyotrophic lateral sclerosis (ALS), neuronal death and rapid paralysis progression are associated with the emergence of activated aberrant glial cells that proliferate in the degenerating spinal cord. Whether pharmacological downregulation of such aberrant glial cells will decrease motor neuron death and prolong survival is unknown. We hypothesized that proliferation of aberrant glial cells is dependent on kinase receptor activation, and therefore, the tyrosine kinase inhibitor masitinib (AB1010) could potentially control neuroinflammation in the rat model of ALS.

Methods: The cellular effects of pharmacological inhibition of tyrosine kinases with masitinib were analyzed in cell cultures of microglia isolated from aged symptomatic SOD1^{G93A} rats. To determine whether masitinib prevented the appearance of aberrant glial cells or modified post-paralysis survival, the drug was orally administered at 30 mg/kg/day starting after paralysis onset.

Results: We found that masitinib selectively inhibited the tyrosine kinase receptor colony-stimulating factor 1R (CSF-1R) at nanomolar concentrations. In microglia cultures from symptomatic SOD1^{G93A} spinal cords, masitinib prevented CSF-induced proliferation, cell migration, and the expression of inflammatory mediators. Oral administration of masitinib to SOD1^{G93A} rats starting after paralysis onset decreased the number of aberrant glial cells, microgliosis, and motor neuron pathology in the degenerating spinal cord, relative to vehicle-treated rats. Masitinib treatment initiated 7 days after paralysis onset prolonged post-paralysis survival by 40 %.

Conclusions: These data show that masitinib is capable of controlling microgliosis and the emergence/expansion of aberrant glial cells, thus providing a strong biological rationale for its use to control neuroinflammation in ALS. Remarkably, masitinib significantly prolonged survival when delivered after paralysis onset, an unprecedented effect in preclinical models of ALS, and therefore appears well-suited for treating ALS.

Keywords: ALS, Aberrant glial cells, Neurodegeneration, Masitinib, M-CSF

Masitinib in Amyotrophic Lateral Sclerosis

The phase 2/3 study in ALS was positive. An application for conditional marketing authorization was filed at EMA.

Trial design

- Double blind, placebo controlled, Phase 2/3 study
- Three treatment arms: 4.5mg/kg/day; 3mg/kg/day; placebo + riluzole
- Treatment duration: 48 weeks
- 394 patients enrolled in Europe, Argentina and Canada
- Broad inclusion criteria (Disease duration 3 years ; FVC \geq 60%, no restriction on ALSFRS score at baseline)
- “Normal” progressors (defined as baseline ALSFRS-R progression <1.1 points/month, accounting for 85% of study population) treated with masitinib 4.5 mg/kg/day was the pre-planned population for primary analysis

Primary endpoint

- Change in ALSFRS-R score at week 48

Secondary endpoints

- Progression Free Survival (PFS): time to event analysis measure the earliest event between death and disease progression measured by 9 point decline in ALSFRS-R score
- ALS-AQ40 (questionnaire to assess quality of life) and Forced Vital Capacity and Survival

Final results

Primary analysis : Normal Progressor and Masitinib 4.5 mg/kg/day

- Primary endpoint 27% better Δ ALSFRS-R score (-9.2 vs -12.6 at 48w). (p= 0.0158)
- Secondary endpoints
 - 25% better PFS time (20 vs 16 months) (p=0.0159)
 - 28.5% better ALSAQ-40 score (19.4 vs 27.2) (p= 0.0078)
 - 22% better FVC slope (-26.4 vs -33.9) (p= 0.0296)

Regulatory status

- Filed at EMA for application for conditional marketing authorization was filed at EMA, which implies to perform a confirmatory study in case of registration.

Commentary

“While a direct comparison of the Phase 3 data for edaravone and the Phase 3 data for Masitinib is not possible, a post-hoc analysis that compared patients with similar characteristics at baseline, which represented about 30% of the patients in the Masitinib study, did suggest that Masitinib would be more effective.”

Source CHARDAN, May 24, 2017

“There is a real reason for our enthusiasm because of what we see in our preclinical models. The science behind this is actually very strong. The mice and rats and all these guys, their results are quite strong and what they’re seeing really makes sense.”

Source Dr Angela Genge, May 18, 2017¹

“I’m excited. I think it’s potentially another drug with positive results for people in ALS. And it works by a completely new mechanism of action.”

Source Dr Merrit Cudkowicz, May 18, 2017¹

Note

¹ Matthew Herper. Hopes -- And Questions -- Are Raised By Study Of French Biotech's ALS. Forbes. May 18, 2017. Available at <https://www.forbes.com/sites/matthewherper/2017/05/18/hopes-and-questions-for-als-patients-are-raised-by-study-of-tiny-biotech-firms-medicine/#34bffe4a6840> Accessed June 26, 2017

Masitinib in Amyotrophic Lateral Sclerosis

The design of the confirmatory phase 3 has been optimized based on the results of the first phase 2/3 to increase the treatment effect of masitinib.

Optimization on the design of the confirmatory trial

- Enrolment of patients with shorter disease duration (18 months) and with less severe stage of the disease at baseline defined based on a threshold on each item of the ALSFRS score at baseline (score of at least 2 on each of the 12 ALSFRS score items)
- This optimization is applied to most recent trials in ALS

Impact of optimization on Primary analysis (Change from baseline to week 48 in ALSFRS-R)

- Improvement in change in ALSFRS by +1.6 points (Δ of 5 points versus Δ 3.4)
- In theory, 50 patients per arm are enough to show a significant treatment effect. Confirmatory trial will enroll 150 patients per arm

AB10015 primary analysis
Normal Progressor

Treatment group	N	LS Mean	Diff. of means [95% CI]	p-value
Placebo	102	-12.63	3.4	0.0158
Masitinib 4.5 mg/kg/day	99	-9.24	[0.65;6.13]	

AB10015 primary analysis

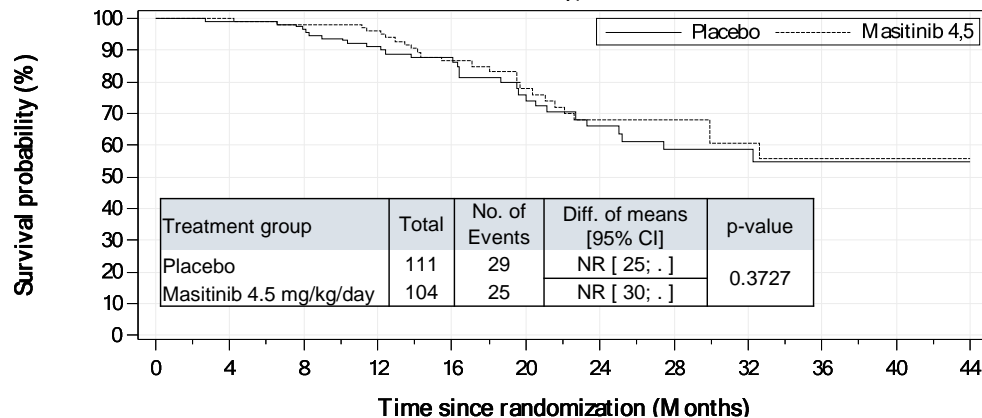
Normal Progressor **with inclusion criteria for confirmatory study**

Treatment group	N	LS Mean	Diff. of means [95% CI]	p-value
Placebo	51	-11.55	5.0	0.0205
Masitinib 4.5 mg/kg/day	39	-6.54	[0.79;9.24]	

Impact of optimization on key secondary analysis (Overall Survival)

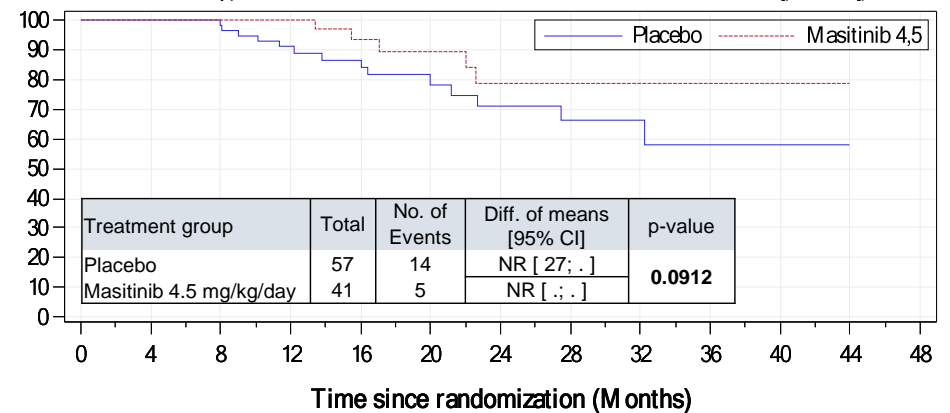
- Trend of survival benefit ($p=0.09$) with 50 patients per arm
- Significant survival benefit ($p<0.05$) can be expected with less than 100 patients per arm. Confirmatory trial will enroll 150 patients per arm

AB10015 overall survival
Normal Progressor



AB10015 overall survival

Normal Progressor **with inclusion criteria for confirmatory study**



Masitinib in Amyotrophic Lateral Sclerosis

IP rights for Masitinib for ALS are secured until 2028 and potentially until 2036 based on a recently filed phase 3 patent.

Protection		Duration of protection	Status
Patent on Composition of matter and PTE	<ul style="list-style-type: none"> Composition of matter has been filed and delivered. It will be further extended until 2028 through patent term extension (PTE) 	Until 2028	Delivered
Synthesis process patent	<ul style="list-style-type: none"> A further protection until 2028 has been achieved through synthesis 'process' patent 	Until 2028	Delivered
Orphan drug status	<ul style="list-style-type: none"> Masitinib has been granted orphan drug designation by both EMA and FDA 	Exclusivity of 7 years for FDA and 10 years for EMA	Delivered
Phase 3 patent	<ul style="list-style-type: none"> Patent filed in March 2016 for treatment of amyotrophic lateral sclerosis 	Until 2036	Filed

Masitinib in Mastocytosis

The phase 3 study in severely symptomatic indolent or smoldering systemic mastocytosis was positive.

Efficacy results in severe indolent systemic mastocytosis (Phase 3; n=129 patients)

Cumulative 75% response rate on the handicaps of pruritus or flushes or depression or asthenia – Primary analysis

M (n=333)	P (n=305)	Difference	p-value*
18.7%	7.48%	11.9%	0.0076

Per Patient 75% response rate 4 Handicaps: Number of patients having response (≥75%) on at least 1 handicap

Overall W8-W24 – Pearson Chi-Square				Overall W8-W24- GEE			
M (n=67)	P (n=62)	Difference	p-value*	M (n=333)	P (n=305)	Difference	p-value
40.3%	24.2%	16.1%	0.0062	26.7%	12.8%	13.9%	0.0212

Per Patient 75% response rate 4 Handicaps: Number of patients having response (≥75%) for each handicap

	Overall W8-W24		
	M	P	Difference
Patient having 2 handicaps at baseline	21.0% (n=19)	0.0% (n=25)	21.0%
Patient having 3 handicaps at baseline	12.5% (n=16)	0.0% (n=18)	12.5%
Patient having 4 handicaps at baseline	16.7% (n=6)	0.0% (n=3)	16.7%

Activity on objective markers of mast cell activation and burden

	Masitinib	Placebo	p-value
Tryptase - Patients with baseline tryptase ≥20 µg/L	46	44	0.0001
Average relative change from baseline Mean±SD	-18.0 ± 21.4	2.2 ± 26.9	
Urticaria Pigmentosa (UP) - Patients with baseline UP	33	36	0.0210
Average relative change from baseline in the Body Surface Area (BSA) covered by UP (Wallace correction)	-12.34 ± 26.41	15.91 ± 59.79	
Darier's sign – Number of patients (baseline)	37	37	0.0187
Response rate for Darier's sign disappearance (Yes/No) in patients with "Darier's sign" at baseline	18.92%	2.70%	

Results were presented at the congress of the European Haematology Association in 2016 and 2017 and published in peer-reviewed journal The Lancet

Lortholary O et al. Masitinib for treatment of severely symptomatic indolent systemic mastocytosis: a randomised, placebo-controlled, phase 3 study

Lancet. 2017 Feb 11;389(10069):612-620. doi: 10.1016/S0140-6736(16)31403-9. Epub 2017 Jan 7

Masitinib in progressive form of Multiple sclerosis

Based on interim analysis, IDMC recommended the continuation of the masitinib phase 3 study in progressive forms of multiple sclerosis with no requirement to increase the study sample size.

Targeted population

- Progressive forms of multiple sclerosis represent around 60% of patients, hence around 400,000 patients in the USA and in the EU alone

Trial design

- Double-blind, randomized, placebo-controlled phase 3 trial designed to assess the safety and efficacy of masitinib in patients with primary progressive or relapse-free secondary progressive multiple sclerosis.
- Treatment period : 96 weeks.
- Primary endpoint : Change in EDSS (Expanded Disability Status Scale) at week 96
- Enrolment : 656 patients.

Patient enrolment

- Enrolment completed

Interim analysis

- Performed once 50% of the study population had reached the 96 weeks treatment duration period.
- IDMC used the conditional power (predictive probability of success) calculation based on the primary endpoint to give its recommendation regarding study continuation and the need for Sample Size Re-estimation or not
- Based on conditional power (CP) calculation using the current sample size, the IDMC recommended the continuation of the study with no re-estimation of sample size, which implies a probability of success of the study above 80% with the current sample size.

Final Analysis

- Planned S1 2019

New compound AB8939

AB Science has developed a new drug candidate targeting microtubules 100x time more potent than doxorubicine and capable of overcoming multidrug resistance.

Drug profile

- **AB8939 is new strong microtubule-destabilising drug.**
 - AB8939 induces, early, irreversible and at as low as 10nM, G2/M cell cycle arrest, while not affecting the actin filaments of the cytoskeleton
 - The microtubules disassembly is observed as early as 1h following treatment with AB8939 100nM
 - AB8939 was assessed against 81 tumour cell lines and has a nanomolar activity against at least 54% of tumour cell lines, especially against haematopoietic cell lines (acute myeloid leukemia, B cell lymphoma, T cell lymphoma and multiple myeloma cell lines)
- **AB8939 is 100 x more potent than doxorubicine and efficient against resistant-doxorubicin human tumor cell lines and is able to overcome multi-drug resistance**
 - AB8939 displayed also a strong anti-proliferative activity against leukemia cell lines HL60 (IC50 of about 6nM) and U937 (IC50 of about 6nM) while doxorubicin is not effective.
 - AB8939 is not transported by the P-gp (P-glycoprotein) *in vitro*, unlike chemotherapies currently used in AML

Targeted Indications

- **Acute Myeloid Leukemia (AML): Patients non-eligible for intensive chemotherapy as second-line, or third-line of treatment**
 - About 20,000 new cases of AML occur annually in the European Union.
 - Overall survival is around 6 months.
 - No drug registered in the targeted claim.

Intellectual Property

- **100% AB Science**



Perspectives for 2018

Clinical Newsflow in 2018

10 important events are expecting in 2018, including regulatory decision in ALS, 1 phase 3 final result, 3 phase 3 interim results, and the launch of a new compound into clinical development.

Timing *		Newsflow	Therapeutic Area
REGULATORY MILESTONE WITH MASITINIB			
Q2 2018	April 2018	EMA decision regarding conditional marketing authorization of masitinib in ALS	Neurology
ONGOING STUDIES WITH MASITINIB			
Q2 2018	April 2018	Phase 3 study in first line prostate cancer - Interim Analysis	Oncology
Q2 2018	April 2018	Phase 2/3 study in 3 rd or 4 th line colorectal cancer - Trend Analysis	Oncology
Q2 2018	April 2018	Phase 2/3 study in 2 nd line of ovarian cancer - Trend Analysis	Oncology
Q2 2018	June 2018	Confirmatory Phase 3 study in pancreatic cancer - Interim Analysis	Oncology
Q2 2018	June 2018	Phase 3 study in severe asthma uncontrolled by oral corticosteroids – Final Analysis	Inflammatory Diseases
Q3 2018		Phase 3 study in Alzheimer's disease - Interim Analysis	Neurology
NEW STUDIES WITH MASITINIB			
Q2 2018	June 2018	Confirmatory Phase 3 study in ALS enrolling US patients - First patient in	Neurology
Q2 2018	June 2018	Confirmatory Phase 3 study in mastocytosis - First patient in	Inflammatory Diseases
NEW STUDY WITH NEW COMPOUND (AB8939)			
Q4 2018		Phase 1/2 study in Acute myeloid leukemia	Oncology

* : Expected timing might change depending on timing of patient enrolment and number of events reached for analysis.

Financial

AB Science has cash until end 2019.

- Cash as of end S1 2017: 49 M€
- Cash as of end S2 2017 : 39 M€
- Cash burn anticipated for 2018 / 2019 : 41 M€
- Equity line in place with CACIB (Credit Agricole Investment Banking) : 2 819 909 Warrant convertible into shares