

AB Science Webconference MASITINIB IN FIRST LINE METASTATIC CASTRATE RESISTANT PROSTATE CANCER (mCRPC)

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EXPERT PANEL





Stéphane Oudard, MD, PhD

Professor of Oncology and Chief of the Oncology Clinical and Translational Research Unit at the Georges Pompidou Hospital in Paris, France. He is professor in Oncology at the René Descartes University, Paris, France.



Theo M. de Reijke, MD, PhD, FEBU Associate Professor at the Amsterdam University Medical Centers, Amsterdam, The Netherlands.



Olivier Hermine, MD, PhD

Professor of Hematology at Paris V-René Descartes University, Chief of adults Hematology staff at Hospital Necker (Paris), member of the French Académie des Sciences

Prostate cancer



Metastatic prostate cancer is still an unmet medical need

- Docetaxel was registered in 2004 by FDA with a label for metastatic prostate cancer progressing after hormono-therapy
- There is no drug registered in combination with Docetaxel
- * The median survival of patients will metastatic prostate cancer is around 2 years and the 5-years survival rate is $30\%^{1}$.

Masitinib Positioning in Prostate Cancer



Masitinib is positioned in combination with docetaxel as first-line treatment of mCRPC eligible to chemotherapy

	Stage of the desease	Main Phase 3	Registered Drug
			Enzalutamide (Xtandi)
1	High-risk non Metastatic Castration Resistant		Abiraterone (Zytiga)
			Apalutamide (Erleada)
		Ipatasertib (Astellas)	Sipuleucel T (Provenge)
		Talazoparib ² (Pfizer)	Abiraterone (Zytiga)
2	Metastatic Castration Resistant Prostate Cancer early stage (before chemotherapy)		Enzalutamide (Xtandi)
			Olaparib ² (Lynparza)
			Rucaparib ² (Rubraca)
2	Metastatic Castration Resistant Prostate	Docetaxel + masitinib	Docetaxel
3	Cancer eligible to chemotherapy	Docetaxel + enzalutamide (Astellas)	
		Atezolizunab (Hoffmann-La Roche)	Abiraterone (Zytiga)
4	Metastatic Castration Resistant Prostate	177Lu-PSMA-617 (Endocyte)	Enzalutamide (Xtandi)
			Cabazitaxel (Jevtana)

Masitinib Profile and Mechanism of Action



Orally-administered kinase inhibitor selectively targeting mast cells and microglia

Masitinib targets mast cells

- Masitinib is a 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 a potent and selective inhibitor of MCSFR-1
- This kinase plays critical roles in the modulation of microglia

Masitinib is a tablet

- Oral route
- Morning and evening

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



Scientific Rationale in Prostate Cancer



Innate immune cells, in particular mast cells and macrophages, are critical components of the tumor microenvironment, promoting angiogenesis and tumor growth, and also contributing to tumorigenesis by suppression of the immune response

- The amount of mast cells (MCs) infiltration into human prostate cancer correlates with prognosis, with lower number of MCs in biopsy specimen leading to better prognosis [1;2].
- There is a positive correlation between MCs infiltrated and tumor microvessel density, indicating a stimulating role of MCs in tumourigenesis [3]
- MCs are essential for the outgrowth of early-stage tumors but not essential at later stage [4]
- MCs increase prostate cancer chemotherapy resistance via modulation p38/p53/p21 signaling [5]
- Prostate cancer bone tumors strongly expressed c-kit [6]
- M2 macrophages promote prostate cancer progression and M1 macrophages may also be associated with poor prognosis [7]

References

[1] Johansson A, et al. Mast cells are novel independent prognostic markers in prostate cancer and represent a target for therapy. Am J Pathol 2010;177:1031–41. [2] Nonomura N, et al. Decreased number of mast cells infiltrating into needle biopsy specimens leads to a better prognosis of prostate cancer. British Journal of Cancer (2007) 97, 952 – 956. [3] Stawerski P, et al. Augmented mast cell infiltration and microvessel density in prostate cancer. Contemp Oncol (Pozn) 2013; 17 (4): 378–382. [4] Colombo MP, et al. The Dark Side of Mast Cell–Targeted Therapy in Prostate Cancer. *Cancer Res* 2012;72:831-835. [5] Lei Li, et al. Infiltrating mast cells increase prostate cancer chemotherapy and radiotherapy resistances via modulation of p38/p53/p21 and ATM signals. Oncotarget, Advance Publications 2015. [6] Wiesner C, et al. C-Kit and Its Ligand Stem Cell Factor: Potential Contribution to Prostate Cancer. Bone Metastasis. Neoplasia (2008) 10, 996–1003. [7] Wu Z, et al. The Landscape of Immune Cells Infiltrating in Prostate Cancer. The Landscape of Immune Cells Infiltrating in Prostate Cancer. The Landscape of Immune Cells Infiltrating in Prostate Cancer. The Landscape of Immune Cells Infiltrating in Prostate Cancer. The Landscape of Immune Cells Infiltrating in Prostate Cancer. The Landscape of Immune Cells Infiltrating in Prostate Cancer. The Landscape of Immune Cells Infiltrating in Prostate Cancer. The Landscape of Immune Cells Infiltrating in Prostate Cancer. The Landscape of Immune Cells Infiltrating in Prostate Cancer. The Landscape of Immune Cells Infiltrating in Prostate Cancer. The Landscape of Immune Cells Infiltrating in Prostate Cancer. The Landscape of Immune Cells Infiltrating in Prostate Cancer. The Landscape of Immune Cells Infiltrating in Prostate Cancer. The Landscape of Immune Cells Infiltrating in Prostate Cancer. The Landscape of Immune Cells Infiltrating in Prostate Cancer. The Landscape of Immune Cells Infiltrating in Prostate Cancer. The Landscape of Immune Cells Infiltrating in Prostate Can

Scientific Rationale in Prostate Cancer



Masitinib has no direct "tumor killer" general activity but has shown efficacy on tumor proliferation in vivo, mediated through the tumor micro-environment

Melanoma Carcinoma **B16** Lung 80000 RLUIS 150000 100000 40000 묻 LLCI 告 200000 50000 NEO TURA an week Adenocarcinoma Breast 500000 MDA-MB-231 RLU/S 400000 300000 200000-100000-In vitro, in tumors which are not driven by c - kit or other relevant kinases tyrosine kinase, inhibition by masitinib did not have any direct effect on tumor proliferation

No direct effect on tumor cells in vitro...

...but decreases tumor volume growth in vivo



In vivo, the observed anti-tumor activity is therefore mediated through the tumor microenvironment.

Masitinib Clinical Development Plan in Prostate Cancer



The development program prostate cancer is comprised of AB07004 proof of concept study, and AB12003 phase 3 study

Phase	Study code	Design	Population	Dosing	Primary endpoint	Patient target
1/2	AB07004	Prospective, open- label, 2-parallel group,	metastatic Hormone Refractory Prostate Cancer (HRPC) in progression after first line of treatment	Masitinib 9.0mg/kg/d + Docetaxel Masitinib 9.0mg/kg/d + Gemcitabine	Overall Survival	34
3	AB12003 (NCT03761225)	Prospective, double- blind, placebo- controlled, parallel groups study	First Line Metastatic Castrate Resistant Prostate Cancer (mCRPC)	Masitinib 6.0mg/kg/d + Docetaxel	Progression Free Survival	580

AB07004 study results in Prostate Cancer



Proof of concept study supported the combination of masitinib with docetaxel in mRCPC

Treatment group	Median OS estimation with Kaplan Meier method (months)	Lower bound of the median one-sided Cl (months)	Alpha set in the protocol
Masitinib + Docetaxel	18.4	17.8	75% (1
Masitinib + Gemcitabine	13.4	8.5	75% CI

AB12003 – Study Design



Study AB12003 evaluated masitinib 6.0 mg/kg/day in combination with docetaxel versus docetaxel alone in first-line treatment of mCRPC

Design:

A prospective, multicenter, randomized, double blind, placebo-controlled, 2-parallel groups, phase 3 study

Groups:

- Masitinib 6.0 mg/kg/day + Docetaxel at 75 mg/m² versus Placebo + Docetaxel at 75 mg/m²
- Randomisation 1:1

Main inclusion criteria

- Patient with histologically or cytologically confirmed metastatic Castrate Resistant Prostate Cancer (medical or surgical castration: androgens deprivation by GnHR agonist or antagonist or patient with surgical castration; hormonal castration confirmed biologically (testosterone < 0.5ng/ml) with one of the following criteria:
 - Pre-treated with abiraterone with progressed disease documented, OR
 - With indication for initiating docetaxel administration (e.g., widespread visceral disease or rapidly progressive disease).
- 2) Patient with evidence of progressive metastatic disease.
- 3) Patient with ECOG ≤ 1

Regulatory : Study conducted under IND from FDA

Patient Enrolment: Patients were enrolled in 16 countries and 67 sites, including 9 countries from Western Europe and North America

AB12003 – Primary Endpoint



The primary endpoint was PFS measured with PCWG2 definition, which is based on the earliest between radiographic progression, PSA progression, Pain progression, or death

Primary endpoint : Progression Free Survival (PFS) as per PCWG2

Variable	Assessment
Radiographic progression*	 Soft-tissue : Use RECIST 1.1
(Bone and Soft tissue lesion)	 Bone : 2 new lesions, confirmed 12 weeks apart
DCA prograccion	Increase ≥ 25% and ≥ 2ng/mL above nadir
PSA progression	 Confirmed 12 weeks apart
Symptom Progression	■ Increase in Present Pain Intensity (PPI) ≥ 25% from baseline
(pain and analgesic)	 Confirmed 12 weeks apart
Death	

* Soft tissue and bone progression are calculated at the time of first progression since most investigators discontinued patients after first progression for ethical reasons

Population analysed : mITT population, defined as all randomized patients with a least one treatment intake

AB12003 – Sensitivity and Secondary Analyses



Primary efficacy analysis was completed with sensitivity analyses of the primary endpoint and several secondary analyses

PFS Sensitivity analyses

Sensitivity analyses were performed for primary analysis with:

- Stratified log rank
- Stratified cox model
- ITT population
- PP population
- Investigator assessment for PCWG2 PFS

Secondary analyses

- Progression Free Survival (PFS)
 - o based on Radiographic progression
 - based on Radiographic or PSA progression
- Time to tumor progression (TTP)
 - based on Radiographic progression
 - o based on Radiographic or PSA progression
 - based on Radiographic or PSA progression or Pain
- Overall Survival
- Response rate
- Quality of Life
- Pain

AB12003 – Population Tested & Control of Alpha Risk



The study pre-specified the overall population and a targeted subgroup defined as patients with ALP \leq 250 IU/mI at baseline

- The study tested the success of the primary endpoint in two populations
 - Overall population
 - Targeted subgroup of interest based on ALP ≤ 250 IU/ml
- Targeted subgroup of interest
 - Patients most likely to respond to masitinib is predefined as patients with lower extent of metastases based on biologic biomarker (ALP ≤ 250 IU/ml)
 - It was assumed that approximately that 67% of the overall population will constitute of the targeted subgroup

Control of Alpha Risk :



Interim analysis

- Used Haybittle-Peto spending Function
- IDMC recommended to continue study in the targeted subgroup with sample size increase
- IDMC recommended to stop enrolment in other patients not in the targeted subgroup



AB12003 – Sample Size



Primary analysis was based on 450 patients in the targeted subgroup and 712 patients in the overall population



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AB12003 – Baseline Characteristics – Overall Population



Baseline characteristics were balanced for age but possibly disbalanced in favor of the control arm for PSA and Chromogranin A

ITT	Masitinib + Docetaxel (N = 356)	Placebo + Docetaxel (N = 358)
Age (Years)		
Mean (std)	67.0 (7.76)	66.5 (7.49)
Median	67.5	66.0
<66 years	157 (44.1)	162 (45.3)
66-75 years	144 (40.4)	150 (41.9)
>75 years	55 (15.4)	46 (12.8)
ECOG [n (%)]		
0	113 (31.7)	118 (33.0)
1	243 (68.3)	240 (67.0)
Visceral Disease [n (%	⁄o)]	
No	231 (64.9)	224 (62.6)
Yes	125 (35.1)	134 (37.4)
Gleason score		
Mean (std)	7.5 (1.36)	7.5 (1.32)
Median	8.0	8.0
Halabi Score		
Mean (std)	29.0 (13.83)	27.9 (10.79)
Median	25.3	25.4

ITT	Masitinib + Docetaxel (N = 356)	Placebo + Docetaxel (N = 358)
LDH		
Mean (std)	399.3 (406.4)	365.0 (303.3)
Median	314	282
Alkaline phosphat	ase	
Mean (std)	406.9 (682.0)	344.0 (444.1)
Median	198	204
Level of serum PSA	A at Baseline (ng/ml)	
Mean (std)	235.5 (545.6)	202.7 (338.1)
Median	71.3	84.8
Chromogranin A-(CgA) at Baseline (ng/ml)	
Mean (std)	158.6 (306.3)	113.4 (129.6)
Median	80.6	69.4

AB12003 – Baseline Characteristics – Targeted Subgroup



Baseline characteristics were balanced for age but possibly disbalanced in favor of the control arm for PSA and Chromogranin A

ITT	Masitinib + Docetaxel (N = 226)	Placebo + Docetaxel (N = 225)
Age (Years)		
Mean (std)	67.0 (7.66)	66.8 (7.50)
Median	67.5	66.0
<66 years	98 (43.4)	100 (44.4)
66-75 years	93 (41.2)	95 (42.2)
>75 years	35 (15.5)	30 (13.3)
ECOG [n (%)]		
0	89 (39.4)	97 (43.1)
1	137 (60.6)	128 (56.9)
Visceral Disease [n (%	(o)]	
No	137 (60.6)	139 (61.8)
Yes	89 (39.4)	86 (38.2)
Gleason score		
Mean (std)	7.5 (1.30)	7.6 (1.34)
Median	8.0	8.0
Halabi Score		
Mean (std)	22.4 (5.68)	22.9 (5.90)
Median	22.1	22.3

ITT	Masitinib + Docetaxel (N = 226)	Placebo + Docetaxel (N = 225)			
LDH					
Mean (std)	333.3 (371.5)	311.3 (247.7)			
Median	256	254			
Alkaline phosphata	se				
Mean (std)	142.5 (56.27)	140.2 (58.64)			
Median	129	130			
Level of serum PSA at Baseline (ng/ml)					
Mean (std)	182.1 (451.5)	170.2 (326.2)			
Median	46.2	63.5			
Chromogranin A-(CgA) at Baseline (ng/ml)					
Mean (std)	143.8 (168.8)	105.2 (127.5)			
Median	82.0	55.2			

AB12003 – PFS Primary analysis (ALP ≤ 250)



The study met its primary analysis in the pre-specified targeted subgroup (patients with ALP ≤ 250 IU/mI), demonstrating a statistically significant increase in PFS (p=0.0272)

Treatment	Patients	No. of Events	Percentage censored	Median [96.1% CI]	Log Rank p-value	Hazard Ratio (96.1% CI)
Masitinib 6.0 mg/kg/day + Docetaxel	225	191	15.11	6.3 [5.6;7.6]	0.0272	0 70 (0 64 0 07)
Placebo + Docetaxel	225	209	7.11	5.4 [4.6;6.0]	0.0272	0.79 (0.64,0.97)

Analysis of Progression Free Survival (PFS) (Overall PFS) - Targeted sub-population

Kaplan Meier Analysis of Overall PFS – Targeted sub-population



AB12003 – Sensitivity analyses of Primary analysis (ALP ≤ 250)



Sensitivity analysis of the primary endpoint were all consistent with a benefit ranging from 21% to 24%

Analysis	Treatment	Patient	No. of Events	Median [95% CI]	Log Rank p-value	Hazard Ratio (95% CI)
Stratified log raph	Masitinib 6.0 + Docetaxel	225	191	6.3 [5.6;7.6]	0.0087	0.79 (0.64,0.97)
Stratilieu log fallk	Placebo + Docetaxel	225	209	5.4 [4.6;6.0]		

Analysis	Treatment	Patient	No. of Events	Median [95% CI]	Log Rank p-value	Hazard Ratio (95% CI)
ITT population	Masitinib 6.0 + Docetaxel	226	191	6.3 [5.6;7.6]	0.0272	0 70 (0 65 0 96)
	Placebo + Docetaxel	225	209	5.4 [4.6;6.0]	0.0272	0.79 (0.05,0.90)

Analysis	Treatment	Patient	No. of Events	Median [95% CI]	Log Rank p-value	Hazard Ratio (95% CI)
DD population	Masitinib 6.0 + Docetaxel	223	190	6.2 [5.6;7.6]	0.0250	0.76 (0.64.0.06)
PP population	Placebo + Docetaxel	221	205	5.4 [4.8;6.0]	0.0258	0.76 (0.64.0.96)

Analysis	Treatment	Patient	No. of Events	Median [95% CI]	Log Rank p-value	Hazard Ratio (95% CI)
Investigator assessment	Masitinib 6.0 + Docetaxel	225	192	6.3 [5.6;7.6]	0.0244	0.70 (0.65.0.06)
for PCWG2 PFS	Placebo + Docetaxel	225	210	4.9 [4.6;6.0]	0.0244	0.79 (0.65,0.96)

AB12003 – PFS Rate (ALP ≤ 250)



The percentage of non-progressors was in favor of masitinib at all timepoints in the targeted subgroup

Non progression (%)	Month 6	Month 12	Month 18	Month 24	Month 30	Month 36	Month 42	Month 48
M (n=225)	56.4	32.0	27.6	23.1	17.3	16.4	15.6	15.1
P (n=225)	45.3	19.6	14.6	12.0	10.2	8.9	8.0	7.6
p-Value	0.0235	0.0035	0.0011	0.0028	0.0396	0.0227	0.0186	0.0167

Analysis of Progression Free Survival (PFS) (Overall PFS) - Targeted sub-population

AB12003 – PFS Analysis - Overall Population



There was no PFS benefit in the overall population

Treatment	Patients	No. of Events	Percentage censored	Median [95% CI]	Log Rank p-value	Hazard Ratio (95% CI)
Masitinib 6.0 mg/kg/day + Docetaxel	225	313	11.83	5.7 [4.9;6.3]	0 2077	0.04 (0.81.1.10)
Placebo + Docetaxel	225	335	6.16	5.4 [4.9;5.9]	0.2977	0.94 (0.01,1.10)

Analysis of Progression Free Survival (PFS) (Overall PFS) – Overall Population

Kaplan Meier Analysis of Overall PFS – Overall Population



AB12003 – PFS Sensitivity analyses on ALP



The lower the ALP level, the greater the masitinib treatment effect, in line with greater treatment effect expected in early metastatic phase

ALP threshold	Treatment	Patients	Percentage subjects	No. of Events	Percentage censored	Median [95% CI]	Median Diference	Log Rank p-value	Hazard Ratio (95% CI)	Risk Benefit		
<250	Masitinib 6.0 + Docetaxel	225	63.38	191	15.11	6.3 [5.6;7.6]	0.0	0 0272	0.79	010/		
\$250	Placebo + Docetaxel	225	63.03	209	7.11	5.4 [4.6;6.0]	0.9	0.0272	(0.65,0.96)	2170		
<200	Masitinib 6.0 + Docetaxel	178	50.14	148	16.85	6.9 [5.8, 7.9]	1 2	0.01 2 6	0 73 [0 58 0 91]	770/2		
5200	Placebo + Docetaxel	176	49.3	164	6.82	5.6 [4.6, 6.2]	1.3	0.0120	0.75 [0.56, 0.91]	2770		
<150	Masitinib 6.0 + Docetaxel	134	37.75	108	19.4	6.9 [5.6, 8.5]	1 /	0.0008	0.63 [0.48, 0.82]	27 0/2		
2150	Placebo + Docetaxel	133	37.25	122	8.27	5.5 [4.6, 6.9]	1.4	0.0000	0.05 [0.40, 0.02]] 5770		
<100	Masitinib 6.0 + Docetaxel	59	16.62	42	28.81	9.0 [7.6, 10.7]	0.1	0.00 22	0 53 [0 35 0 70]	47 0/-		
2100	Placebo + Docetaxel	72	20.17	66	8.33	6.9 [5.5, 7.9]	2.1	2.1	2.1	0.0022	0.00 [0.00, 0.79]	4/%

Analysis of Progression Free Survival (PFS) (Overall PFS) based on ALP level

AB12003 – Overall Survival (ALP ≤ 250)

There was not benefit on overall survival in the targeted subgroup

Treatment	Patients	No. of Events	Percentage censored	Median [95% CI]	Log Rank p-value	Hazard Ratio (95% CI)
Masitinib 6.0 mg/kg/day + Docetaxel	225	152	32.44	25.4 [22; 28]	0.0572	1.02 (0.82.1.20)
Placebo + Docetaxel	225	155	31.11	24.0 [20; 27]	0.9573	1.03 (0.82,1.29)

Analysis of Overall Survival (OS) – Targeted sub-population

Kaplan Meier Analysis of Overall Survival – Targeted sub-population



- OS may have been impacted by hormonotherapy that is registered after Docetaxel
- There was no recording of treatments taken after progression with Docetaxel in this study

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AB12003 – TTP Analysis on Overall Population



In the overall population, there was a statistically significant increase in TTP (+4 months, p=0.0493), proving that masitinib is active against prostate metastases

Treatment	Patients	No. of Events	Percentage censored	Median [95% CI]	Log Rank p-value	Hazard Ratio (95% CI)
Masitinib 6.0 mg/kg/day + Docetaxel	355	63	82.25	20.5 [16; 44]	0.0402	0.74 (0.52.1.02)
Placebo + Docetaxel	357	87	75.63	16.5 [14; 20]	0.0495	0.74 (0.33,1.02)

Analysis of Time to Radiographic Progression – Overall Population

Kaplan Meier Analysis of Time to Radiographic Progression – Overall Population



AB12003 – Safety Overview

The safety of masitinib was consistent with its known tolerability profile

	Masitinib + Docetaxel (N = 355)	Placebo + Docetaxel (N = 357)
At least one AE	343 (96.6)	344 (96.4)
Fatal AE	19 (5.4)	22 (6.2)
At least one serious AE (non-fatal)	98 (27.6)	67 (18.8)
At least one Severe AE	281 (79.2)	261 (73.1)
At least one AE leading to study treatment permanent discontinuation excluding Fatal AE	94 (26.5)	66 (18.5)

Summary of Adverse Events

Intellectual Property



A new patent was filed based on results from study AB12003, which would permit AB Science to retain exclusive rights on the use of masitinib in Prostate Cancer until 2042

Protection	Item	Duration of protection	Status
Patent on composition of matter and PTE	Patent on 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	A further protection until 2028 has been achieved through synthesis 'process' patent	Until 2028	Delivered
Phase 2/3 'Method of use' patents	New patent based on results from study AB12003	Until 2042	Provision patent filed

Market



The market potential is significant with 125,000 eligible patients in EU and US

Indication	Prevalence US Patients EU Patients		Estimated number of potential eligible patients		Annual cost of drugs registered in
			the indication		
Prostate Cancer	113 / 100,000 ¹		50.000	75.000	Sipuleucel T (Provenge) : 93,000 USD Enzalutamide (Xtandi) : 90,000 USD Rucaparib (Rubraca) : 72,000 USD
Target population – mCRPC eligible to chemotherapy*	13%²		50,000	75,000	Abiraterone (Zytiga) :60,000 USDOlaparib (Lynparza) :60,000 USDCabazitaxel (Jevtana) :48,000 USDDocetaxel :~300 USD

* : expressed as percentage of Prostate Cancer population

Source :

Population : <u>https://data.worldbank.org/indicator/SP.POP.TOTL</u> and <u>https://ec.europa.eu/eurostat/web/population-demography-migration-projections/population-data/main-tables</u>

1. National Cancer Institute, Prostate Cancer statistics

2. Scher 2015 – PLoSONE - Symptomatic mCRPC that has not been treated with or not progressed on chemotherapy

