



Investor Update

Clinical Data from ASCO and EHA

June 11, 2009

Overview



- Oncology focused biopharmaceutical company
 - New small molecule drugs
 - Addressing unmet medical needs in leukemia
- Late-stage product pipeline
 - Submission for drug approval in US and EU in 2009
 - Potential approval and launch in US in early 2010 and EU in Q3 2010
- Strong board and senior management team



Leadership Team

Management

Greg Collier, PhD*

Adam Craig, MD, PhD, MBA

James Campbell, PhD, MBA

Luana Staiger, BS

Don Joseph, JD

Chief Executive Officer and Managing Director

Senior Vice President and Chief Medical Officer

Chief Financial Officer and Chief Operating Officer

Vice President of Regulatory Affairs

Head of Corporate Development

Board of Directors

Brett Heading, LLB (Chairman)

Dan Janney, BA, MBA

Geoff Brooke, MBBS, MBA

Elmer Schnee, BCom Mkting

George Morstyn, MBBS, PhD

Don Santel, BSE, MS

Julie Cherrington, PhD

Dennis Brown, PhD

Jean-Luc Tétard

McCullough Robertson Lawyers

Alta Partners

GBS Partners

Merck Serono

Former SVP and CMO, Amgen

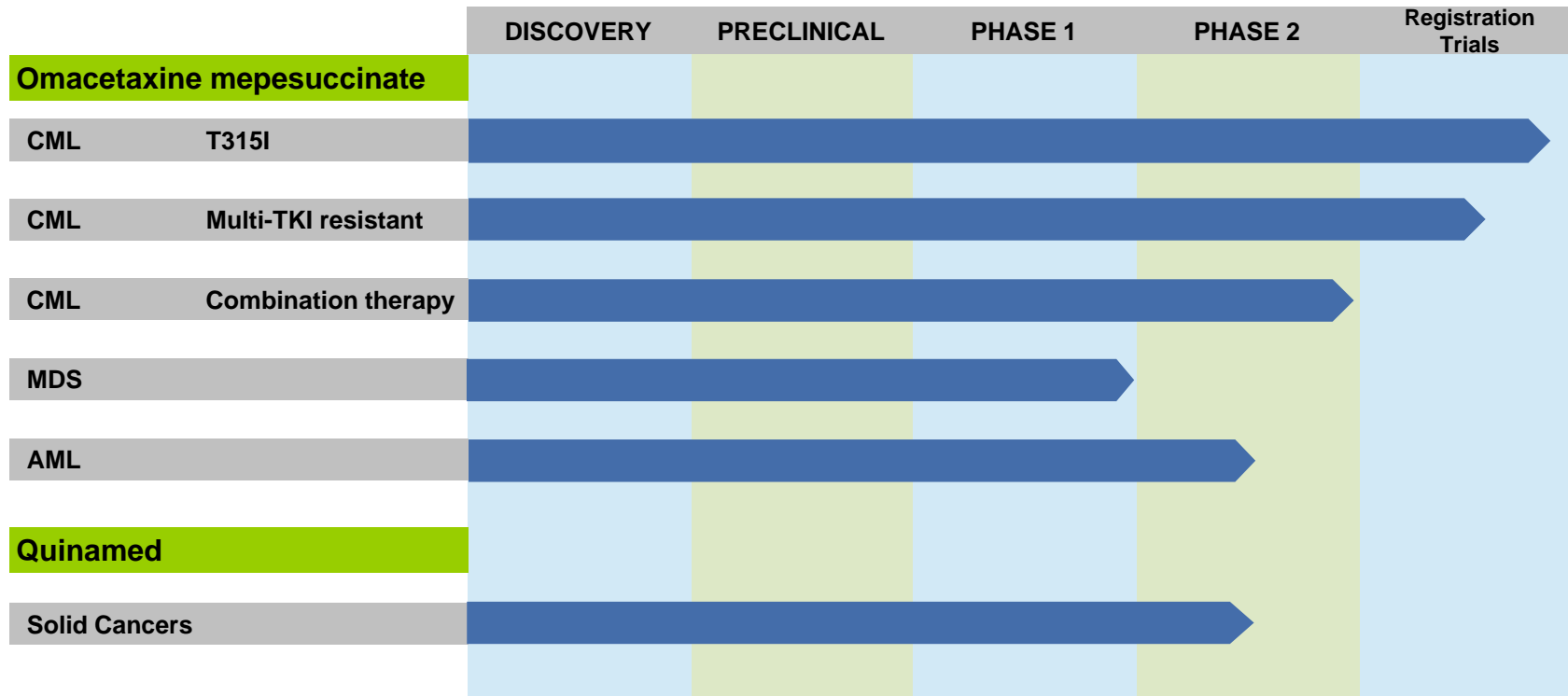
Former CEO, Co-Therix

President, Phenomix Corporation

Former Chief Scientific Officer

President, Stragen Pharma

Pipeline



Key Upcoming Events

Event	Timing
Complete filing for drug approval in USA	Q3 2009
Establish corporate partnership(s) for EU	H2 2009
Complete filing for drug approval in Europe	Q4 2009
Targeted approval and launch of omacetaxine in USA	Q1 2010
Targeted approval and launch of omacetaxine in Europe	Q3 2010

Omacetaxine mepesuccinate

A first-in-class cetaxine with
demonstrated clinical activity
in CML, MDS and AML

Omacetaxine Profile

- Multiple indications
 - Registration studies underway in CML
 - T315I Bcr-Abl mutation (demonstrated responses)
 - Multi-TKI resistant CML
 - Additional opportunities in MDS and AML
- Novel mode of action for blood malignancies
- Patient convenient administration
 - Subcutaneous injection at home
- US patents covering manufacturing, uses and formulations
- Orphan drug designation in US and EU in CML
- Speed-to-market strategy – T315I+ CML

Omacetaxine – Novel Mechanism of Action

- Omacetaxine is a first in class cetaxine that specifically binds the ribosomal A-site cleft inhibiting protein translation¹
- Omacetaxine treatment selectively reduces the levels of short-lived oncoproteins that are upregulated in leukemic cells²
 - Cyclin-D1 - proliferation
 - Mcl-1 - apoptosis
 - c-Myc - differentiation

¹ Gurel, G. *et al.* J. Mol Biol. (2009), doi:10.1016/j.jmb.2009.04.005

² Robert F *et al.* (2009), PLoS ONE 4(5): e5428 doi:10.1371/journal.pone.0005428

Omacetaxine – Effect on Leukemic Stem Cells

- TKIs are largely ineffective on leukemic stem cells
 - Resulting in minimal residual disease
 - Patients often relapse when TKIs are discontinued
- Omacetaxine has been demonstrated, *in vitro*, to be effective at killing human CML stem cells as well as peripheral leukemic cells¹

¹Allan E. *et al.* Omacetaxine - cytotoxic activity against chronic myeloid leukaemia stem cells , 14th Congress of the European Hematology Association, Abstract No. 1088, June 2009

Omacetaxine for the treatment of CML

Current Treatment Options in CML

- World CML prevalence >200,000 patients and growing
- Gleevec[®] (imatinib) approved in 2001 - first effective treatment
 - Targeted tyrosine kinase inhibitor (TKI)
 - Global sales of US\$3.7 billion in 2008 (60-70% in CML)
- Current challenges with Gleevec
 - Gleevec is not a cure for CML
 - Resistance is an emerging issue in CML (after 4y up to 50%)¹
- Two approved second line therapies
 - Sprycel[®] (dasatinib) by BMS approved in June 2006
 - Tassigna[®] (nilotinib) by Novartis approved in October 2007

What are the Unmet Clinical Needs in CML?

- Resistance is linked to gene mutations
 - 44% of Gleevec failures have mutations¹
 - T315I most frequent (15-20%)²
- No approved drugs are effective in T315I patients
 - Second generation and TKIs in development are NOT effective against T315I mutation
- No approved therapeutic options for 3rd line interventions after failing existing therapies
- Omacetaxine may be used initially in T315I CML patients and has potential to expand to patients that develop resistance to current therapies

¹ NCCN clinical practice guidelines in oncology – chronic myelogenous leukemia v.2.2009 10 16 0

² Nicolini FE *et al.* Leukemia. 2006; 20: 1061-1066

Omacetaxine for the treatment of CML

First Opportunity: T315I+ CML patients

Omacetaxine Clinical Trial Phase 2/3 in T315I+ CML (Study 202)

Design	<ul style="list-style-type: none">▪ Open label, adaptive design
Patients	<ul style="list-style-type: none">▪ 80-100 patients planned
Sites	<ul style="list-style-type: none">▪ ~35 in US, EU and Australasia
Inclusion criteria	<ul style="list-style-type: none">▪ Patients who have failed imatinib and have T315I Bcr-Abl mutation▪ Patients categorized according to chronic, accelerated and blast-phase CML
Dose	<ul style="list-style-type: none">▪ Induction phase: 1.25 mg/m² by subcutaneous injection two times a day for 14 days, every 28 days; up to 6 cycles▪ Maintenance phase: As per induction, but 7 days treatment every 28 days
Primary and secondary endpoints	<ul style="list-style-type: none">▪ Hematologic and cytogenetic response
Regulatory status	<ul style="list-style-type: none">▪ Fast Track status granted by FDA in November 2006▪ FDA and EMEA orphan drug designation for CML

Study 202 – Hematologic and Cytogenetic Responses

Response	No. (%)		
	CP N=40	AP N=16	BP N=10
Hematologic	34 (85)	8 (50)	4 (40)
CHR	34 (85)	5 (31)	2 (20)
HI	NA	2 (13)	1 (10)
RCP	NA	1 (6)	1 (10)
Cytogenetic	11 (28)	1 (6)	-
MCyR	6 (15)	-	-
CCyR	4 (10)	1 (6)	-
PCyR	2 (5)	-	-
Minimal	5 (13)	-	-

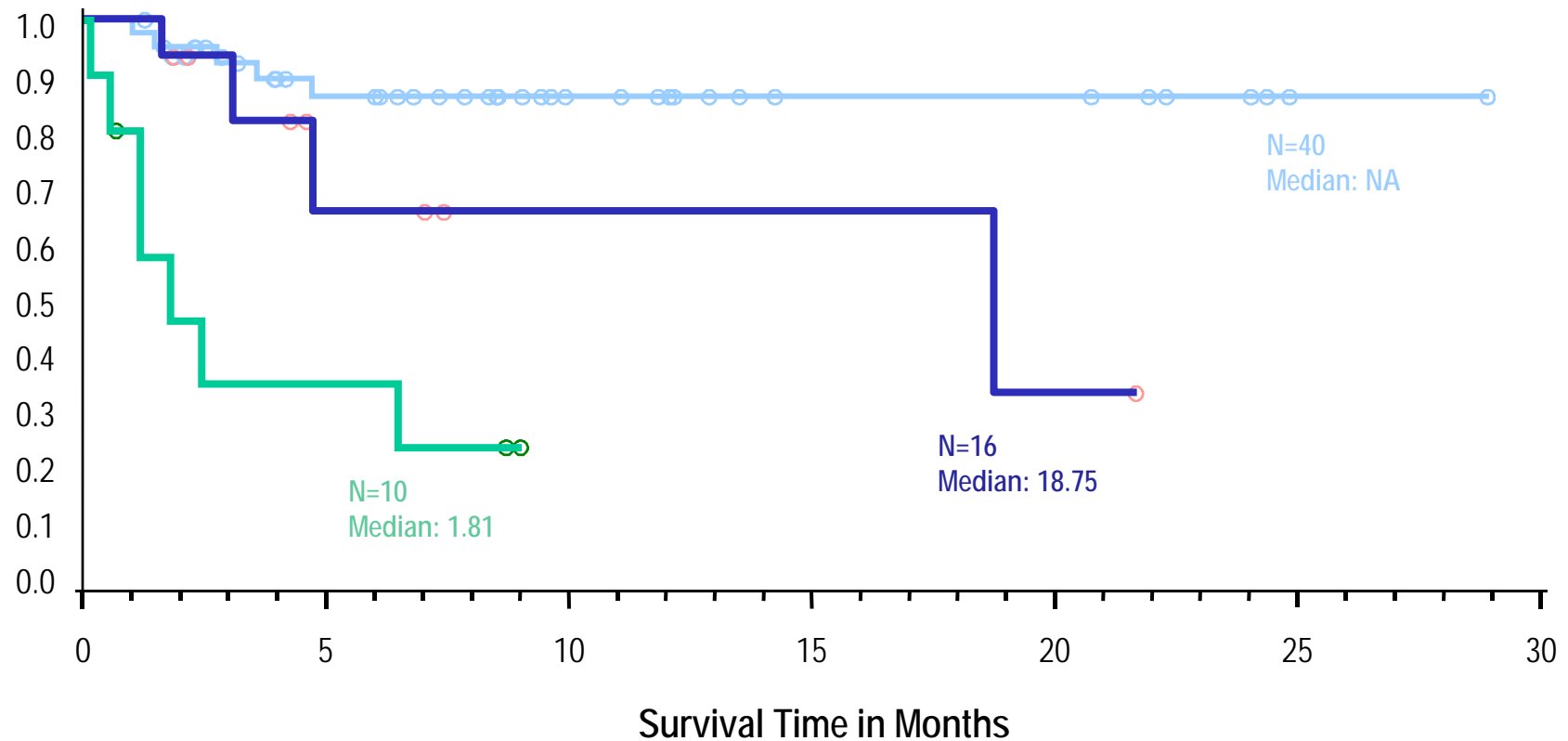
Data independently adjudicated by Data Monitoring Committee

Study 202 – Duration of Response

Response	Median (range) in months		
	CP N=40	AP N=16	BP N=10
Hematologic			
CHR	8.9 (1.7 – 23.9)	4.1 (0.9 – 15.0)	3.3 (2.2 – 4.4)
HI	NA	3.4 (3.1 – 3.7)	6.5
RCP	NA	3.7	1.2
Cytogenetic			
MCyR	6.1 (0.7 – 16.2)	1.8	-
CCyR	7.4 (5.1 – 16.2)	1.8	-
PCyR	2.0 (0.7 – 3.3)	-	-

Data independently adjudicated by Data Monitoring Committee

Omacetaxine in T315I+ CML Pts - Overall Survival



— Chronic Phase — Accelerated Phase — Blast Phase



Omacetaxine in T315I+ CML Patients - Conclusions

- Subcutaneous omacetaxine is generally well tolerated
- Myelosuppression is the most common toxicity
- Convenient and safe self-administration
- Durable hematologic & cytogenetic responses (85% CHR, 28% cytogenetic response in CP)
- Omacetaxine represents a new potential therapy for patients with T315I+ CML

T315I+ CML FDA / EMEA Approval Strategy

- Rolling NDA submission per Fast Track designation
 - Non-clinical section – Submitted
 - CMC section – Submitted
 - Clinical Section – Q3 2009
 - Anticipated approval and launch Q1 2010
- Clinical pre-NDA meeting completed
- EMEA – centralized filing
 - Letter of intent submitted March 2009
 - MAA submission end Q4 2009
 - Anticipated approval and launch late Q3 2010



Omacetaxine for the treatment of CML

Second Opportunity: Multi-resistant CML patients

Omacetaxine Clinical Trial Phase 2/3 in TKI-Resistant CML (Study 203)

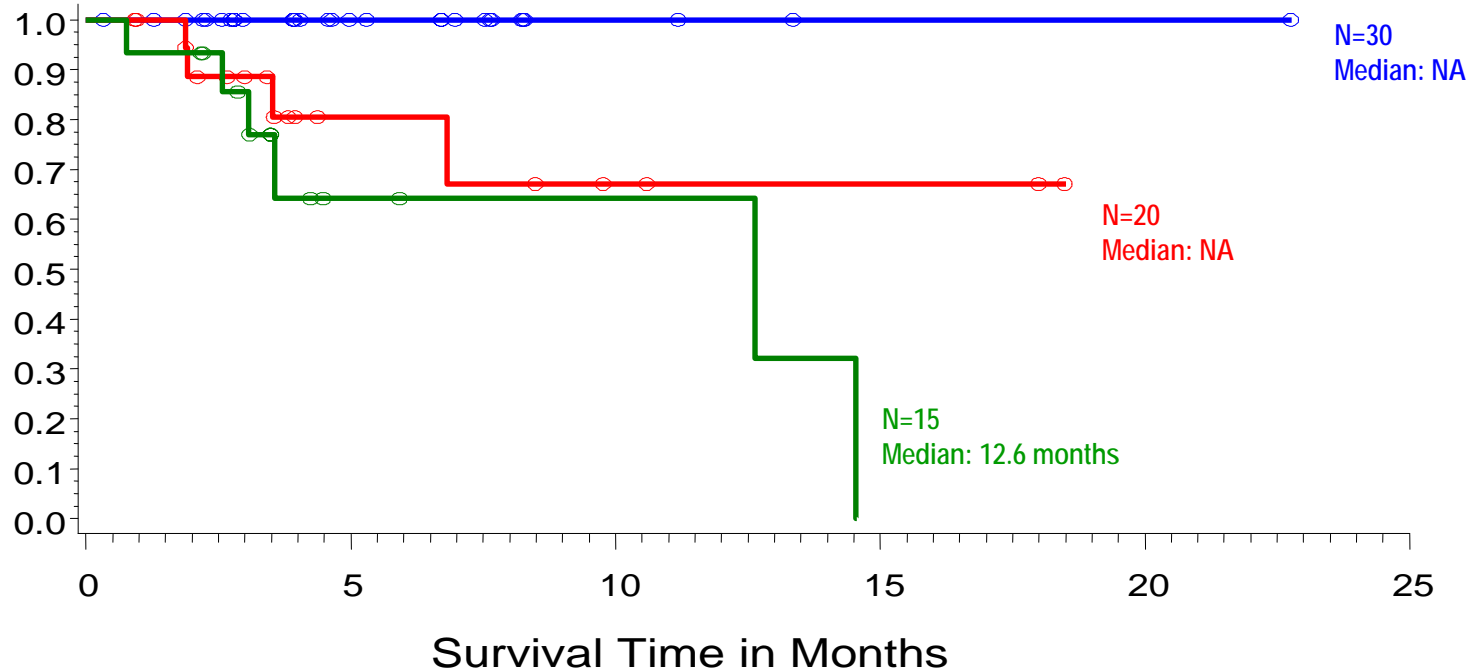
Design	<ul style="list-style-type: none">▪ Open label, adaptive design
Patients	<ul style="list-style-type: none">▪ 80-100 patients planned
Sites	<ul style="list-style-type: none">▪ ~35 in US, EU and Australasia
Inclusion Criteria	<ul style="list-style-type: none">▪ Patients who have failed two or more tyrosine kinase inhibitors▪ Patients will be categorized according to chronic, accelerated and blast-phase CML
Dose	<ul style="list-style-type: none">▪ Induction phase: 1.25 mg/m² by subcutaneous injection two times a day for 14 days, every 28 days▪ Up to 6 cycles of induction therapy▪ Maintenance phase: As per induction, but 7 days treatment every 28 days
Primary and secondary endpoints	<ul style="list-style-type: none">▪ Hematologic and cytogenetic response
Regulatory Status	<ul style="list-style-type: none">▪ FDA and EMEA orphan drug designation for CML

Study 203 – Hematologic and Cytogenetic Responses

Response	No. (%)		
	CP N=30	AP N=20	BP N=15
Hematologic	24 (80)	16 (80)	8 (53)
CHR	24 (80)	12 (60)	6 (40)
HI	NA	1 (5)	-
RCP	NA	3 (15)	2 (13)
Cytogenetic	6(20)	1 (5)	-
MCyR	6 (20)	1 (5)	-
CCyR	3 (10)	-	-
PCyR	3 (10)	1 (5)	-

Data independently adjudicated by Data Monitoring Committee

Omacetaxine for Multi-TKI Resistant CML: Overall Survival



— Chronic Phase — Accelerated Phase — Blast Phase



Omacetaxine for Multi-TKI Resistant CML: Conclusions

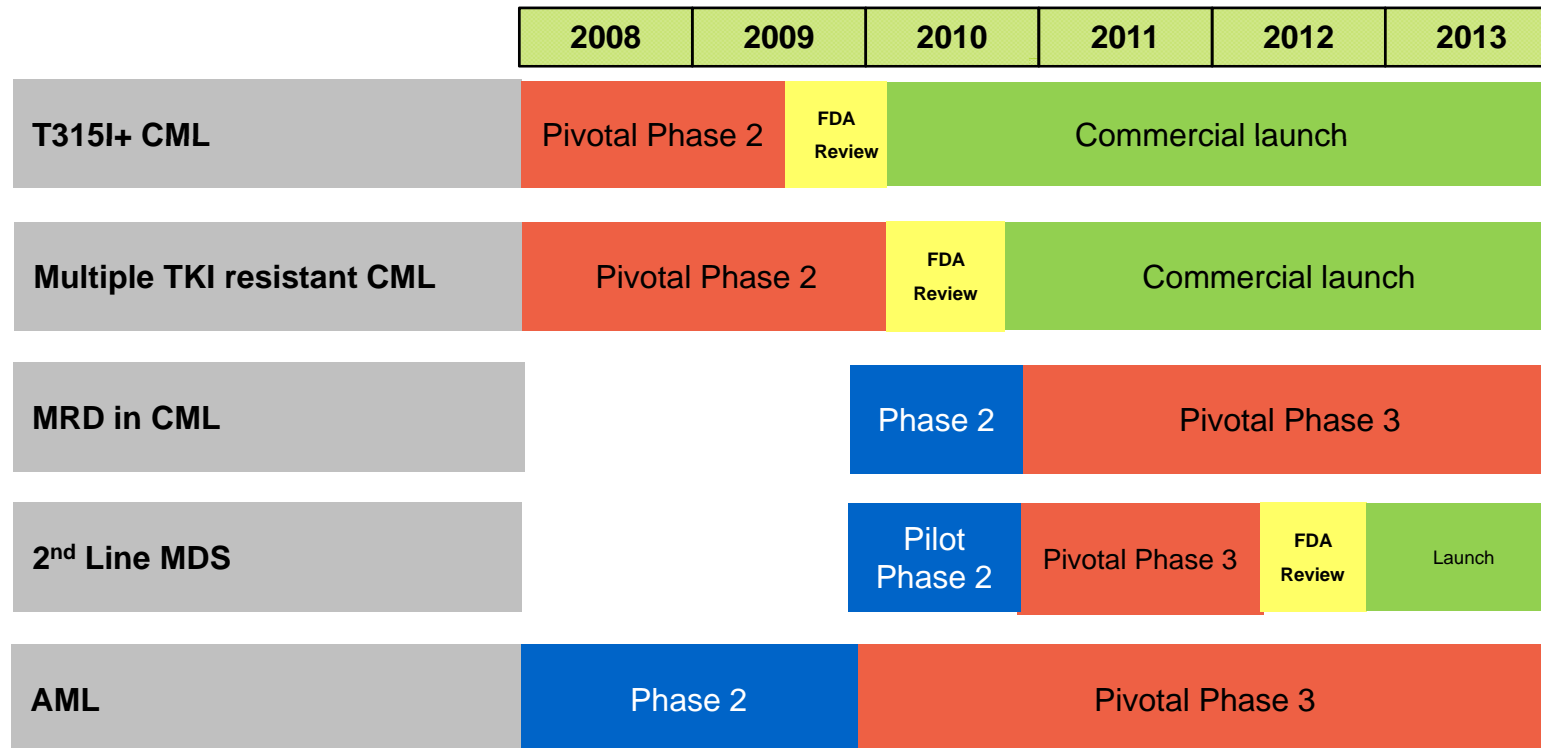
- Subcutaneously administered omacetaxine is generally well tolerated
- Myelosuppression is the most common toxicity and is usually manageable
- Convenient and safe self-administration
- Hematologic & cytogenetic responses (80% CHR, 20% cytogenetic response in CP)
- Overall survival at 18 months is 100% in chronic phase patients
- Omacetaxine may be an option for CML patients with multi-TKI resistance



Omacetaxine

Potential in CML, MDS and AML

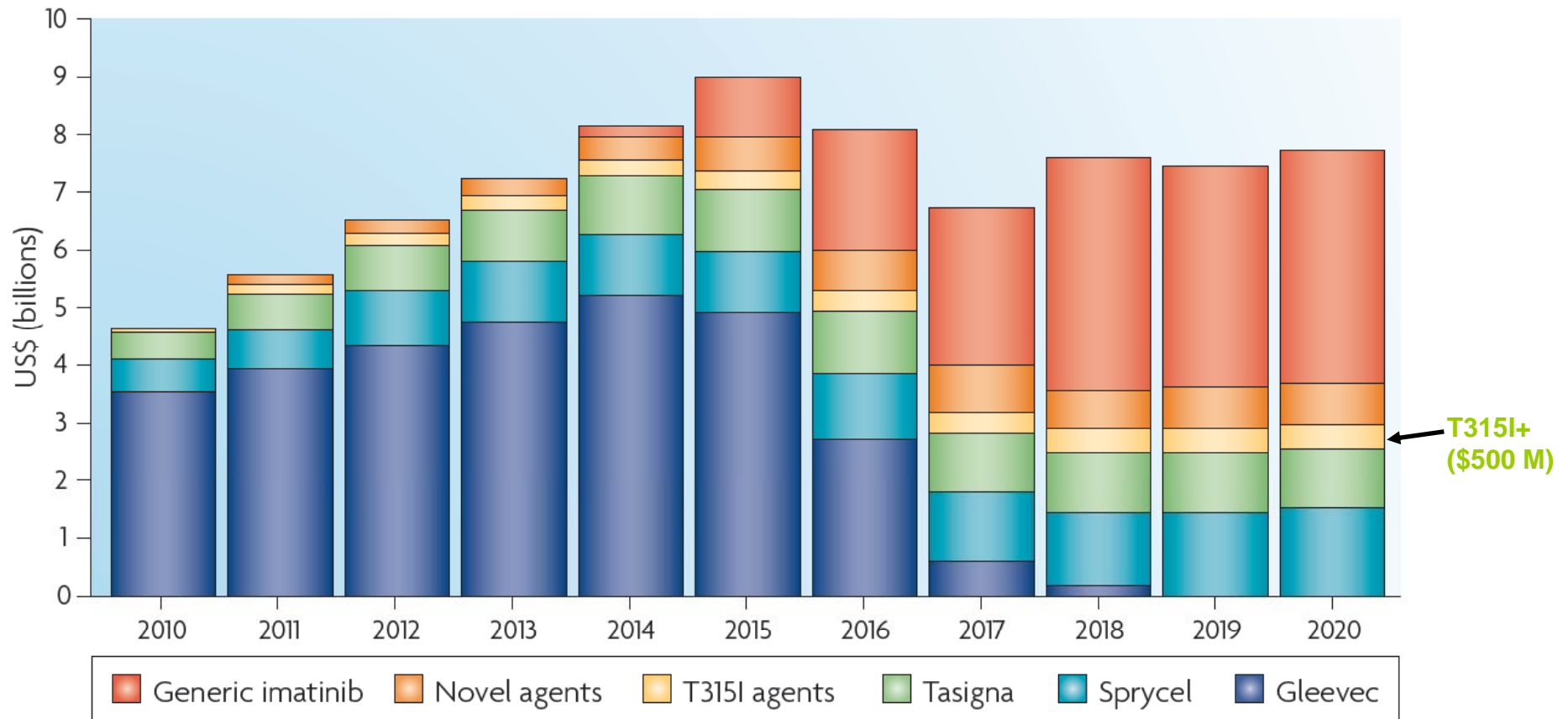
Omacetaxine Clinical and Regulatory Timeline - USA



Commercialization

Significant market for T315I CML
Ex-US partnering targeted for 2009
Cost-effective US launch in 2010

Estimate of T315I+ CML Market



Commercialization Strategy



- Strategic goal
 - Retain product rights in the USA
 - Out-license other territories to fund product development



- Ideal European partner profile
 - European focused, commercial infrastructure
 - Hematology/oncology product and sales force presence
 - At least 5 major markets plus distributor relationships



Financial Snapshot

Financial Parameter	Measurement
ASX#	CXS 283 million shares
NASDAQ Small Cap	CXSP (1 ADR = 15 shares)
Market Capitalization*:	A\$ 155 million
Cash held*:	A\$ 20 million
Significant Shareholders	Alta Partners (15%), Stragen Pharma (13%), Orbis Investments (10%), Merck KGaA (9%), GBS (8%)

* Effective 10th June 2009

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