

ASCO 2011 Analyst Briefing

June 6, 2011

Forward Looking Statements

- Our discussions during this meeting will include forward-looking statements. Actual results could differ materially from those projected in the forward-looking statements.
- The factors that could cause actual results to differ are discussed in Pfizer's 2010 Annual Report on Form 10-K and in our reports on Form 10-Q and Form 8-K.
- These reports are available on our website at www.pfizer.com in the "Investors—SEC Filings" section.

Leadership in Oncology

Mace Rothenberg, MD, Senior Vice President of Clinical Development and Medical Affairs



The Evolution of Pfizer Oncology: Delivering on the Promise of Innovation and R&D

1st regulatory approval of Epirubicin in France

1982

Irinotecan launches for metastatic colorectal carcinoma that has recurred or progressed after treatment with 5-FU chemotherapy

1996

FDA approves Aromasin in advanced breast cancer in post-menopausal patients

1999

Accelerated FDA approval of Sutent in advanced RCC and imatinib-resistant and -intolerant GIST

2006

Oncology Business Unit forms with 3 marketed agents (Aromasin, Camptosar, Sutent)

2008

Pfizer acquires Wyeth - adding Torisel and 3 investigational compounds neratinib, bosutinib, and inotuzumab to portfolio

2009

OBU plans to submit three new molecular entities for regulatory review

2011

AND WE'RE GROWING

Sutent

Advanced pancreatic
neuroendocrine tumors

Crizotinib*

ALK-positive advanced NSCLC

Axitinib*

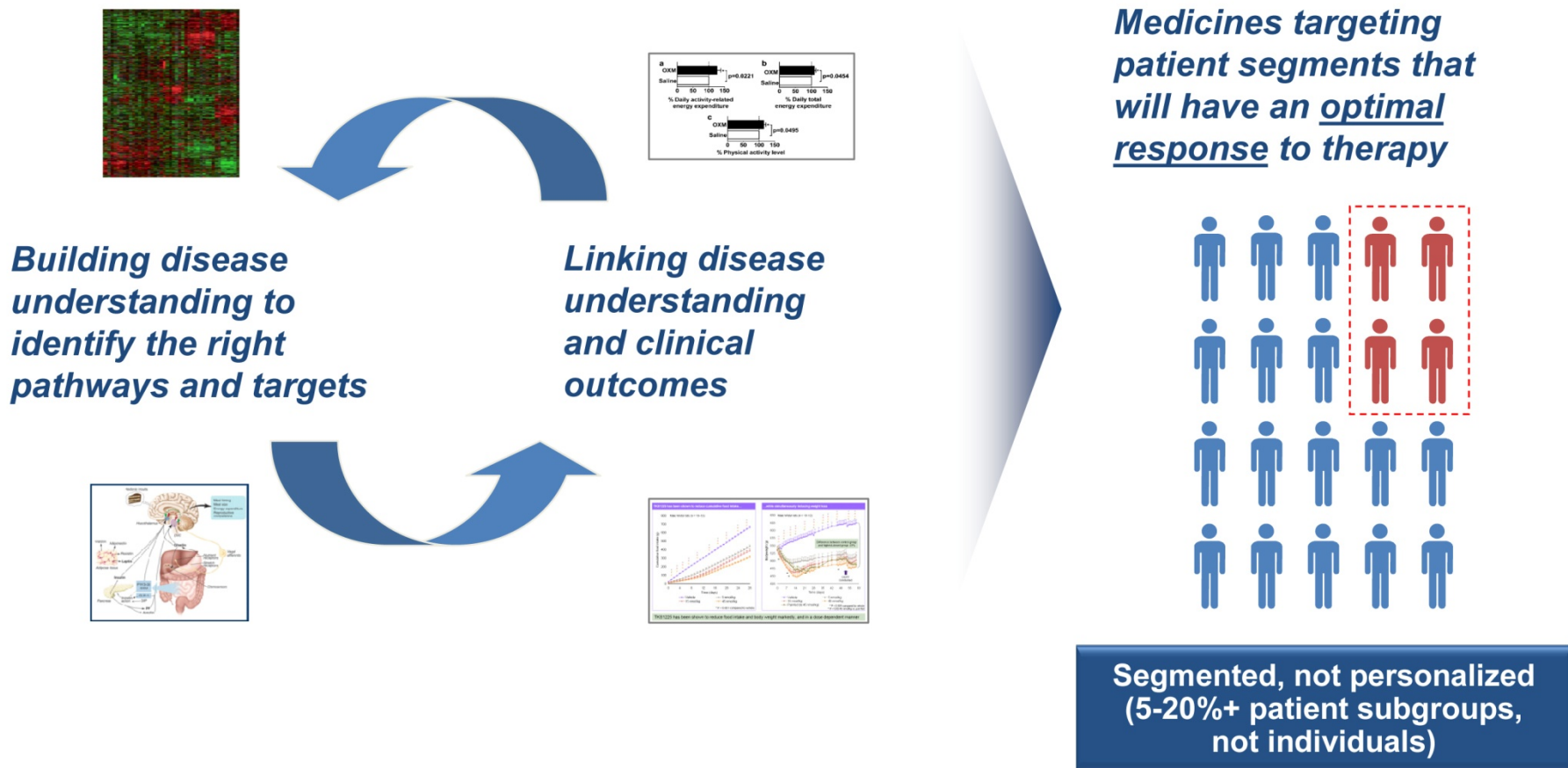
Advanced renal cell carcinoma

Bosutinib*

Chronic Myeloid Leukemia

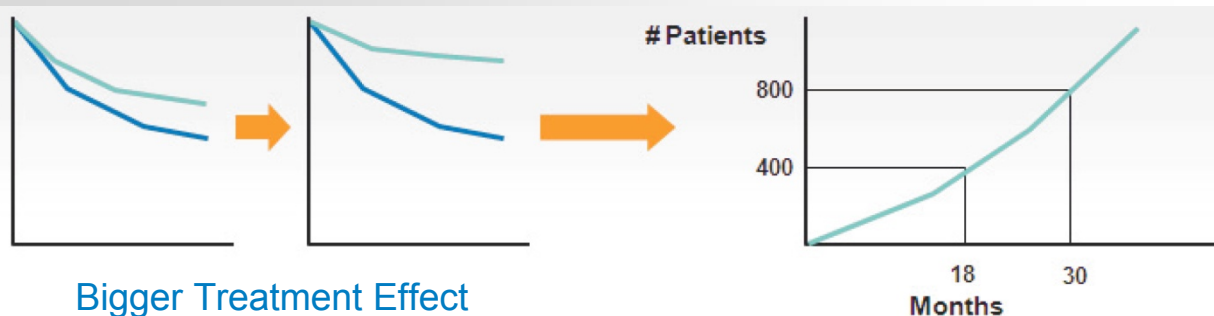
*Investigational agents

Right Target, Right Drug(s), Right Patient

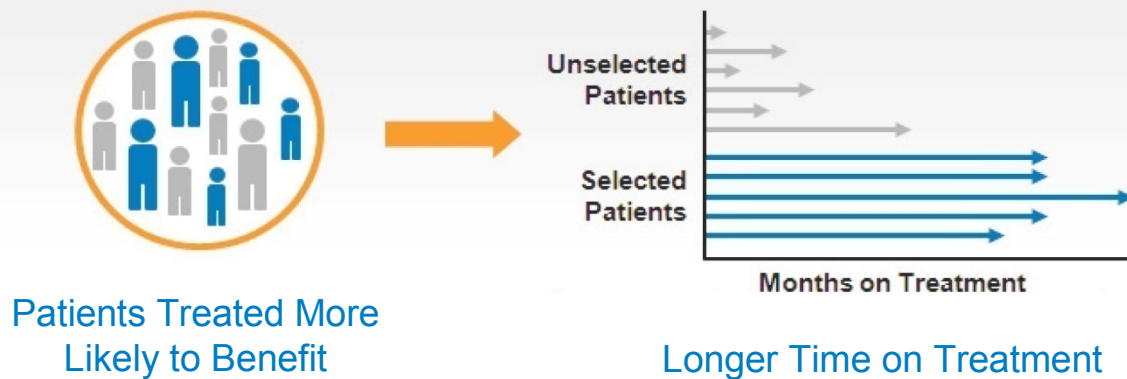


Driving Improved Drug Development and Commercial Advantage

Clinical Development



Commercial Benefits



Smaller Clinical Trials
+
Less Costly, Faster
Trial Completion

Earlier Regulatory
Submission
+
Earlier Launch

More Effective in
Selected Patient
Segments
↓
Value of Treatment
Easier to Demonstrate
to Payers

Pfizer Oncology Data at ASCO 2011



Pfizer Data at ASCO

RCC

LUNG

HEMATOLOGY



Axitinib

Crizotinib

PF-00299804

Inotuzumab
Ozogamicin

Bosutinib

Advanced RCC: A Patient Population with Continued Unmet Needs

Despite recent advances in the treatment of advanced RCC, **patients are still in need of additional therapeutic options**

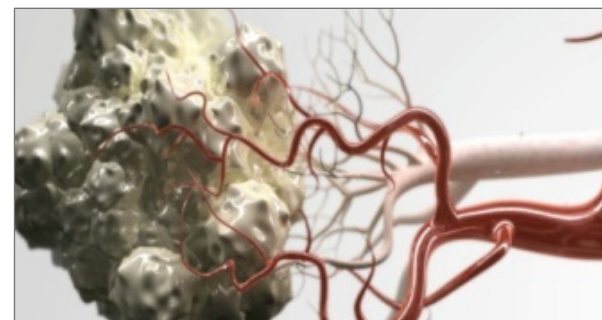
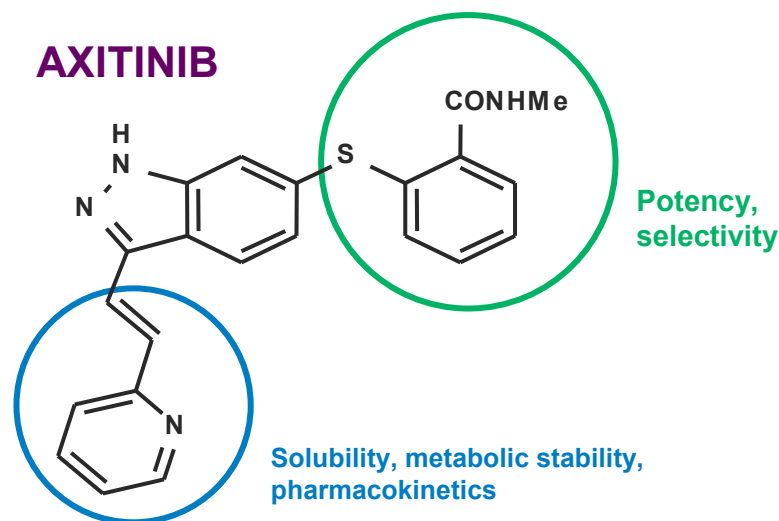
Up to **30% of RCC patients present with metastatic disease** worldwide; Approximately 1/3 experience a recurrence

Globally, five-year **survival rates** for patients with advanced disease **remain low**, at around 20%

In the U.S., approximately 13,000 patients were expected to die from RCC in 2010

Axitinib is an Oral and Selective Inhibitor of VEGF Receptors 1, 2, 3

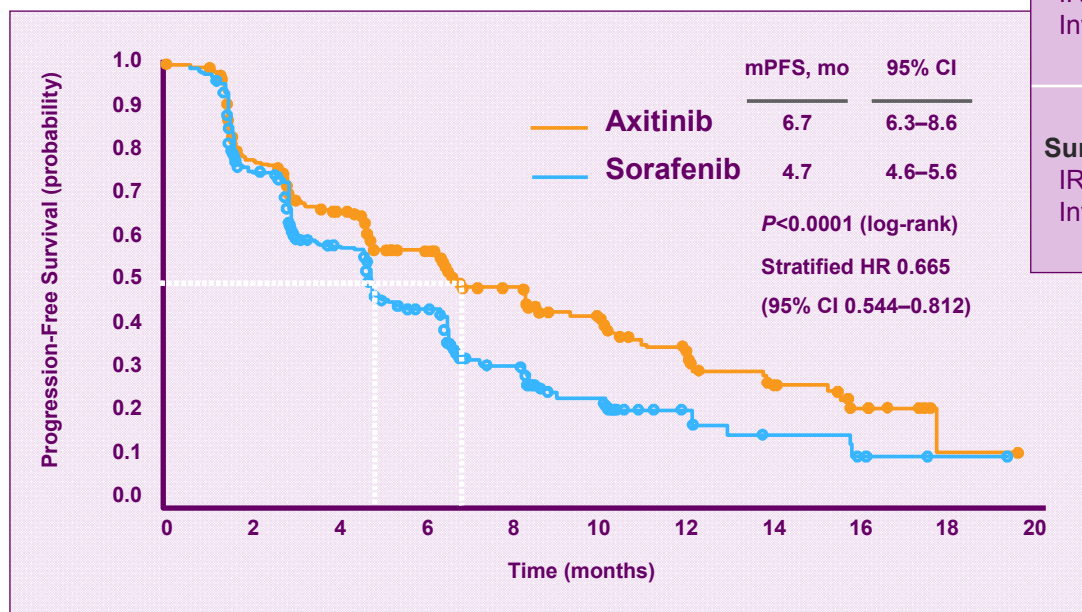
Axitinib's structure-based drug design allows strategic optimization of critical binding elements



- Simultaneously blocks VEGF 1,2,3 receptors
- VEGF signaling pathways play a key role in both the growth and progression of tumors

Phase 3 AXIS Trial: Axitinib 1st to Demonstrate Efficacy vs. Targeted Agent in Phase 3

Progression-free Survival (IRC Assessment)



Subjects at risk, n

Axitinib	361	256	202	145	96	64	38	20	10	1	0
Sorafenib	362	224	157	100	51	28	12	6	3	1	0

IRC = Independent Review Committee

PFS by Prior Regimen

Prior Treatment Regimen	Axitinib (n=361)	Sorafenib (n=362)	HR	P value*
Cytokines (n=251)				
IRC	12.1	6.5	0.464	<0.0001
Investigator	12.0	8.3	0.636	0.005
Sunitinib (n=389)				
IRC	4.8	3.4	0.741	0.011
Investigator	6.5	4.5	0.636	0.0002

*One-sided log-rank test stratified by ECOG PS

Global Burden of Lung Cancer

With about **85% of all lung cancers** being classified as NSCLC

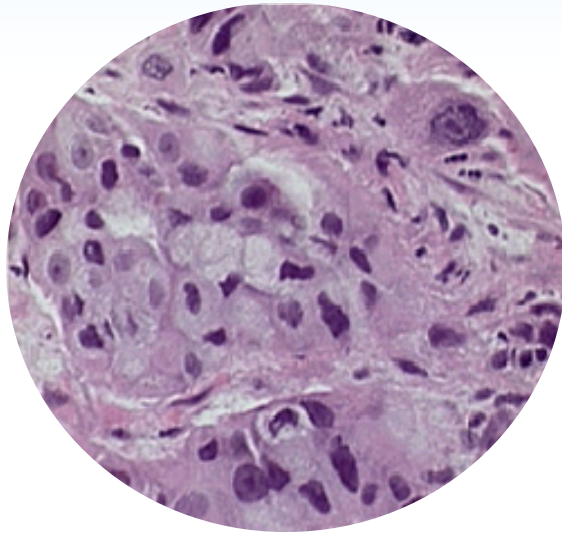
- Beyond histology, some patients have combination of tumor-driving genetic mutations or alterations with distinct molecular characteristics
 - Such as EGFR and anaplastic lymphoma kinase (ALK)

Approximately 40,000 new ALK-positive NSCLC patients are expected to be identified per year worldwide

Using the Genetic Makeup of A Person's Tumor to Classify and Treat Lung Cancer

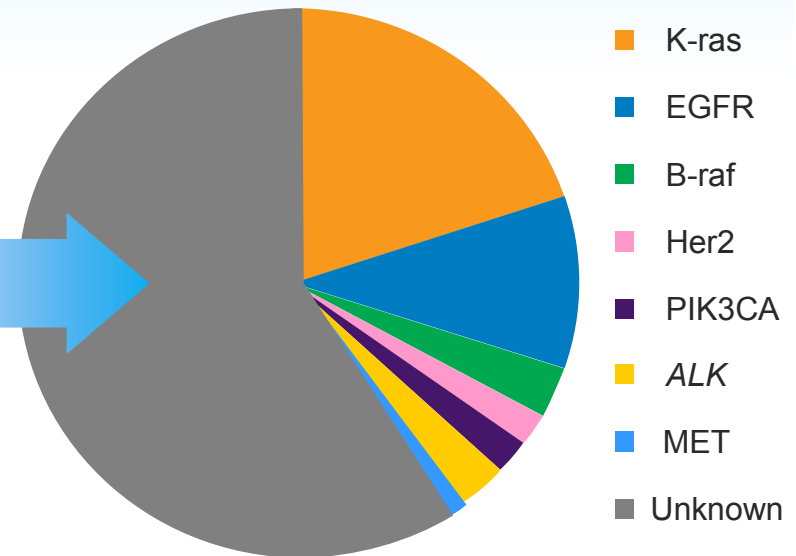
1999

Adenocarcinoma
Histology-driven selection



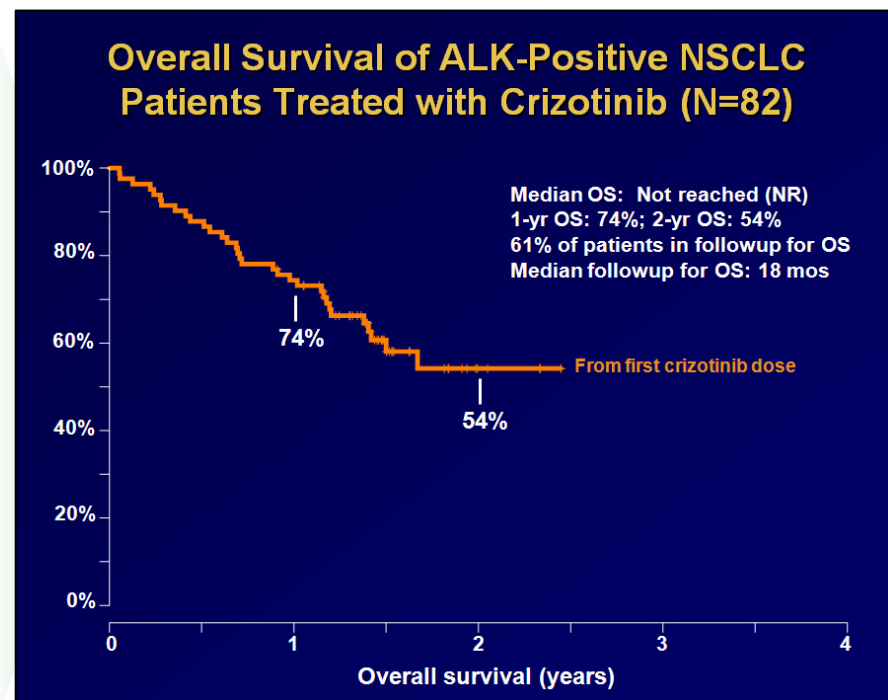
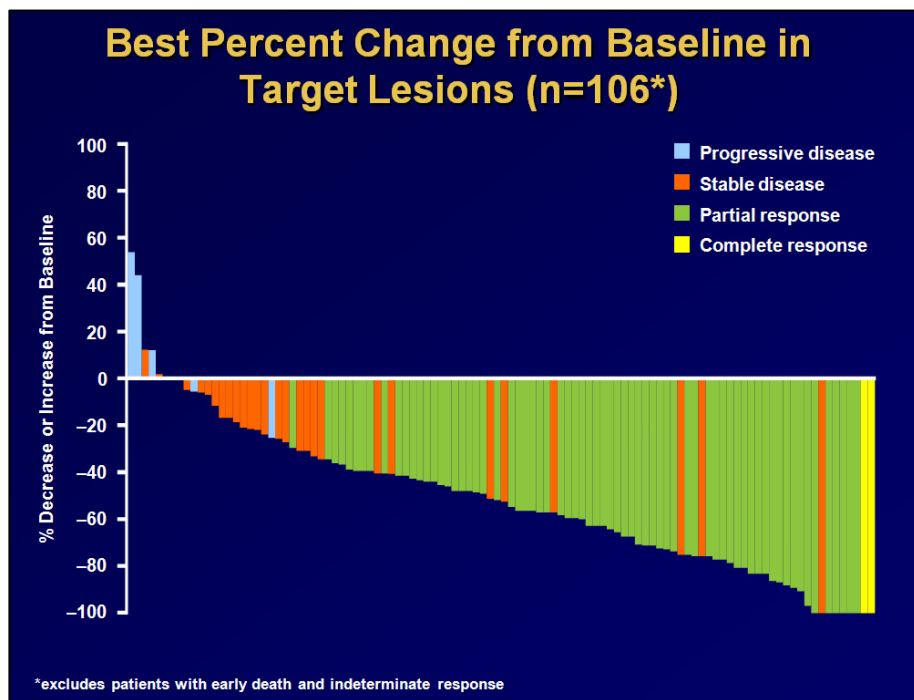
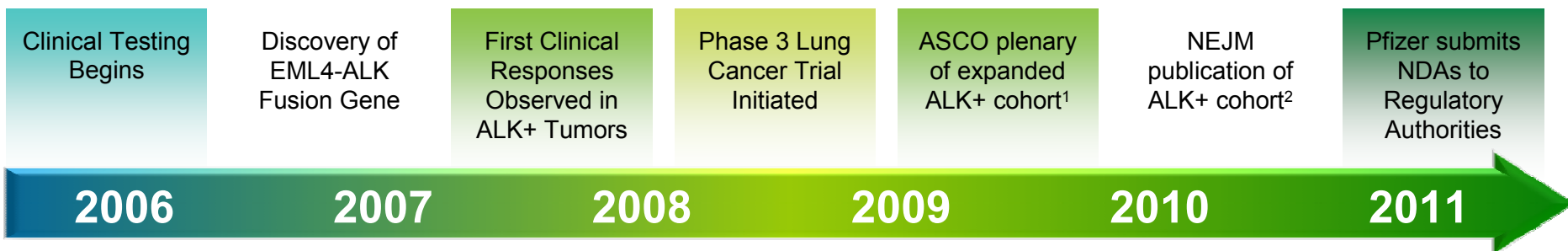
2011

Adenocarcinoma
Targeting oncogenic drivers



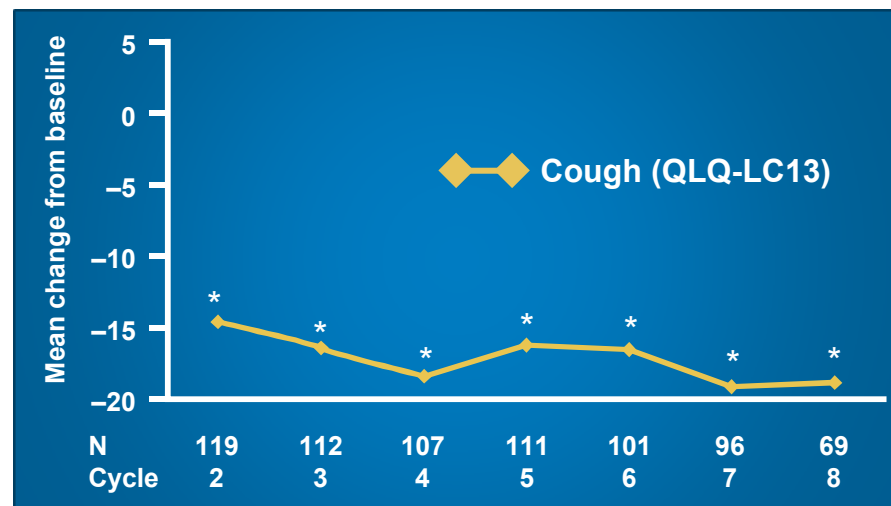
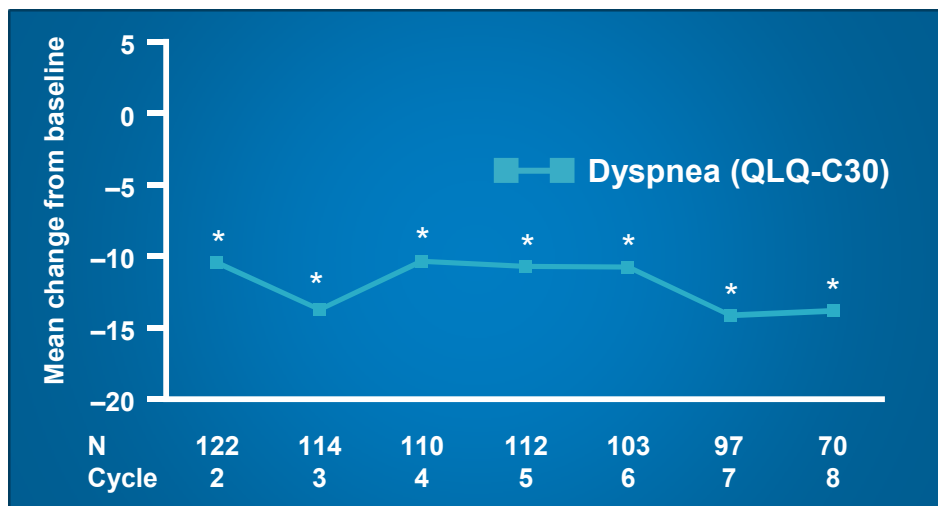
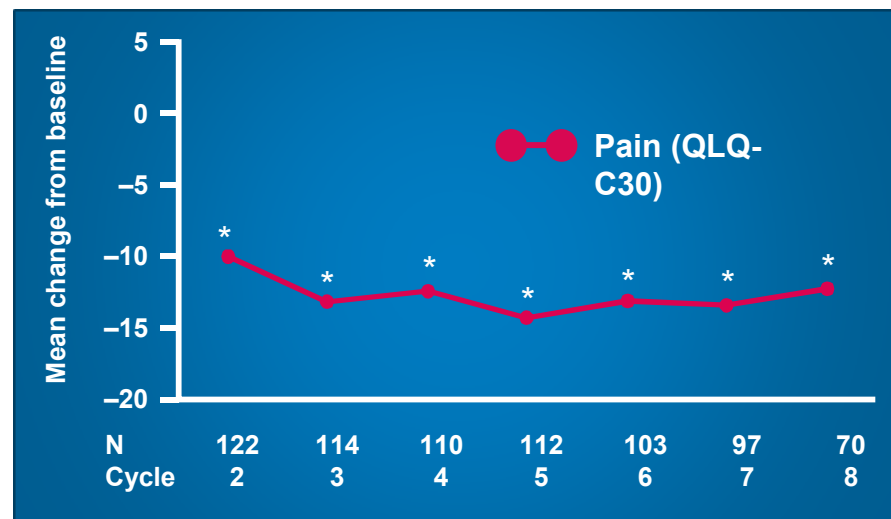
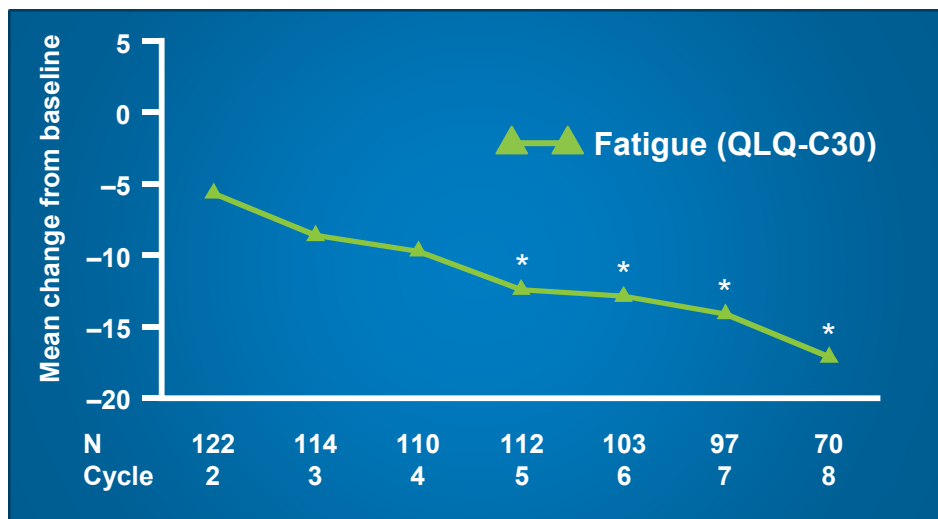
***CRIZOTINIB – A Potential Scientific Breakthrough:
Targeting the ALK fusion gene, a direct driver of oncogenesis***

Rapid Timeline from Compound Identification, Target Discovery and Clinical Results



Study in ALK-Positive NSCLC Patients Treated with Crizotinib

In Phase 2 Study of Crizotinib, 51% Overall Response Rate & PRO Show Clinically Meaningful Benefits*



Burden of Hematologic Malignancies

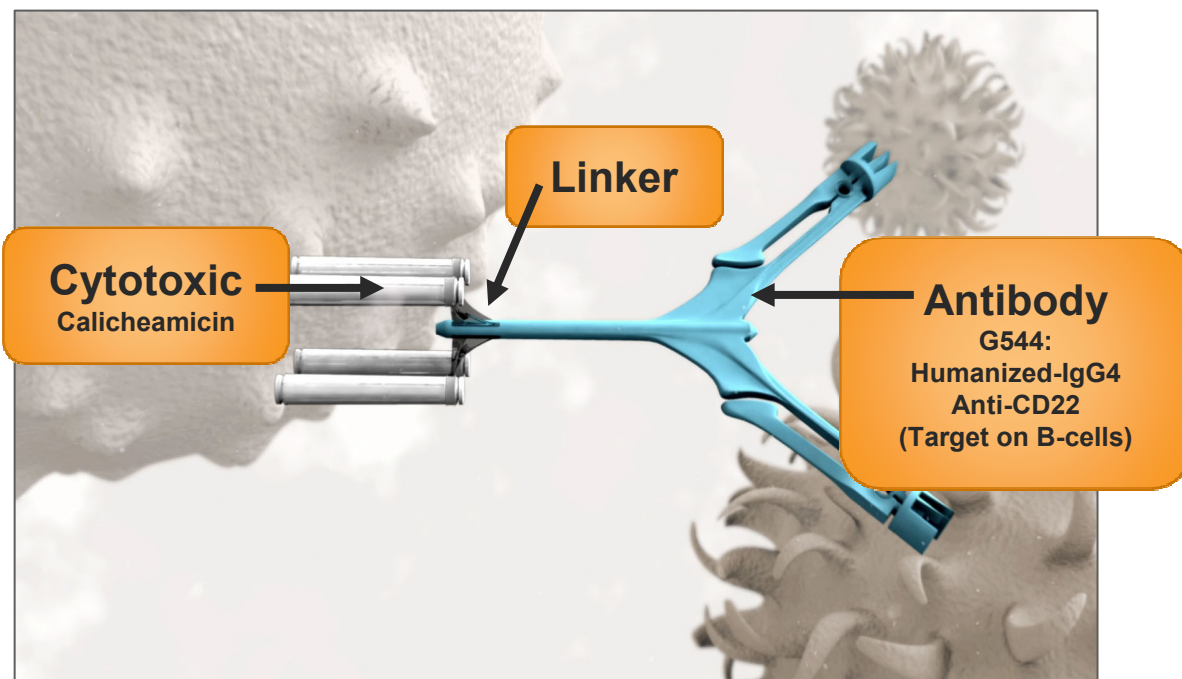
Hematologic malignancies represent the **5th most commonly occurring cancers** and the second leading cause of cancer death worldwide

Despite availability of existing treatments for patients with chronic myeloid leukemia, which accounts for **15% of all leukemias** worldwide, a need remains for more options for newly diagnosed and relapsed patients, given problems with treatment-related toxicities and resistance in this patient population

Currently **about half of Non-Hodgkin's lymphoma patients relapse** following treatment with first-line therapy

- For patients who fail 1st-line therapy and are not candidates for autologous stem cell transplant, **average survival is <1 year**

Inotuzumab Ozogamicin



CLINICAL TRIAL SNAPSHOT

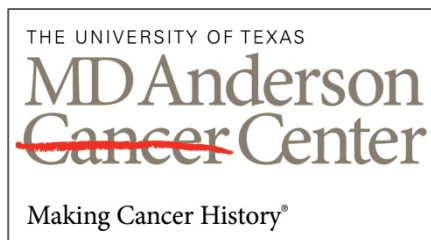
PHASE 2

- Inotuzumab in indolent NHL refractory to or relapsed after rituximab and chemotherapy or radioimmunotherapy
- Inotuzumab + rituximab in diffuse large B-cell lymphoma eligible for autologous stem cell transplantation

PHASE 3

- Inotuzumab + rituximab in relapsed/refractory aggressive NHL tumors

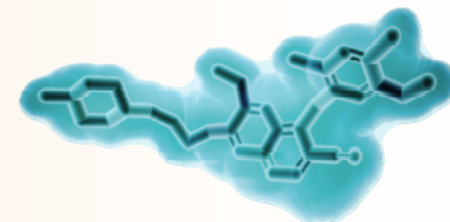
INVESTIGATOR INITIATED TRIAL IN ACUTE LYMPHOCYTIC LEUKEMIA (ALL) AT ASCO



“A **response rate of more than 50 percent** in this patient population probably makes inotuzumab ozogamicin **the most active single-agent therapy ever for ALL**,” said Hagop Kantarjian, M.D., MD Anderson’s Department of Leukemia and study senior investigator.”

Bosutinib

- 2ND generation dual inhibitor of Abl and Src family kinases
 - Potent ATP-competitive inhibitor of Bcr-Abl oncogene
 - 200x more potent than Gleevec
 - Unlike competitors, minimally inhibits PDGR [should this be PDGFR?] and cKIT



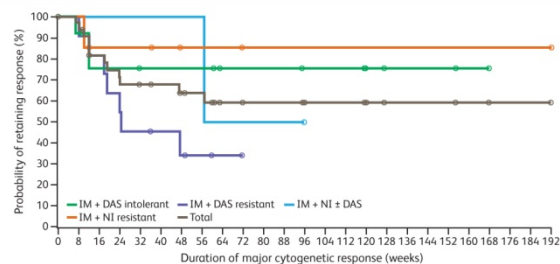
STUDY 200 – CP Ph+ CML previously treated with both imatinib and dasatinib or nilotinib

Major cytogenetic response rates of 32%

Median follow-up of 28.5 months, <5% of subjects progressed to advanced or blast phase while on bosutinib

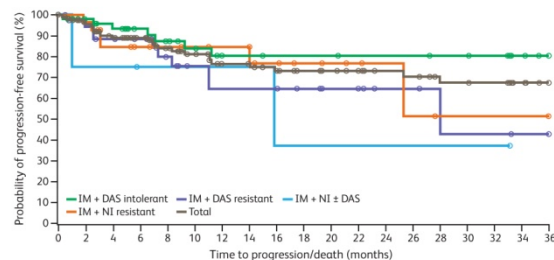
77% progression free at one year; 73% at 2 years
-- 91% and 81% of patients still alive at 1 and 2 years

Figure 1. Duration of MCyR.



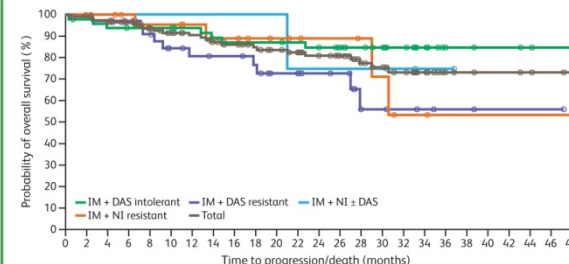
	n	Median duration of MCyR (95% CI), wk	Kaplan-Meier estimate of retaining response at 2 years,* %
Total responding population	35	NA (47.3–NA)	59
IM + DAS resistant	11	24.1 (18.0–NA)	34
IM + DAS intolerant	13	NA (NA–NA)	76
IM + NI resistant	9	NA (NA–NA)	86
IM + NI ± DAS ^b	2	NA (57.0–NA)	50

Figure 2. Progression-free survival.



	n	Median PFS (95% CI), mo	PFS estimate at 1 year,* %	PFS estimate at 2 years,* %
Total population	118	NA (NA–NA)	77	73
IM + DAS resistant	37	27.9 (11.0–NA)	65	65
IM + DAS intolerant	50	NA (NA–NA)	81	81
IM + NI resistant	27	NA (25.3–NA)	85	77
IM + NI ± DAS ^b	4	15.8 (0.9–NA)	75	38

Figure 3. Overall survival.

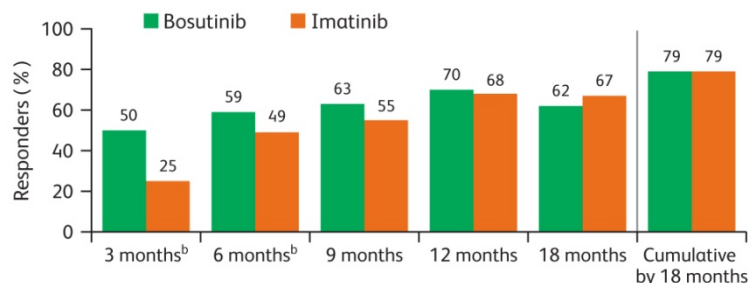


	n	Overall survival estimate at 1 year,* %	Overall survival estimate at 2 years,* %
Total population	118	91	83
IM + DAS resistant	37	83	75
IM + DAS intolerant	50	94	85
IM + NI resistant	27	96	92
IM + NI ± DAS ^b	4	100	75

BELA (STUDY 3000) – 18 Month Follow-up in newly diagnosed chronic phase Philadelphia chromosome (Ph+) CML

Figure 1. Rates of response in the ITT population.

A. CCyR^a



B. MMR^c

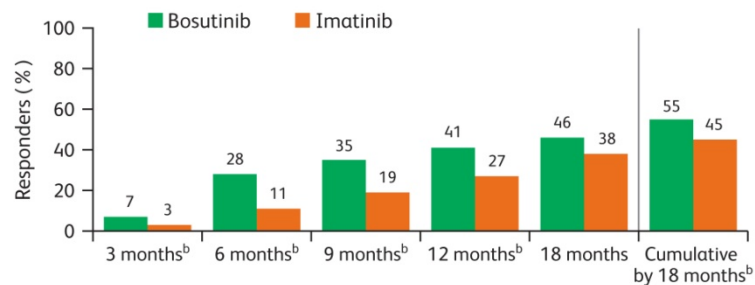
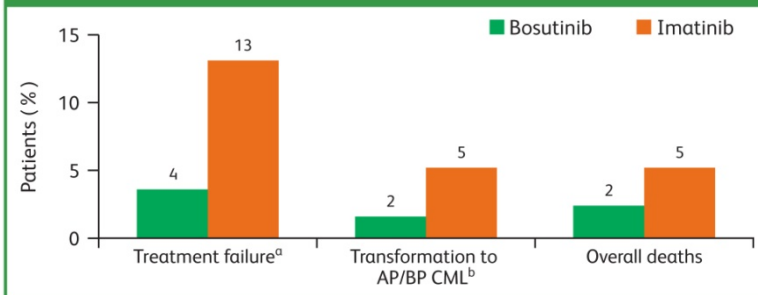


Figure 2. Disease progression.



- MMR rate remains numerically favorable for bosutinib compared to imatinib (at 18 months 46% vs 38%, by 18 months 55% vs. 45%)
- Rate of transformation: no new events in bosutinib arm vs 3 in imatinib arm
- Still no difference in CCyR rate between bosutinib and imatinib at 18 months (62.4% vs. 67.5%) or by 18 months (78.8% vs 79%)

Broad Portfolio Delivers Innovation

Multiple Investigational Compounds Addressing Unique Pathways

SMO and Hedgehog Signaling Inhibitor

PF-04449913

small molecule, oral inhibitor of smoothened (SMO)

Irreversible pan HER Inhibitor

PF-00299804

oral, once daily, pan-HER inhibitor

Activin Receptor- Like Kinase -1 (ALK-1) Inhibitor

PF-03446962

(ALK-1) monoclonal antibody

PI3K/mTOR PF-04691502 (oral) & PKI-587/PF- 05212384 (IV)

dual inhibitors of class 1A & 1B PI3K family members, and mTOR

CDK 4/6 Inhibitor

PD 0332991

orally active & highly selective inhibitor of the CDK4 and CDK6 kinases

PF-00299804: Small Molecule Inhibitor of HER 1 (EGFR), HER-2 and HER-4 Tyrosine Kinase

Irreversible Pan-HER Inhibition

HER 1
(EGFR)

T

PF-00299804

HER2

T

PF-00299804

HER3

No kinase
activity

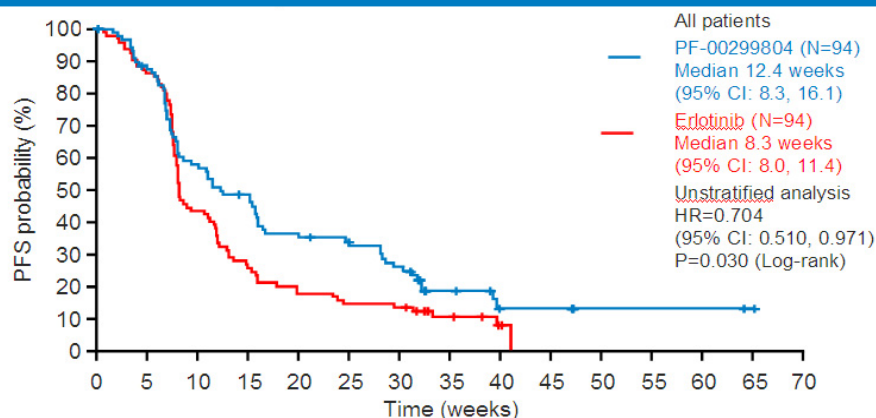
HER4

T

PF-00299804

In lung cancer, pan-HER inhibition may more comprehensively block HER signaling pathway than currently marketed HER1 (EGFR) inhibitors

Phase 2 PF-00299804 vs. Erlotinib 2nd/3rd-Line Post-Chemotherapy (Data Presented at ESMO 2010)



P
H
A
S
E

3

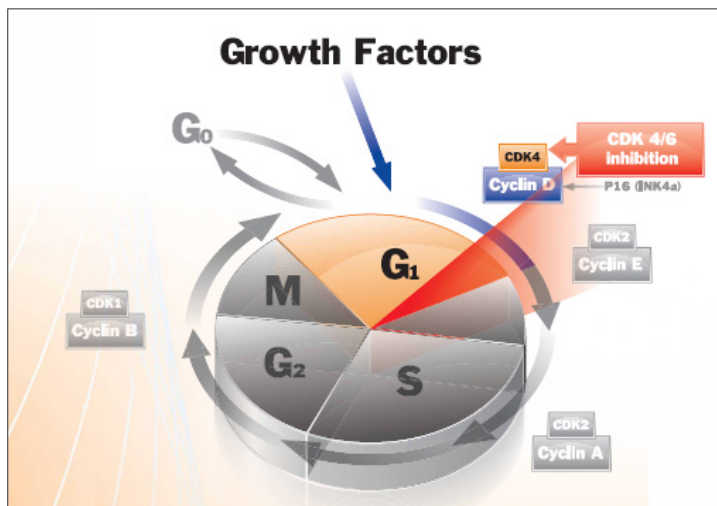


PF-00299804 vs erlotinib in 2nd or 3rd line therapy for advanced NSCLC

BR.26 *Led by the NCIC Clinical Trials Group*

PF-00299804 in patients with advanced NSCLC after progression on chemotherapy and EGFR inhibitor therapy

CDK 4/6 Inhibitor



CDK 4/6 inhibition has demonstrated antitumor activity in human xenograft models

More than 90% of human tumors circumvent control mechanisms for progression from G₁ to S phase

Phase I/II Breast Cancer Trial

Letrozole With or Without PD 0332991 for 1st-line Treatment of Estrogen-Receptor Positive, HER2-negative Advanced Breast Cancer

Open-label, multicenter study evaluating safety, efficacy, and pharmacokinetics

Protocol Amended

Preclinical data suggested biomarker selection [cyclin D1 (CCND1) amplification and/or loss of CDKNK1 (p16)] may confer susceptibility to PD 0332991-- forward enrollment of Phase II only includes patients who test positive for these biomarkers

Phase I/II Multiple Myeloma Trial

PD 0332991 in Combination With Bortezomib and Dexamethasone in Patients With Refractory Multiple Myeloma

Open-label, interventional, nonrandomized, multicenter efficacy, safety, and dosing trial in patients who have previously been treated for multiple myeloma

Key Takeaways

Pfizer Oncology is...

- 1** Delivering on the promise of personalized medicine as a leader in Oncology
Rapid discovery of the ALK target to clinical results for crizotinib for patients with advanced ALK-positive NSCLC
- 2** Making steady progress in accelerating science in areas of unmet need
Heritage in RCC continues with Pfizer's ability to meet the needs of another RCC patient population
- 3** Laying the foundation for the future with a rich and deep portfolio of novel compounds
Late-stage compounds (bosutinib, inotuzumab, PF-299) represent our understanding of key mechanisms and promising pathways in cancer
- 4** A committed partner in maximizing the understanding of our portfolio in the interest of cancer patients worldwide
Key collaborations, partnerships and alliances allow enrichment of our portfolio, as well as access to new technologies

Q&A

ASCO 2011 Analyst Briefing

June 6, 2011