Exelixis 3rd Annual R&D Day

December 5, 2007

Mandarin Oriental Hotel

New York, NY

Forward-Looking Statement

This presentation contains certain statements that are forward-looking, including, without limitation, statements relating to the future development and potential efficacy of XL647, XL880, XL184, XL019 and other Exelixis compounds; the timing of the initiation of clinical trials for XL019, XL184, XL647, XL880, XL652, XL147, XL765, XL518, XL820, XL281 and other Exelixis compounds; the availability of data for XL019 and other Exelixis compounds; the expected timing of the completion of clinical trials for XL647, XL844, XL281 and XL228; the anticipated filing of additional IND applications for XL139 and other Exelixis compounds; the timing of potential compound selections by GlaxoSmithKline and Genentech; the timing and success of further business activities and our estimated future revenues and expenses, estimated future balances of cash and cash equivalents. Words such as “predicted,” “possible,” “encouraging,” “expected,” “appears” “believes,’ "may," “would,” “continue,” “can,” “will” “could,” “expect,” “should,” “anticipate,” “initiate,” “indicate,” “suggest,” “plan,” “intend,” “potential,” “promising” and similar expressions are intended to identify forward-looking statements. These statements are only predictions and are based upon our current expectations. Forward-looking statements involve risks and uncertainties. Our actual results and the timing of events could differ materially from those anticipated in such forward-looking statements as a result of these risks and uncertainties, which include, without limitation: (1) risks related to our dependence on and relationship with GlaxoSmithKline and Symphony Evolution; (2) risks related to our need for additional financing, including our ability to enter into new collaborations, continue existing collaborations and receive milestones and royalties derived from future products developed from research efforts under collaborative agreements; (3) the potential failure of XL647, XL880, XL184, XL019, XL652 or any of our other compounds to demonstrate safety and efficacy in clinical testing; (4) our ability to complete and initiate clinical trials at the referenced times; (5) our ability to conduct clinical trials sufficient to achieve positive completion; and (6) our ability to successfully advance and develop additional compounds. These and other risk factors are discussed under “Risk Factors” and elsewhere in our Quarterly Report for the quarter ended September 30, 2007 and our other reports filed with the Securities and Exchange Commission. We expressly disclaim any duty, obligation or undertaking to release publicly any updates or revisions to any forward-looking statements made in this discussion to reflect any change in our expectations with regard thereto or any changes in events, conditions or circumstances on which any such statements are based.

Opening Remarks
George Scangos, President & CEO

Exelixis Overview

Broad clinical pipeline with significant commercial potential

Remarkable productivity

Multiple programs moving forward minimize risk, maximize upside

- Building substantial value
- Success of one or two drives upside
- Limited downside with compound failure

Exelixis Overview

- Excellent data sets on several compounds
  - Ultimate goal is marketed compounds
  - Making significant progress towards pivotal trials and market
- Compounds with weaker data sets will be stopped
  - Large pipeline enables choices to put resources behind compounds with greatest potential
- Moderate net cash use
  - Continued ability to generate non-dilutive cash going forward
- Several significant upcoming events
  - Data presentations
  - Opt-in decisions, milestones
  - New partnerships

Exelixis R&D Day - Agenda

Welcome	G. Scangos
Exelixis 2007 R&D in Review	M. Morrissey
Resistance in NSCLC	V. Miller
• XL647 NSCLC Pivotal Trial Plans	G. Schwab
Therapeutic Opportunities in Thyroid Cancer	S. Sherman
• XL184 MTC Pivotal Trial Plans	G. Schwab
Exelixis 2008 Pipeline – Intro	M. Morrissey
• Clinical Update	G. Schwab
• Preclinical Update	P. Lamb
Financial Overview	F. Karbe
Closing	G. Scangos
Senior Management Q&A	

R&D Overview
Michael Morrissey, President R&D

2007 - Transformational Year for Exelixis

Product Focus – Main strategic driver for company

- Numerous near-term shots-on-goal for oncology products
- Potential large commercial upside in broad range of tumor types

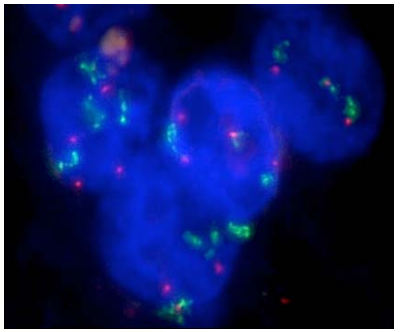
Integrated R&D group realigned to maximize success

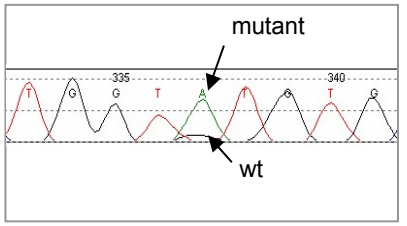
- Exelixis discovery continues to deliver high quality candidates
- Integration of translational medicine into R&D process
- Enhanced development productivity and output
  - 30 active clinical trials in 2007
  - ~300 patients enrolled in Exelixis clinical trials in 2007

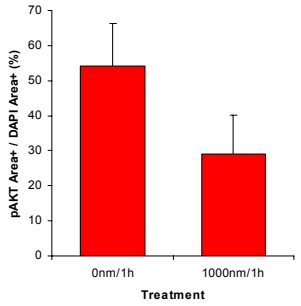
Translational Medicine in 2007

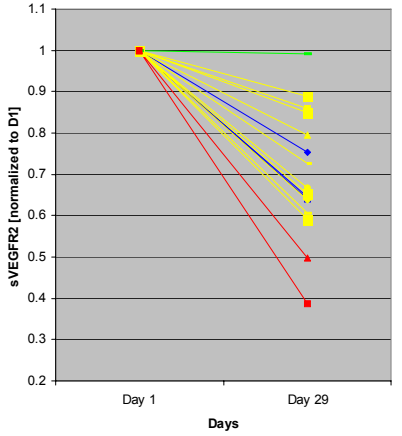
Broad implementation of translational medicine approaches across all clinical programs

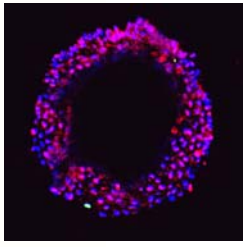
- Genotyping of patient tumor samples
- Plasma biomarkers and proteomics
- Circulating tumor cell analyses
- Histological analyses
- Key preclinical data to help guide development strategies

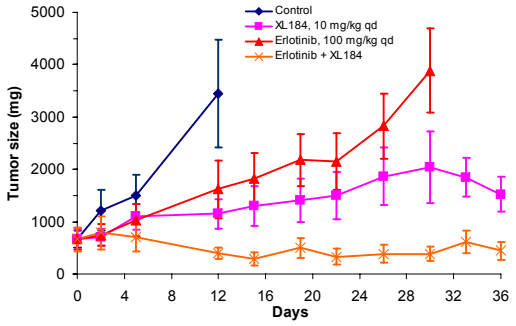












2007 - Transformational Year for Exelixis

Early stage assets rapidly advancing into full development

POC data obtained & report submitted in 2007

- XL647 (NSCLC), XL880 (PRC), XL784 (DN) submitted to GSK
- Additional opt-in decisions in 2008

Clinical activity observed for XL184 (MTC) & XL019 (MF)

- Initiate pivotal trials for XL647, XL019 and XL184 in mid-2008
- XL019 phase 1 data in MF to be presented at ASH on Dec 10th

Push to POC in 2007: Plan, Generate & Present Key Data

Advance early pipeline - rapid go/no go decisions

- Shift POC activities to Phase 1 from Phase 2 when feasible

Exelixis POC criteria:

- Good human PK
- Acceptable tolerability at exposure that leads to anti-tumor activity in preclinical models
- Plasma biomarker and tumor PD
- Objective responses in enriched patient populations with appropriate genetic lesions

POC in Phase 1 = XL184, XL019, XL518, XL147 & XL765

Push to POC in 2007: Plan, Generate & Present Key Data

XL647, XL880, XL184, XL518 and XL019***

- Potential first-in-class and/or best-in-class compounds
- POC drives partner's opt-in decision and defines ownership

Key scientific meetings = forum for data release

- ISLAC, ASCO, ASH, EORTC, ASN = high impact meetings
- XL880 oral presentation at ASCO
- 13 posters at EORTC
- 4 abstracts at ASH with oral presentations for XL019 & XL228

Increase transparency of data and news flow

Key 2007 YTD Milestones

5 DCs and pre-DCs:	XL139, XL888, XL652, EXEL-5413, EXEL-5465
4 INDs:	XL418, XL147, XL765, XL019
7 "First Time in Human" (FTIH) studies initiated:	XL418, XL281, XL518, XL228, XL765, XL147, XL019
5 "First Patient In" (FPI) studies initiated:	XL844-002, XL880-204, XL647-203, XL880-203, XL999-002
Implement Phase 1 POC:	XL184, XL019, XL765, XL147 & XL518
POC Data for XL647:	Presented to GSK (May); IASLC (Sept)
POC Data for XL880:	Presented to GSK (Sept); EORTC (Oct)
POC Data for XL784:	Presented to GSK (Oct); ASN (Nov)

Exceed Goals and Expectations for R&D collaborations

GSK collaboration

- POC data reports submitted for XL647, XL880 and XL784

BMS oncology drug discovery:

- Extremely strong start to collaboration – update in 2008

Genentech MEK Inhibitor collaboration

- XL518 phase 1 investigation advancing quickly – update in 2008

BMS LXR discovery collaboration:

- DC selected - FTIH on track for December; collaboration extended

BMS oncology Target ID:

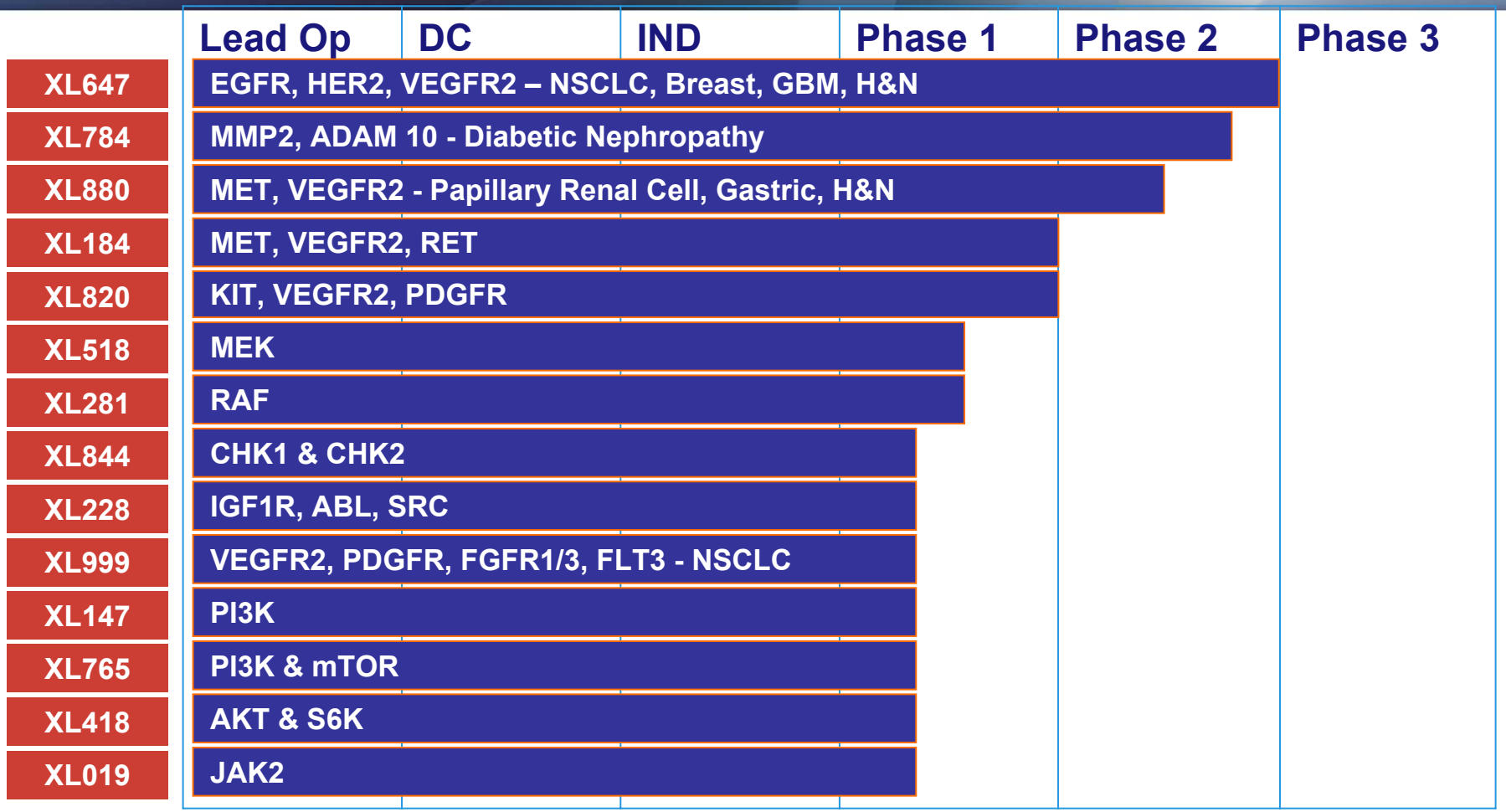
- Additional targets identified leading to milestone payment

GNE Notch:

- Key reagents/assays delivered; expand regenerative medicine capability

Up to Five Opt-in Decisions in 2008

Exelixis Pipeline



The compounds XL647, XL784 and XL999 have been out-licensed to Symphony Evolution, Inc. and are subject to a repurchase option. Pursuant to a product development and commercialization agreement between Exelixis and GlaxoSmithKline, GlaxoSmithKline has the option, after completion of clinical proof-of-concept by Exelixis, to elect to develop up to three compounds in Exelixis' product pipeline, including XL784 and XL999, but excluding XL647, XL518, XL147, XL765 and XL019. Finally, the compound XL518 is the subject of a co-development collaboration between Genentech and Exelixis.

Exelixis Pipeline – 2008 Preview

Three compounds moving into pivotal trials

- XL647 (NSCLC), XL184 (MTC) and XL019 (MF)

Numerous compounds moving into phase 2

- XL147/765 (PI3K), XL518 (MEK), XL820 (KIT), XL281 (RAF)

Two compounds discontinued

- XL418 (low exposure), XL999 (CV safety)
- De-prioritize XL784 investment pending internal review

New compounds advance from preclinical

- DCs: XL139 (HH), XL888 (HSP90), XL652 (LXR)
- Pre-DCs: EXEL-5413 (cdc7), EXEL-5465 (11β-HSD)

Acquired Resistance to EGFR Tyrosine Kinase Inhibitors in Non-small Cell Lung Cancer

Vincent A. Miller, M.D.
Thoracic Oncology Service
Memorial Sloan-Kettering Cancer Center

Lung Cancer

Divided into small cell (15-20%) and non-small cell carcinomas (80-85%)

Leading cancer killer of men and women in U.S.

~ 1,000,000 cases annually world wide

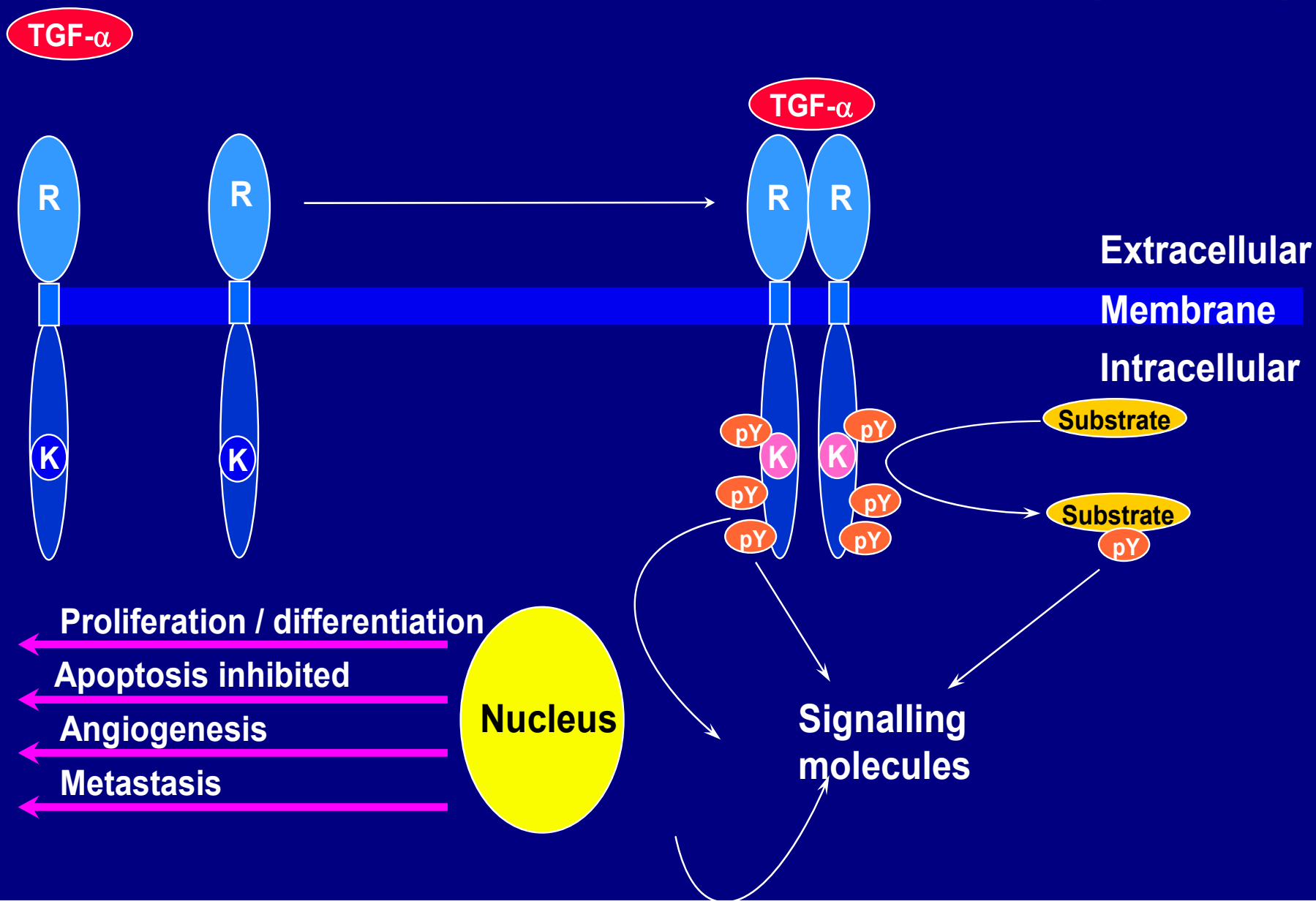
Epidemiology markedly different in Pacific Rim countries (e.g.- South Korea)

EGFR TK domain mutations:

~ 13% of US lung cancers

~ 30-40% of lung cancers in Pacific Rim

Epidermal Growth Factor Receptor (EGFR)



EGFR Hypothesis in NSCLC

Block EGFR-mediated effects

Arrest non-small cell lung cancers *dependent* on EGFR signaling

EGFR Mutations in Lung Cancer: Correlation with Clinical Response to Gefitinib Therapy

J. Guillermo Paez,[1,2*] Pasi A. Jänne,[1,2*] Jeffrey C. Lee,[1,3*]
Sean Tracy,[1] Heidi Greulich,[1,2] Stacey Gabriel,[4] Paula Herman,[1]
Frederic J. Kaye,[5] Neal Lindeman,[6] Titus J. Boggon,[1,3]
Katsuhiko Naoki,[1] Hidefumi Sasaki,[7] Yoshitaka Fujii,[7]
Michael J. Eck,[1,3] William R. Sellers,[1,2,4†]
Bruce E. Johnson,[1,2†] Matthew Meyerson[1,3,4†]

Activating Mutations in the Epidermal Growth Factor Receptor Underlying Responsiveness of Non–Small-Cell Lung Cancer to Gefitinib

Thomas J. Lynch, M.D., Daphne W. Bell, Ph.D., Raffaella Sordella, Ph.D., Sarada Gurubhagavatula, M.D., Ross A. Okimoto, B.S., Brian W. Brannigan, B.A., Patricia L. Harris, M.S., Sara M. Haserlat, B.A., Jeffrey G. Supko, Ph.D., Frank G. Haluska, M.D., Ph.D., David N. Louis, M.D., David C. Christiani, M.D., Jeff Settleman, Ph.D., and Daniel A. Haber, M.D., Ph.D.

EGF receptor gene mutations are common in lung cancers from "never smokers" and are associated with sensitivity of tumors to gefitinib and erlotinib

William Pao*†¶, Vincent Miller†§, Maureen Zakowski¶, Jennifer Doherty*, Katerina Politi*, Inderpal Sarkaria‖, Bhuvanesh Singh‖, Robert Heelan**, Valerie Rusch‖, Lucinda Fulton††, Elaine Mardis††, Doris Kupfer††, Richard Wilson††, Mark Kris†§, and Harold Varmus*

2004

Prospective Trials of EGFR-TKIs in Patients with EGFR Mutations

Study	Agent	RR (%)	***PFS****	OS*/1 yr (%)
Paz-Ares	Erlotinib	31/38 (82)	***13.3***	NR/81
Morikawa	Gefitinib	13/20 (65)	***N.R.***	NR
Sunaga	Gefitinib	16/21(77)	***13***	NR
Sutani	Gefitinib	21/27 (77)	***9.4***	15.4/NR
Inoue	Gefitinib	12/16 (75)	***9.7***	NR
Asahina	Gefitinib	12/16 (75)	***8.9***	NR
		105/138 (76)		

***measured in months**

iTARGET (Sequist, et al)

1st prospective U.S. trial

Exons 18-24 sequenced

Response rate 55% (95% CI 36-73)

- Slightly higher (65%) in exon 19 del and exon 21 L858R

Median PFS 11.4 months

Median OS 20.8 months/1 yr survival 73%

No additive value of FISH to mutation analysis

GECP 06/01
opened February 2007

Grupo Español de Cáncer de Pulmón
Spanish Lung Cancer Group

- **Stage IV NSCLC**
- **Mutations in exon 19 or 21 of *EGFR*.**
- **Measurable or evaluable disease.**
- **ECOG PS ≤ 2.**

***Stratified by*:**

- **ECOG PS**
- **In-frame del (exon 19) *vs*. L858R (exon 21)**
- **Mutation analysis in serum vs. both in serum and tumour tissue.**

→ **RANDOMIZE**

Arm A
- **Erlotinib** 150 mg/day

N: 146 patients

Arm B

- **CDDP** 75 mg/m^2 plus **Gemcitabine** 1.250 mg/m^2

OR

- **CDDP** 75 mg/m^2 plus **Docetaxel** 75 mg/m^2

Courtesy of Dr. Rafael Rosell

Acquired Resistance: Background

Virtually all patients sensitive to gefitinib or erlotinib eventually have progression of disease.

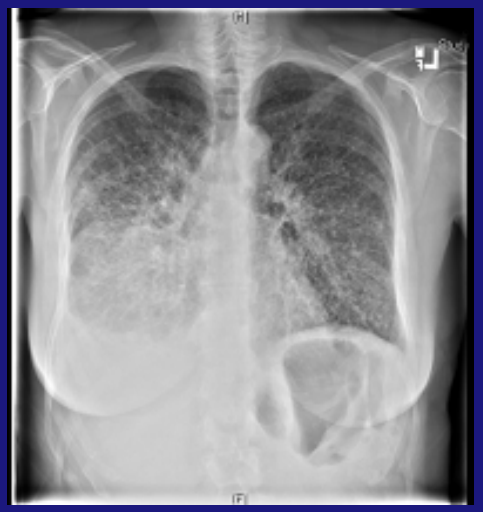
Patients with CML have a high rate of response to treatment with imatinib.

A proportion of patients acquire resistance after an initial response.

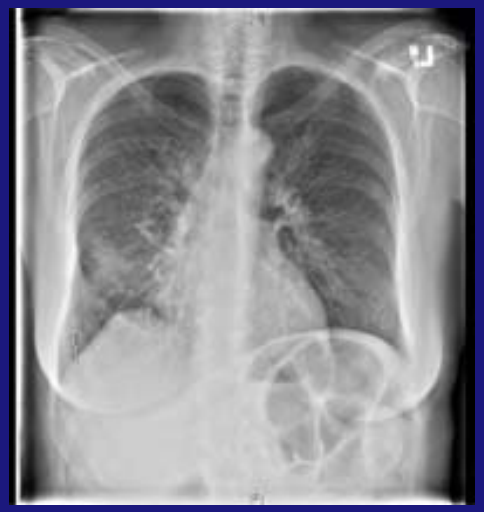
In vivo **there are a small number of conserved changes in the ABL tyrosine kinase domain which confer resistance.**

Other patients with acquired resistance have amplification of the *BCR-ABL* gene.

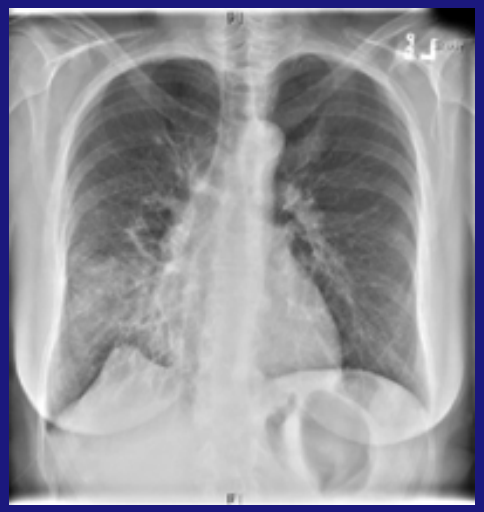
Patient 1 – Response and Progression



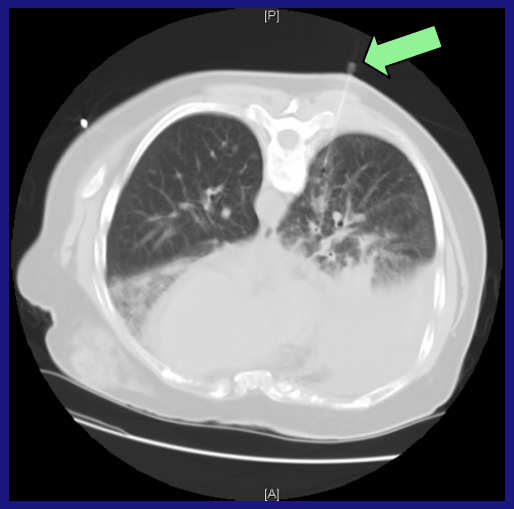
Day 0



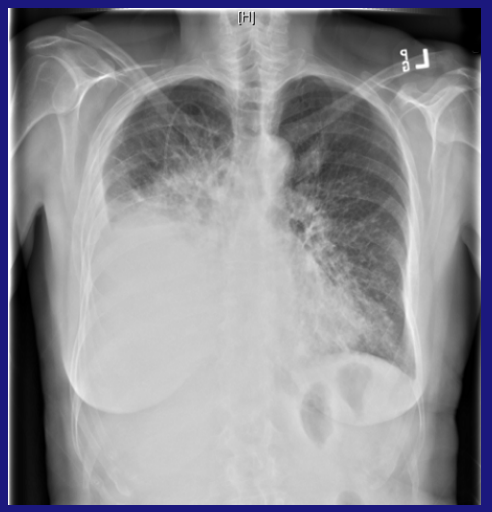
14 days



9 months

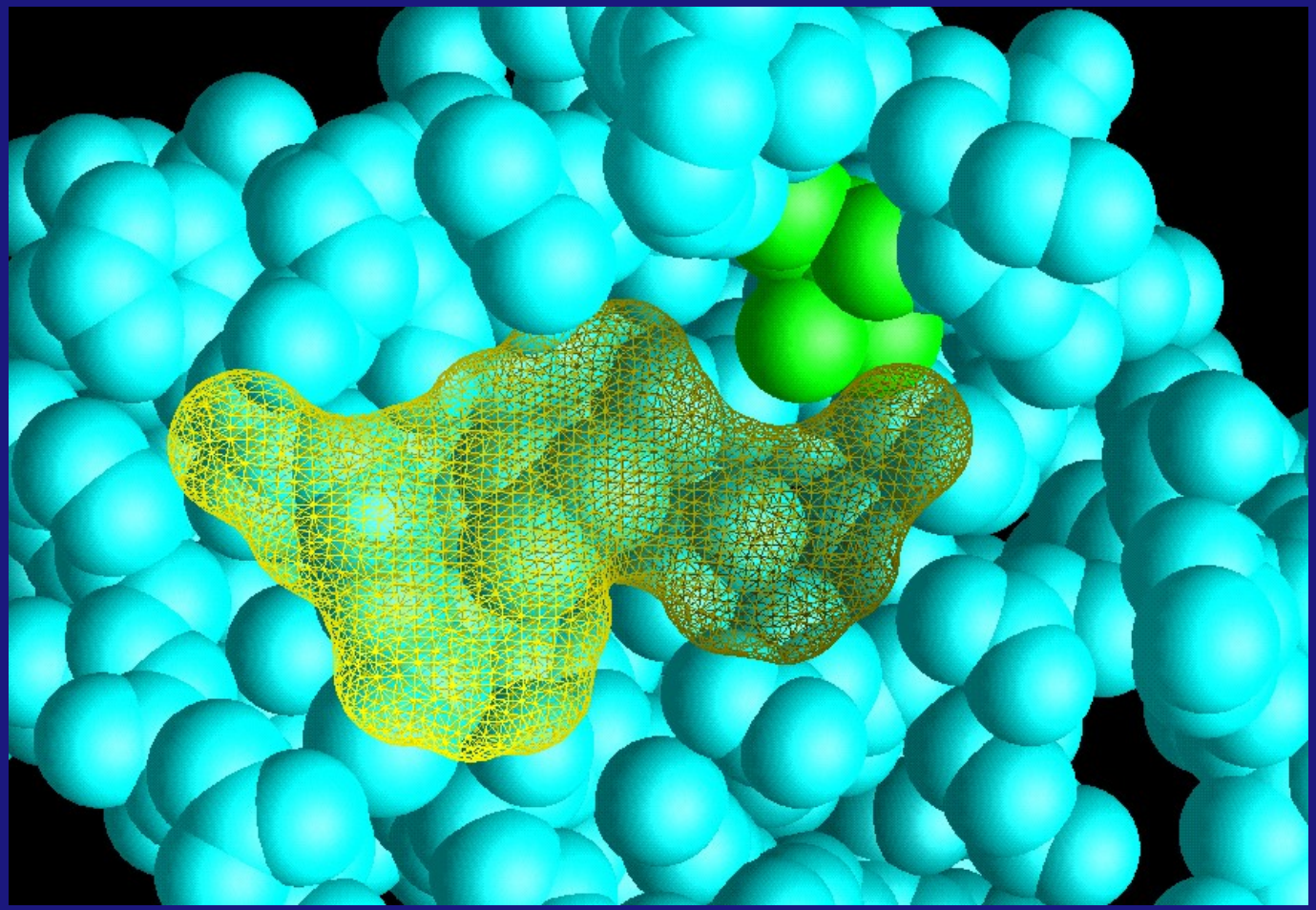


CT-guided biopsy



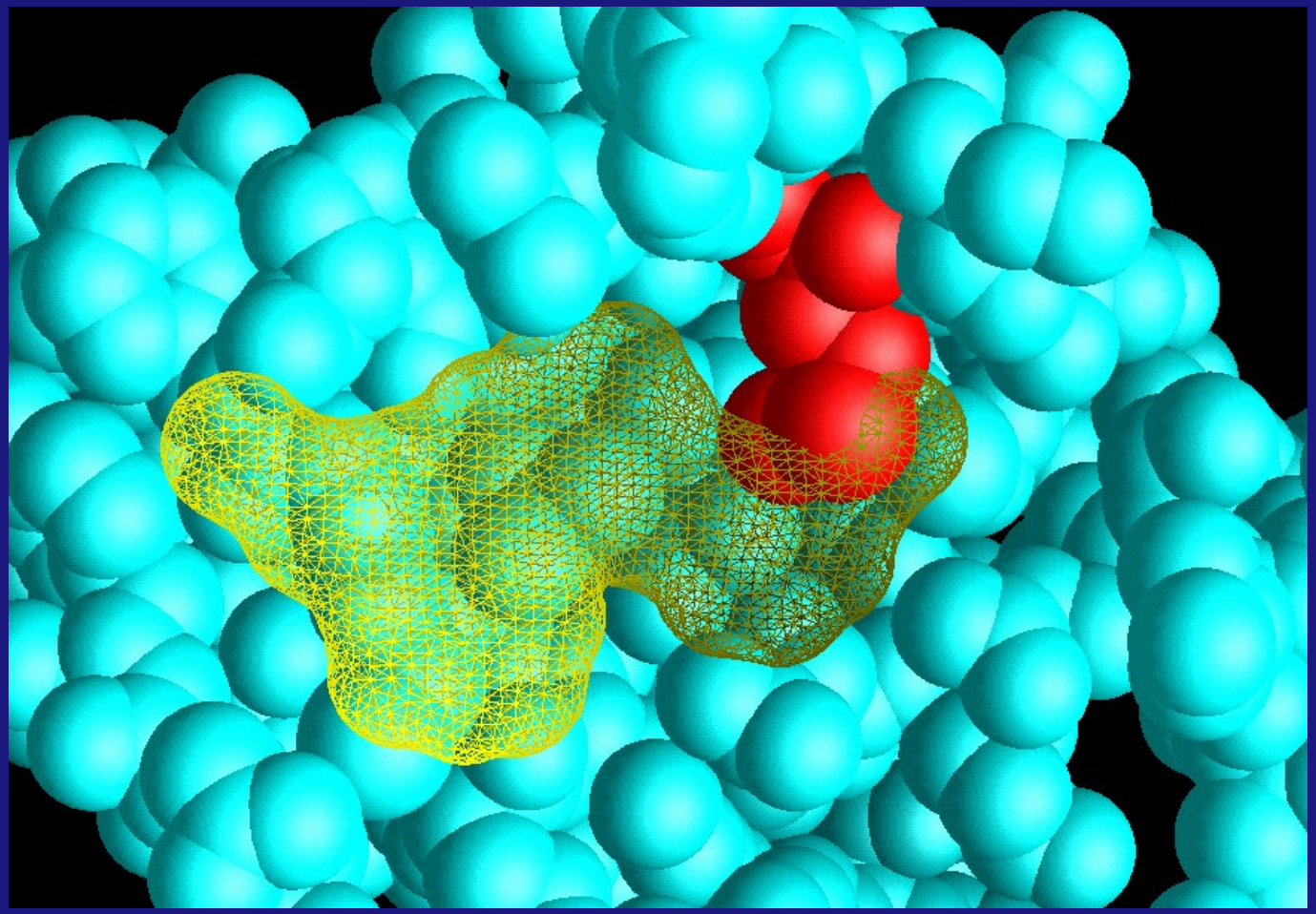
12 months

Erlotinib and T790



Courtesy of Nikola Pavletich

Erlotinib and T790M



Courtesy of Nikola Pavletich

T790M is Common in Acquired Resistance to Gefitinib or Erlotinib

Report	Frequency of T790M
Balak *CCR* (MSKCC)	9/21
Kobayashi *NEJM*	1/1
Kwok *PNAS*	2/2
Gow *PLoS Med*	1/1
Mitsudomi *CCR*	7/14
Total	20/39

Balak et al CCR 2006,

What About Other Mechanisms of Acquired Resistance?

Array CGH

244K Agilent chips

12 patients, 12 samples with acquired resistance

Compare to aCGH data (44K chips) from 38 patients -- all EGFR mutant never treated with TKI

MET Amplification in Acquired Resistance

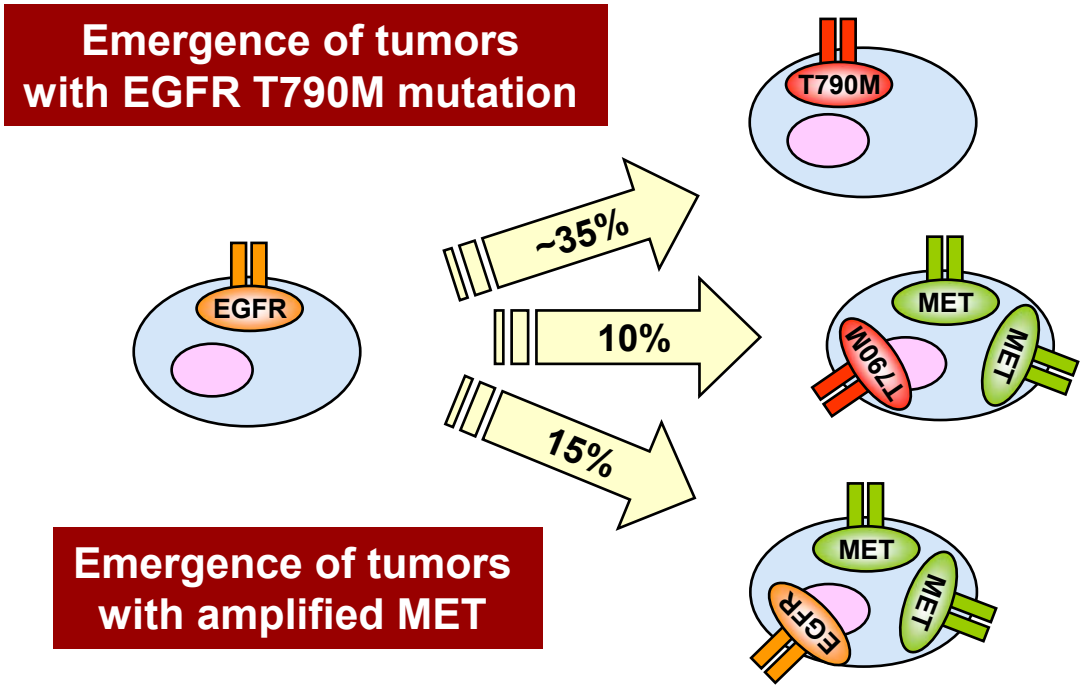
9/43 (21%) with increased copies of MET relative to MTHFR by q PCR

Among 10 tumors from 9 patients:

- **4 with T790M**
- **6 without T790M**

2 had met amplification pre-treatment

Mechanisms Driving Relapse of NSCLC Tumors Following Response to EGFR Inhibitors

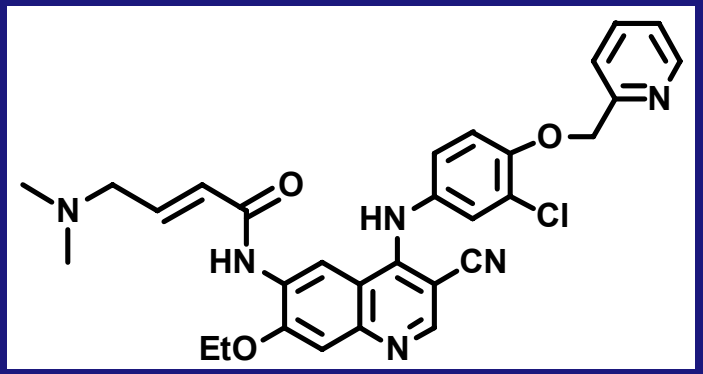


Treat erlotinib – relapsed patients
Treatment up front may prevent or delay emergence of resistant tumors

Strategies to Overcoming Acquired Resistance to EGFR-TKIs in the Clinic

- Change in dose or schedule of EGFR-TKI
- Change to a "different" TKI-e.g.-BIBW 2992, CI1033, EKB569, HKI-272, XL647
- Use of geldanamycin derivatives (e.g.-IPI-504)
- Met inhibition

HKI-272



- **Irreversible** pan erbB receptor tyrosine kinase inhibitor

In Vitro ErbB Receptor Tyrosine Kinase Assay of HKI-272 Inhibition

	IC_{50} (nM)		
Assay	**EGFR**	**HER2**	**HER4**
Autophosphorylation	**92**	**59**	**-**
Substrate phosphorylation	**12**	**39**	**19**

Rabindran et al. Cancer Res 64: 3958, 2004

- **Covalently** binds to EGFR and HER2 at the ATP binding site and inhibits tyrosine kinase activity

Biologic Activity of HKI-272

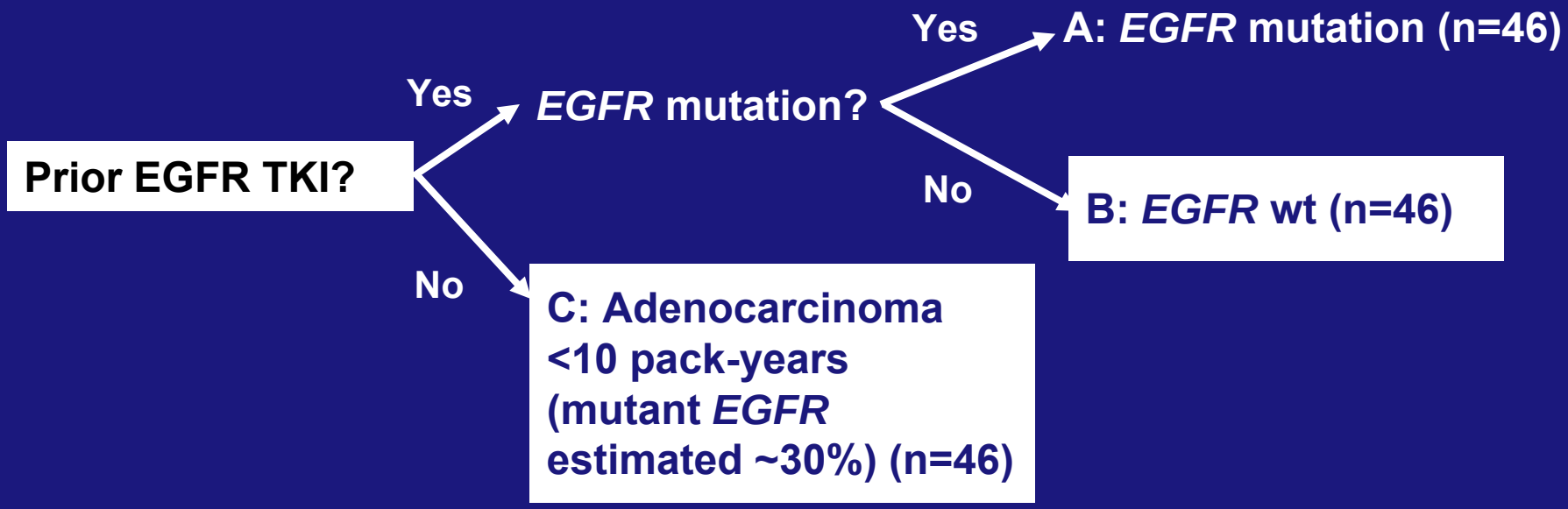
- **Inhibits the growth of tumor cells in culture or xenografts that:**
  - **Express EGFR and HER2**
  - **Contain sensitizing and resistance-associated EGFR mutations (Kwak et al, Proc Natl Acad Sci USA 102:7665, 2005)**
- **Shrinks *de novo* mouse lung tumors driven by:**
  - **EGFR kinase domain mutations (Ji et al, Cancer Cell, in press)**
  - **EGFRvIII mutations (Ji et al, Proc Natl Acad Sci USA, in press)**

HKI-272 Phase 1 Study: Conclusions

HKI-272 was administered to patients with solid tumors on a once-daily oral treatment schedule.

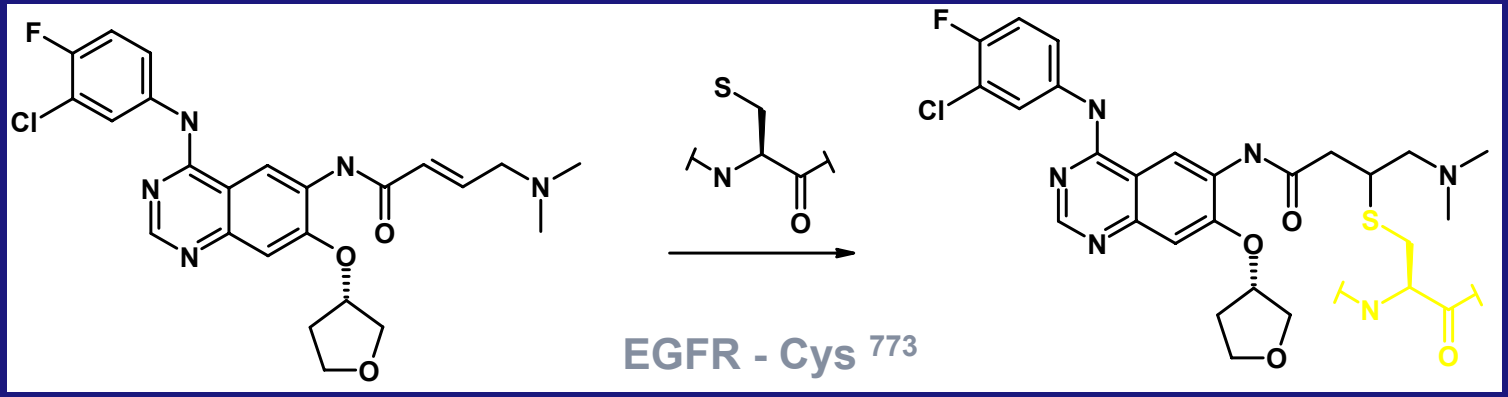
- **The MTD was 320 mg HKI-272.**
- **Safety observations**
  - **The most frequently occurring grade 1-3 HKI-272- related treatment-emergent adverse events (TEAEs) were diarrhea, nausea, and asthenia.**
  - **The most frequently occurring grade 3 HKI-272-related TEAEs were diarrhea and asthenia.**
  - **No grade 4 HKI-272-related TEAEs occurred.**

Study Populations: HKI-272 Phase 2 study



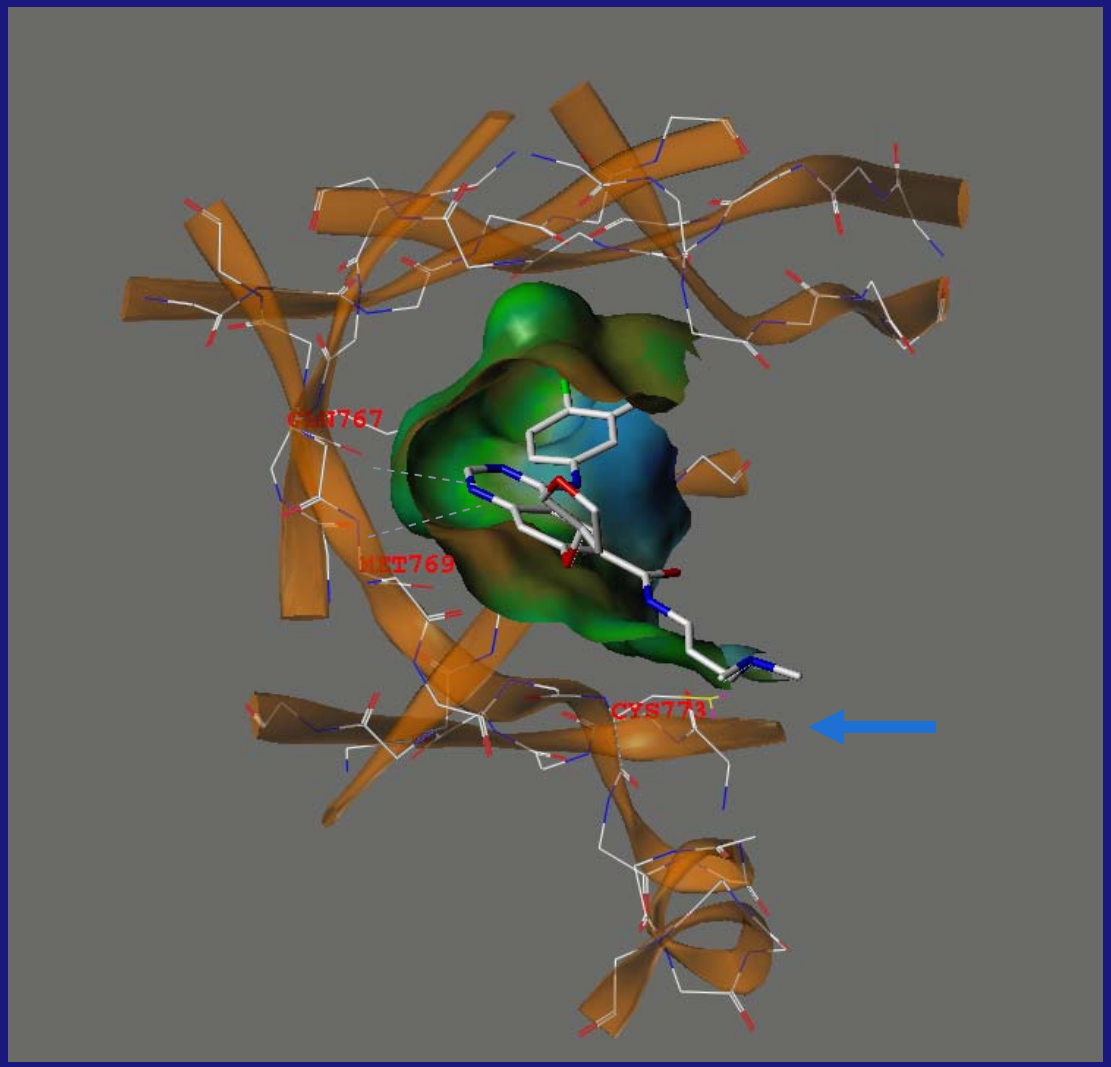
BIBW 2992 - Irreversible Dual EGFR /HER2 Inhibitor

- Anilino quinazoline derivative
- Irreversible dual EGFR/HER2 inhibitor
- Binds covalently to Cys ^{773} of the EGFR and Cys ^{805} of HER2



Solca et al., 17th EORTC-NCI-AACR Symposium on " Molecular Targets and Cancer Therapeutics“ Philadelphia, PA, USA, November 14 – 18, 2005

BIBW 2992 Docking to EGFR



Kinase Inhibition Profile
IC_{50}-

EGFR Kinase	**0.5 nM**
HER2 Kinase	**14 nM**

BIRK
>100,000 nM

VEGFR-2	**>100,000 nM**
HGFR	**13,000 nM**
c-src	**> 4,000 nM**

Solca et al., 17th EORTC-NCI-AACR Symposium on " Molecular Targets and Cancer Therapeutics“ Philadelphia, PA, USA, November 14 – 18, 2005

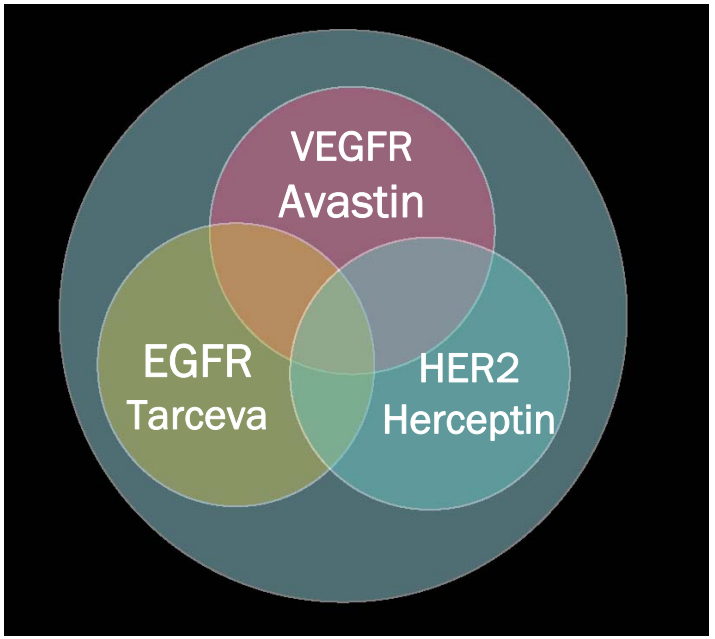
XL647 – Overview

Potent inhibition of kinase spectrum:

- **VEGFR2, EGFR and HER2**
- **Excellent PK**
- **Sustained inhibition of targets in preclinical PD models**

Active *in vitro* and *in vivo* against EGFR-T790M mutation

- **Preclinical H1975 model, erlotinib resistant**
- **Clinical Cancer Research 13, 3713, 2007**



XL647 Inhibits WT and Mutant EGFR

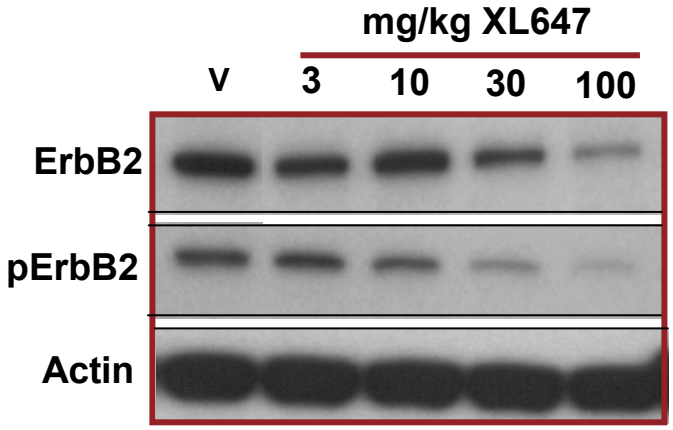
	XL647	ZD6474	gefitinib	erlotinib
EGFR_WT	12	>216	19	25
EGFR_L858R	7	>216	8	5
EGFR_L861Q	10	>216	15	15
EGFR_vIII	48	>3000	77	170

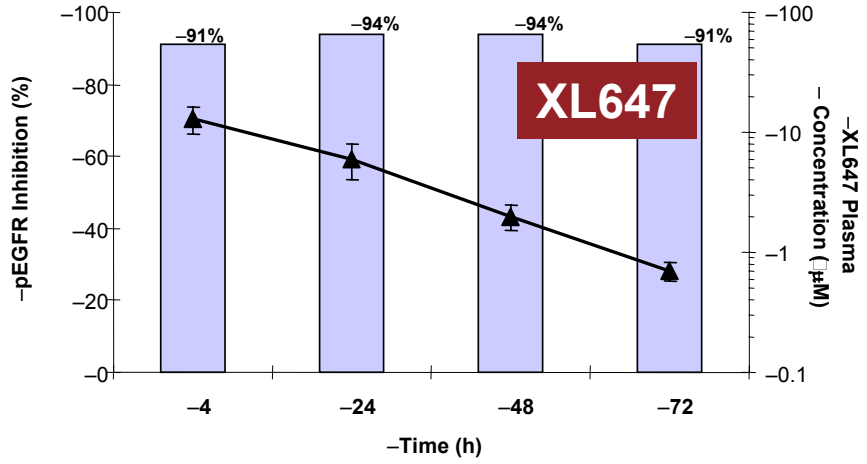
IC_{50} = nMol

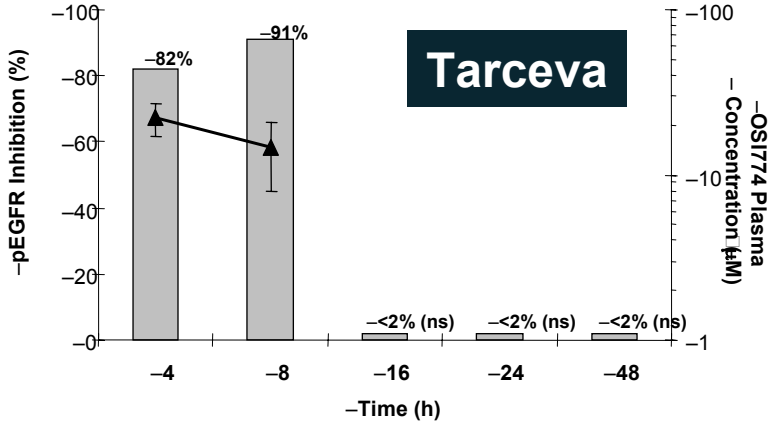
XL647 inhibits activated EGFR mutants in cells with similar or better potency compared to other EGFR-TKIs

XL647: In Vivo Target Modulation

	KDR	EGFR	ErbB2
Est. ED_{50} (mg/kg)	3.5	2.8	7
Duration of Action		>72 Hr	>72 Hr







Erlotinib-Resistant NSCLC Cell Line is Sensitive to XL647

EXELIXIS

XL647 inhibits the proliferation of cell lines expressing WT EGFR and L858R/T790M EGFR at clinically achievable concentrations

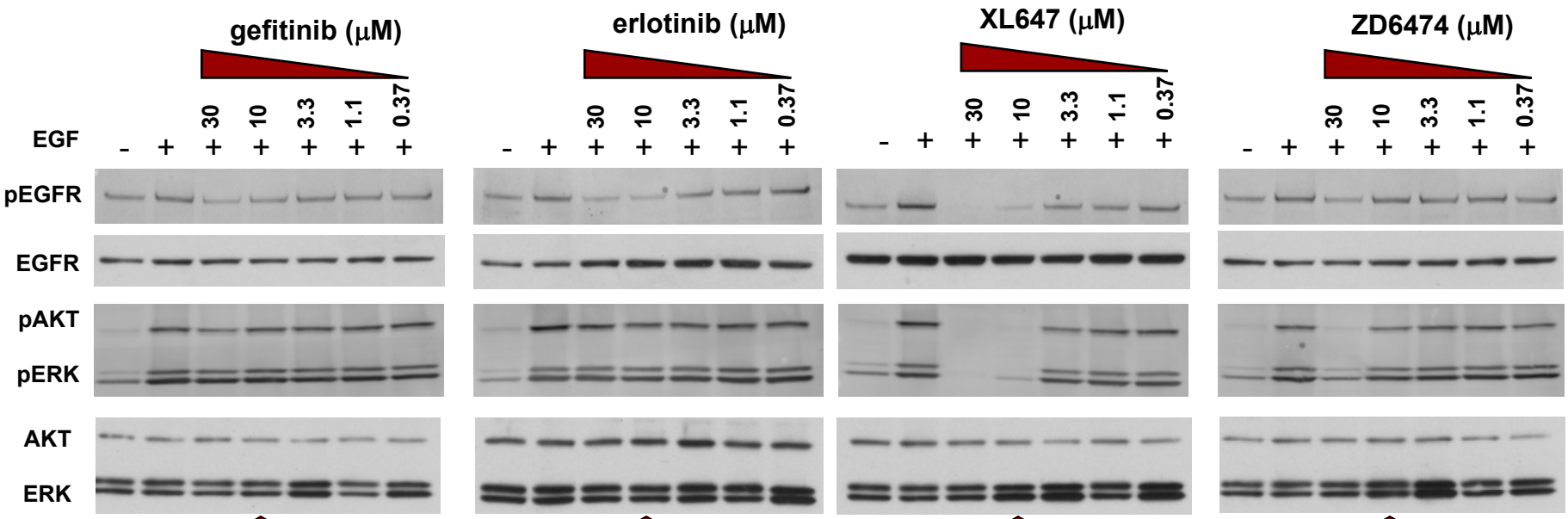
- **Erlotinib and gefitinib highly active against mutant EGFR**
- **Activity vs WT EGFR is at concentrations close to or above clinical Cmax**
- **Activity vs T790M form of EGFR at concentrations> clinical Cmax**

Cell Line	A431	H3255	H1975	Clinical Cmax (dose)
Genotype	EGFR WT*	EGFR L858R	EGFR L858R / T790M	
Erlotinib	2.3 uM	<0.005 uM	3.2 uM	2.5 uM (150 mg)
Gefitinib	6.0 uM	<0.005 uM	10.8 uM	1 uM (250 mg)
XL647	0.63 uM	0.002 uM	0.26 uM	0.8 uM (300 mg)

* highly amplified

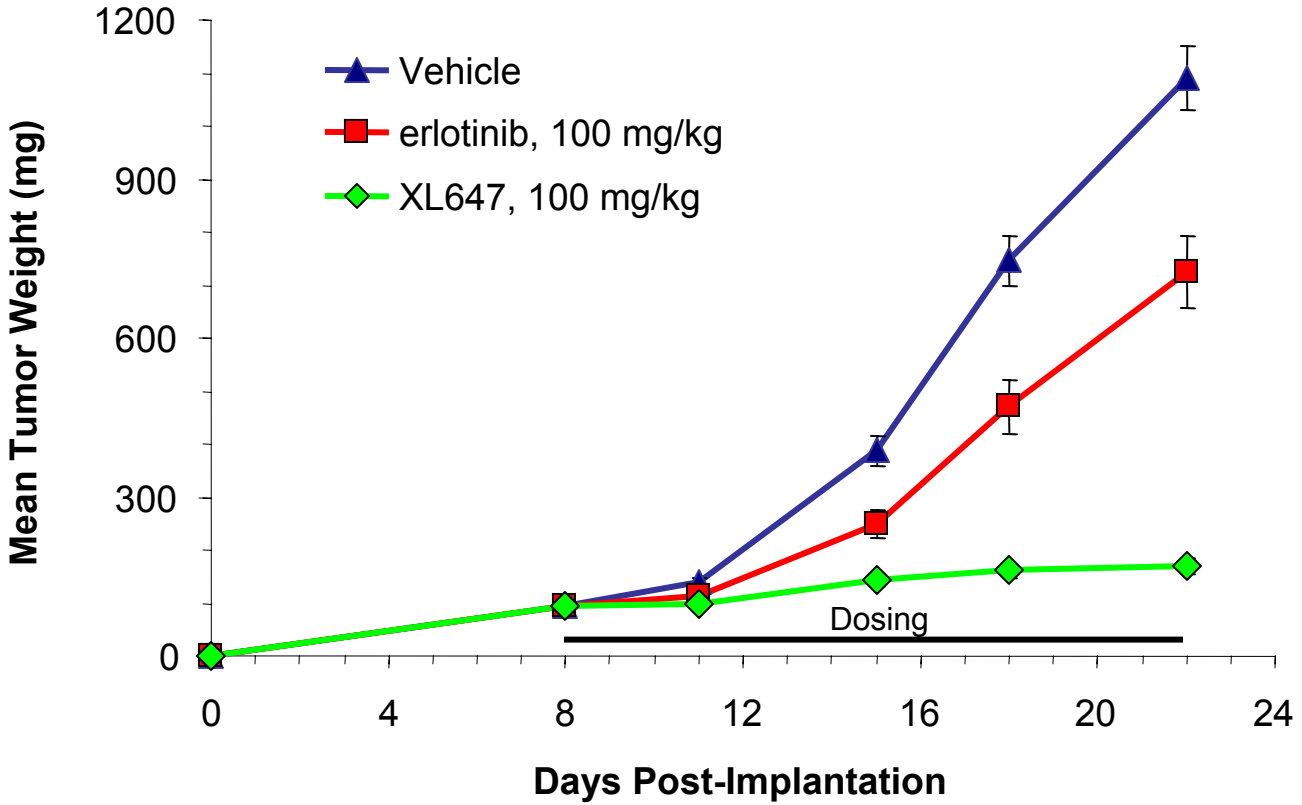
Proliferation IC_{50}s

XL647 Retains Activity in H1975 Cells



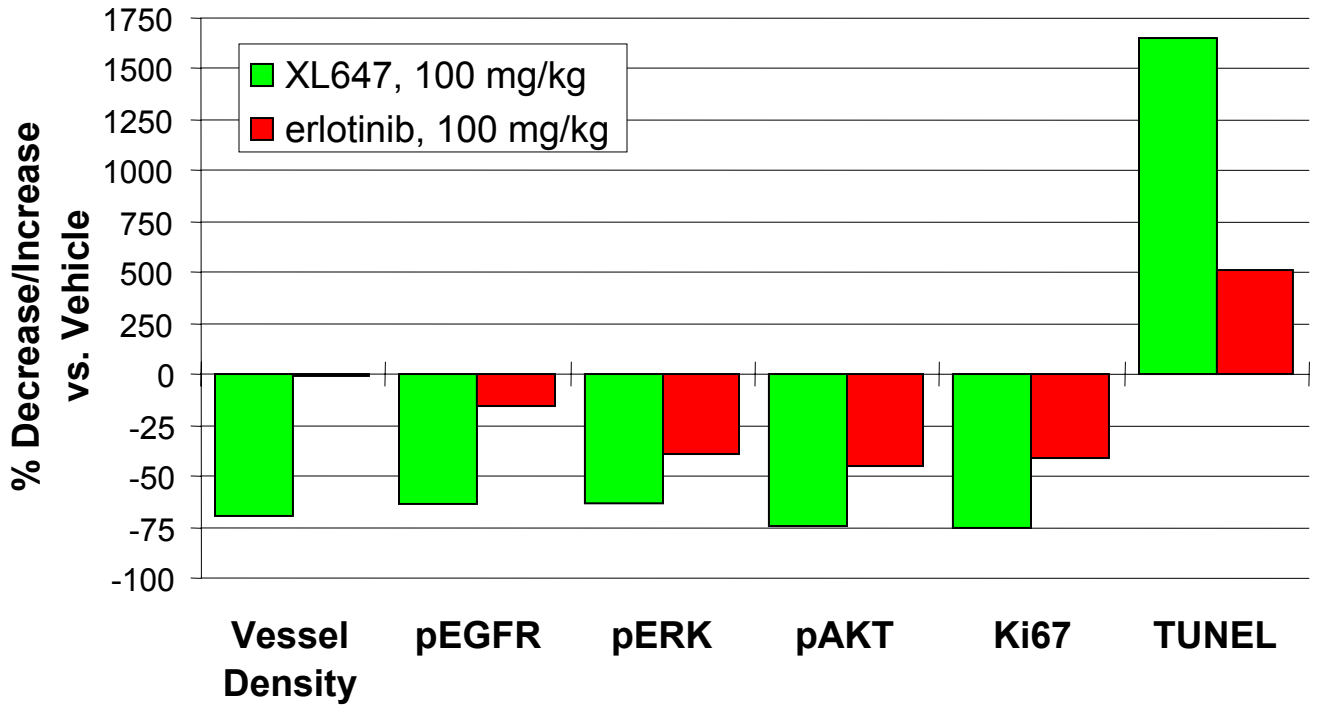
XL647 is more potent than erlotinib, gefitinib or vandetanib in the inhibition of signaling in H1975 cells

XL647 Inhibits the Growth of H1975 Tumors with T790M Erlotinib Resistant Mutation in EGFR



XL647 is superior to erlotinib in the H1975 human lung xenograft tumor model with mutant EGFR

XL647 – Targeting Mutant EGFR *In Vivo*



XL647 is superior to erlotinib for inhibition of multiple IHC endpoints in the H1975 xenograft tumor model

XL647 Clinical Studies

	Design	Schedule	Patient Population
Phase 1	3 + 3 Dose Escalation Expanded MTD	Intermittent 5 and 9	Advanced Solid Tumors
Phase 1	3 + 3 Dose Escalation Expanded MTD	Daily	Advanced Solid Tumors
Phase 2	Simon 2 Stage 1^{ary} Endpoint RR	Intermittent 5and 9 350 mg	1st Line NSCLC Clinically Selected
Phase 2	*Simon 2 Stage* *1^{ary} Endpoint RR*	*Daily* *300 mg*	*NSCLC* *Progression after Benefit from EGFRI*

EXELIXIS™

A Phase I Dose-Escalation and Pharmacokinetic (PK) Study of XL647, a Novel Spectrum Selective Kinase Inhibitor, Administered Orally Daily to Patients With Advanced Solid Malignancies (ASM)

Julian Molina,[1] Heather A. Wakelee,[2] Joanne Fehling,[2] Janet L. Lensing,[1] John Calcagni,[2] Roel P. Funke,[3] Dale Miles,[3] Branimir I. Sikic[1]

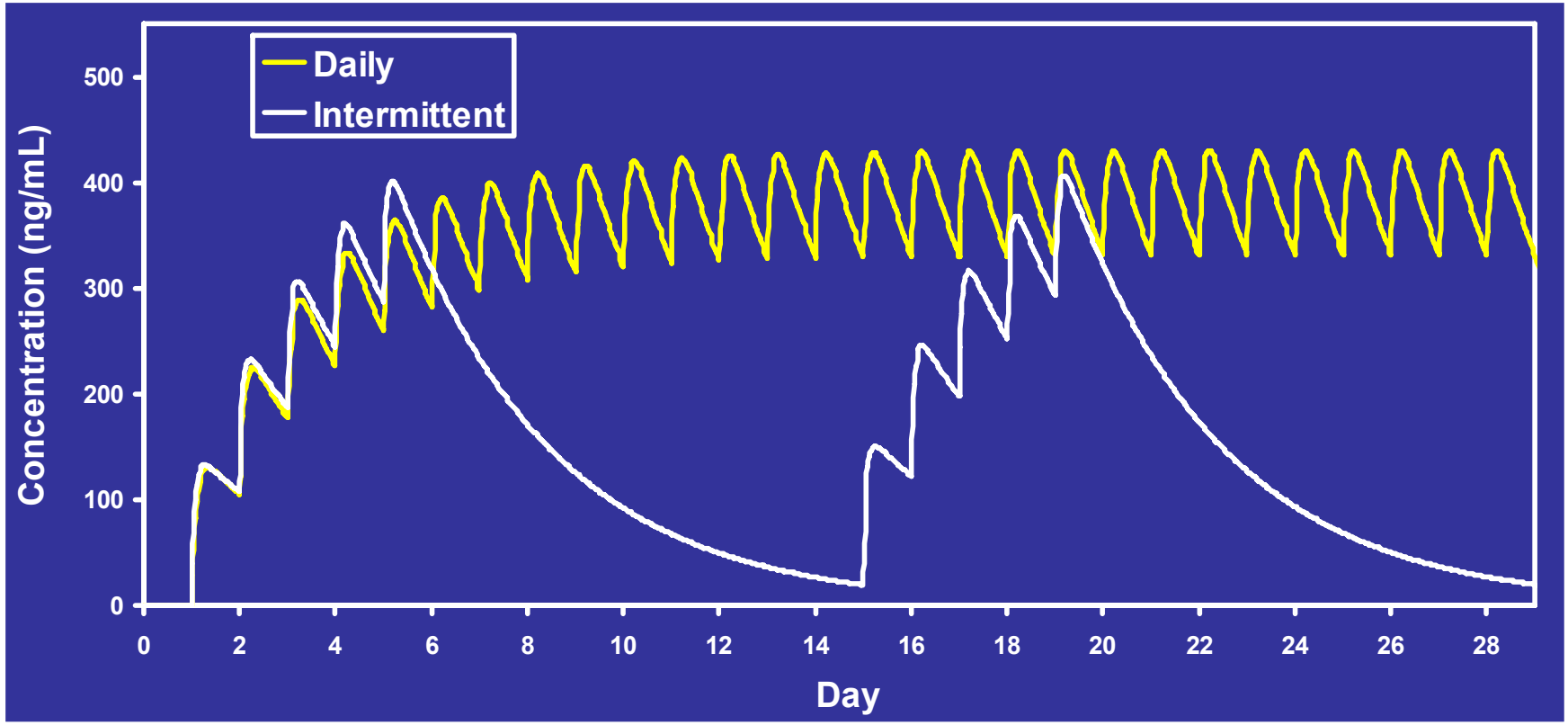
[1]Mayo Clinic, Rochester, MN; [2]Stanford University, Stanford, CA; [3]Exelixis, Inc., South San Francisco, CA

B242

XL647 Daily Dose Phase 1 Experience

- Daily oral XL647 was generally well tolerated
- The MTD has been determined to be 300 mg
- Pharmacokinetic steady state reached by approximately Day 15
- Once-daily administration XL647 (300 mg) is predicted to yield an approximately 2-fold increase in average exposure compared with intermittent 5 & 9 dosing (350 mg) while maintaining a similar tolerability profile
- > 3 months SD observed in 11/21 evaluable patients

XL647 PK: Daily vs 5 on 9 off Dosing



EXELIXIS™

A Phase II Study of XL647 in Patients With Non–Small Cell Lung Cancer (NSCLC) Enriched for Presence of EGFR Mutations

N. A. Rizvi,[1] M. Kris,1 V. Miller,[1] L. Krug,1 S. Bekele,[1] A. Dowlati,2 K. Rowland,[3] M.E. Tonda,[4] B. Mitchell,[4] J.C. Ruckdeschel,[5] R. Chaplen,[5] N. Aggarwal,[5] S. Gadgeel[5]

[1]Memorial Sloan-Kettering Cancer Center, New York, NY; [2]Case Western Reserve University, Cleveland, OH; [3]Carle Cancer Center, Urbana, IL; [4]Exelixis, Inc., South San Francisco, CA; [5]Karmanos Cancer Institute, Detroit, MI;

Baseline Characteristics

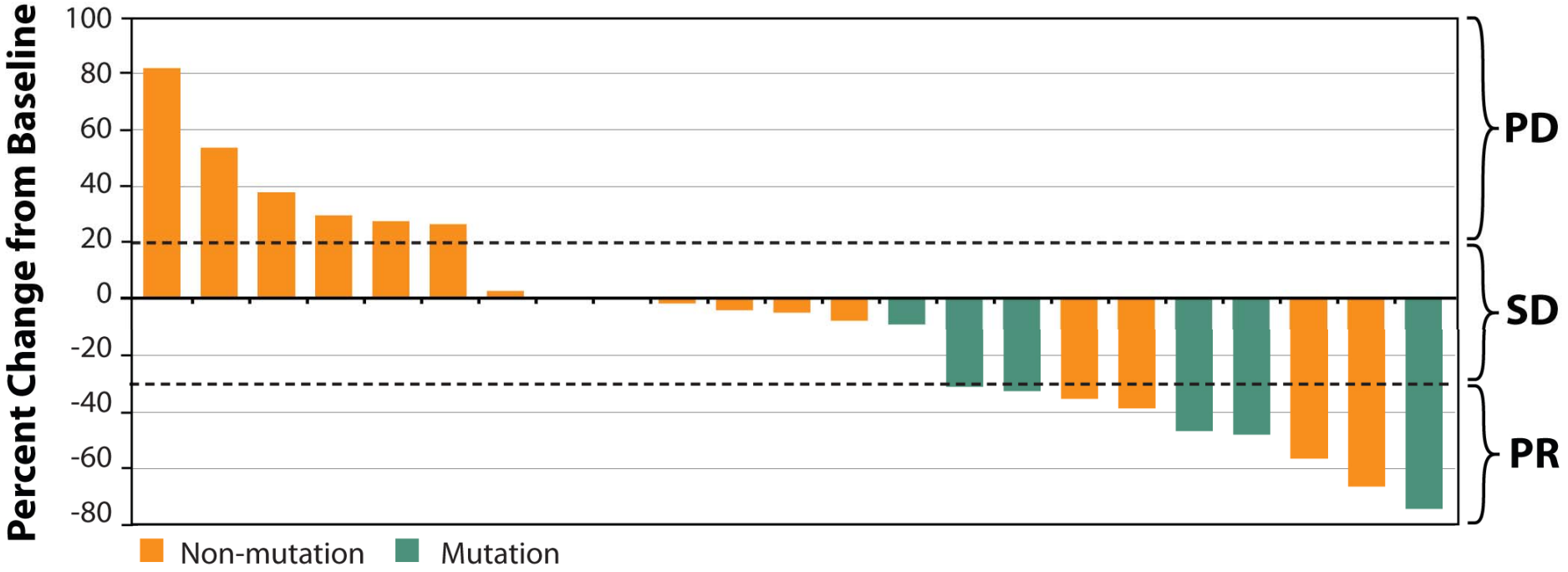
Characteristic	No. of patients (N = 37)
Median age (range), years	65 (44-86)
Gender (M/F)	11/26
Race	
Asian	1
White	31
African American	2
Pending	3
Smoking history	
Never	17
<15 pk-yr	12
>15 pk-yr	5
Pending	3
Tumor EGFR mutational status	
L858R	2
Exon 19 deletion	5
Wild type	13
NA[a]	17
Tumor KRAS mutational status	
Wild type	13
NA[a]	24

Response to XL647 in 1st Line NSCLC (n = 34)

	N	RR	Clinical Benefit PR + SD > 3 mths
First-line in clinically selected	34	~30% (10/34)	~68%
EGFR activating mutation	7	86% (6/7)	100% (7/7)
EGFR Wildtype	13	23% (3/13)	77% (10/13)
EGFR Status Pending	14	7% (1/14)	42% (6/14)

Time on study ranges up to 12+ months

Waterfall Plot: Best Response for Target Lesions by Patient Based on the Maximal Percentage of Tumor Reduction

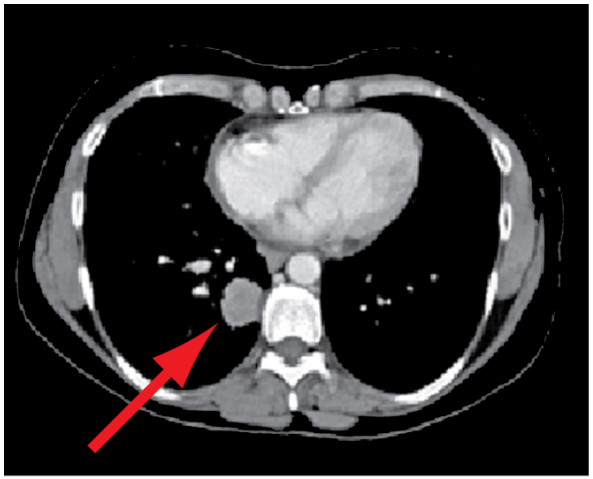


Five patients with PD are not included: 4 had new non-target lesions and 1 experienced clinical progression.
Six patients are not included due to pending tumor measurements (5 with SD and 1 with uPR [patient has *EGFR* exon 19 deletion])

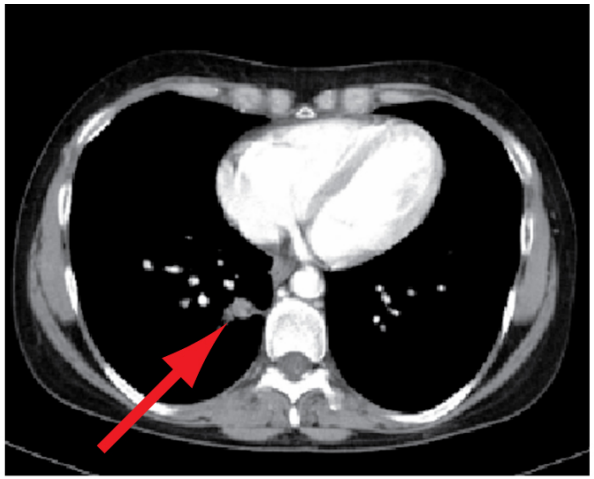
PD, progressive disease; SD, stable disease; PR, partial response.

Representative CT Scan Images

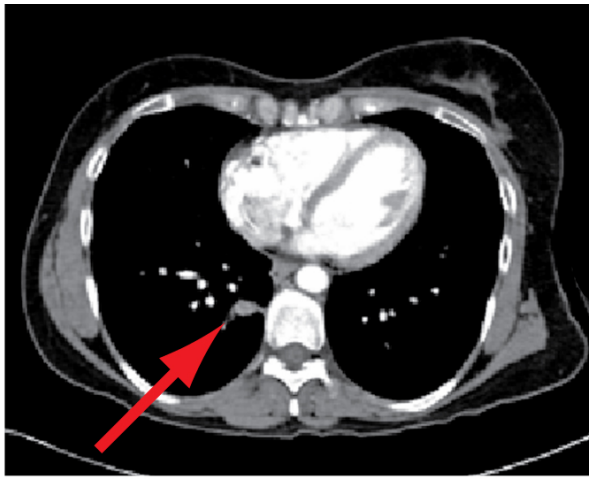
Patient with a documented EGFR exon 19 deletion who experienced a partial response.



Pre-treatment



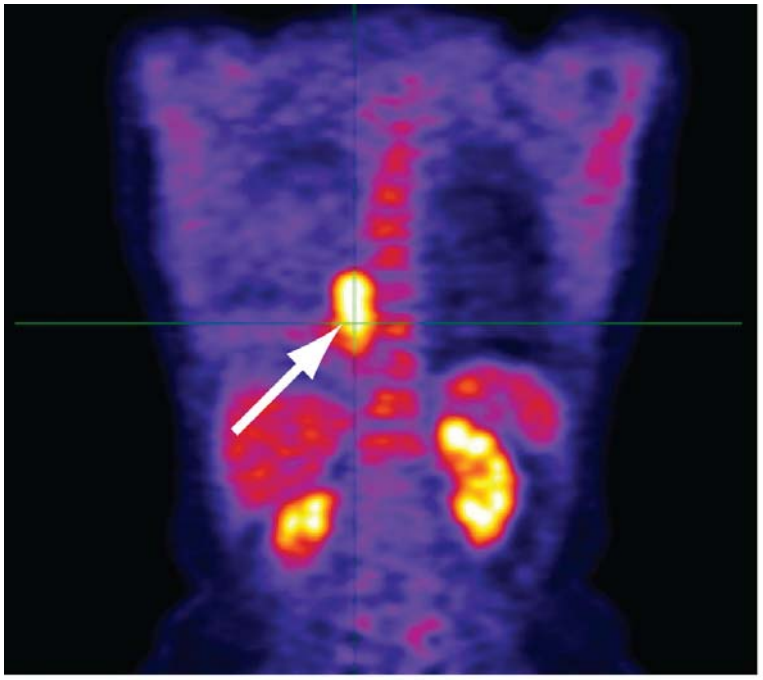
Post-treatment (8 wks)



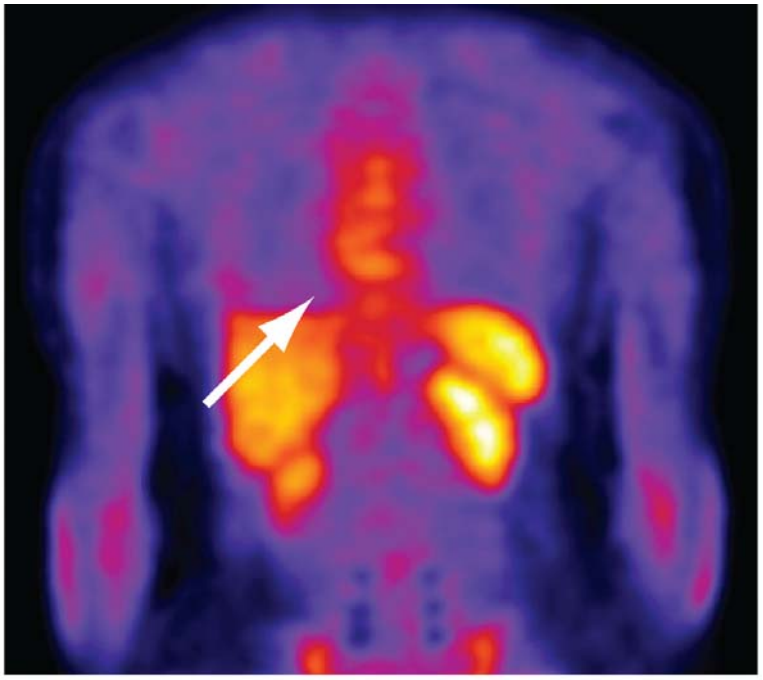
Confirmation (14 wks)

PET Scans (Same Patient as Previous Slide)

Baseline and after 11 weeks of treatment with XL647. No FDG-avid activity is evident in the post-treatment scan.



Pre-treatment



Post-treatment (11 wks)

Summary of XL647-Related Adverse Events (Reported by >10% of Patients; n = 32)

Adverse events[a]	No. patients (%) with severity Grade 1	No. patients (%) with severity Grade 2	No. patients (%) with severity Grade ≥3	Total no. patients
Diarrhea	15 (47)	2 (6)	–	17 (53)
Rash[b]	10 (31)	2 (6)	–	12 (38)
Fatigue	9 (28)	1 (3)	–	10 (31)
Nausea	8 (25)	–	–	8 (25)

[a]Adverse events graded using the Common Terminology for Adverse Events Version 3.0. Terms are the Preferred Term/MedDRA 10.
[b]Rash includes acne, rash, rash macular, rash maculo-papular, and rash pustular.

No dose modifications for rash or diarrhea

Clinically asymptomatic QTc prolongation: Grade 1: n = 8; Grade 2: n = 8
Grade 3: n = 2 of which 1 was ineligible due to Grade 2 QTc at baseline

Conclusions

- XL647 has demonstrated clinical benefit in 68% of patients (10 partial response and 13 stable disease as their best response).
- Patients with and without mutations responded to XL647:
  - 5 had EGFR exon 19 deletions,
  - 1 had an L858R point mutation,
  - 3 were wild type
  - 1 has pending mutational analysis
- All 7 patients with EGFR-activating mutations demonstrated clinical benefit (6 partial response, 1 stable disease).
- XL647 is generally well tolerated in this patient population.
- The most frequently reported adverse events assessed as being related to XL647 were diarrhea, rash, fatigue, and nausea, all of which were Grade 1 or Grade 2 in severity.

XL647 in EGFR-TKI Acquired Resistance

Patient Population:

- NSCLC
- Progressive disease following benefit from previous EGFR-TKI (≥ 12 week SD or PR)
- Expected frequency of T790M: 50% of patients

Simon 2 stage design

Daily dosing at 300 mg

Primary endpoint: RR

Actively accruing patients

XL647 Profile To Date

Generally well tolerated at doses of 350 mg qd on intermittent 5&9 and 300 mg daily dosing

- Few Grade 3 events related to XL647
- Rash
- Diarrhea
- Clinically asymptomatic QTc prolongation observed

Anti-tumor activity in NSCLC with 68% clinical benefit (PR and SD)

- Clear anti-tumor activity in tumors with EGFR activating mutations and wild type EGFR

Exposure with daily dosing at MTD approximately 2 fold exposure observed with intermittent 5&9 dosing at MTD with similar tolerability profile

MET Amplification in Acquired Resistance

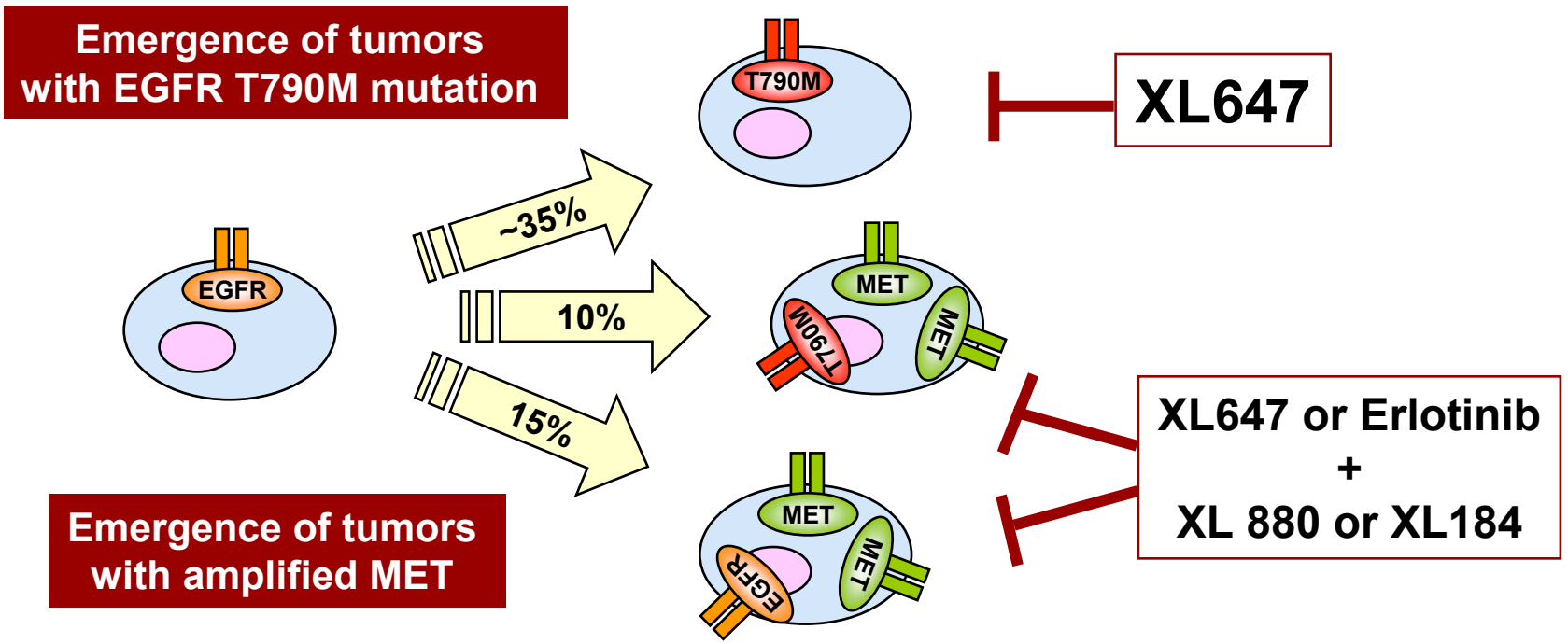
9/43 (21%) with increased copies of MET relative to MTHFR by q PCR

Among 10 tumors from 9 patients:

- 4 with T790M
- 6 without T790M

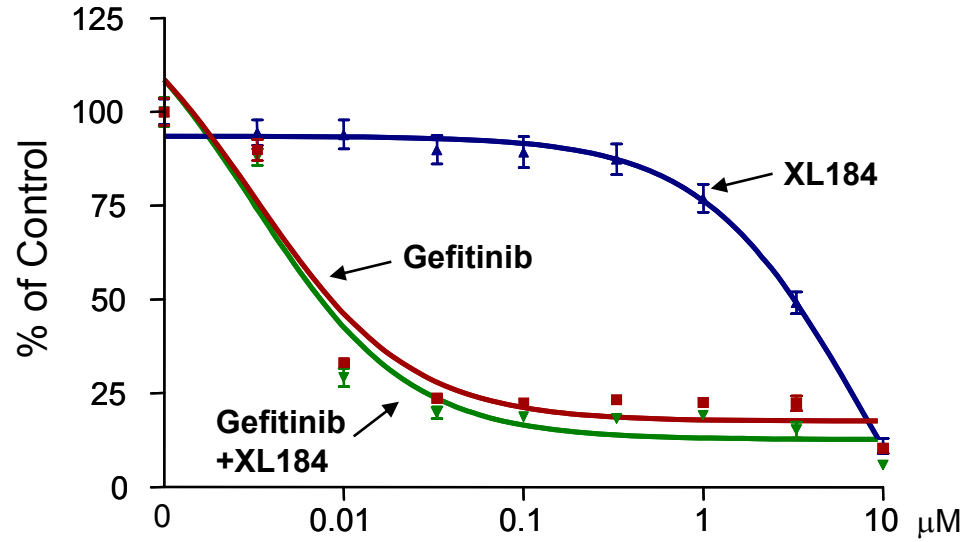
2 had met amplification pre-treatment

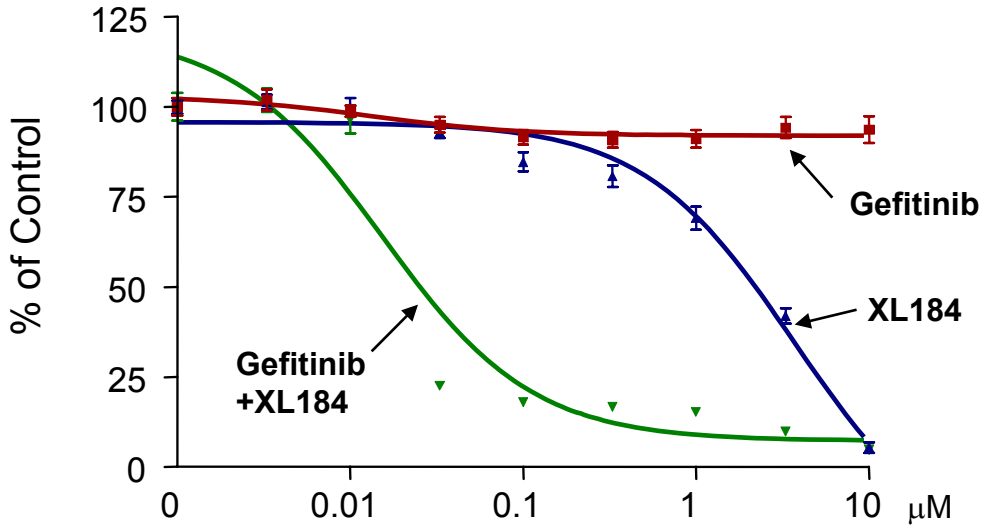
Mechanisms Driving Relapse of NSCLC Tumors Following Response to EGFR Inhibitors



Treat erlotinib – relapsed patients
Treatment up front may prevent or delay emergence of resistant tumors

XL184 Re-sensitizes Gefitinib - Resistant Cells





Data courtesy of Dr P. Janne, Dana Farber Cancer Inst.

Gefitinib sensitive

HCC827
EGFR-Ex19del/amp

Gefitinib Resistant

HCC827 GR6
EGFR-Ex19del/amp
MET amp

Sensitive to the Combination of Gefitinib and XL184

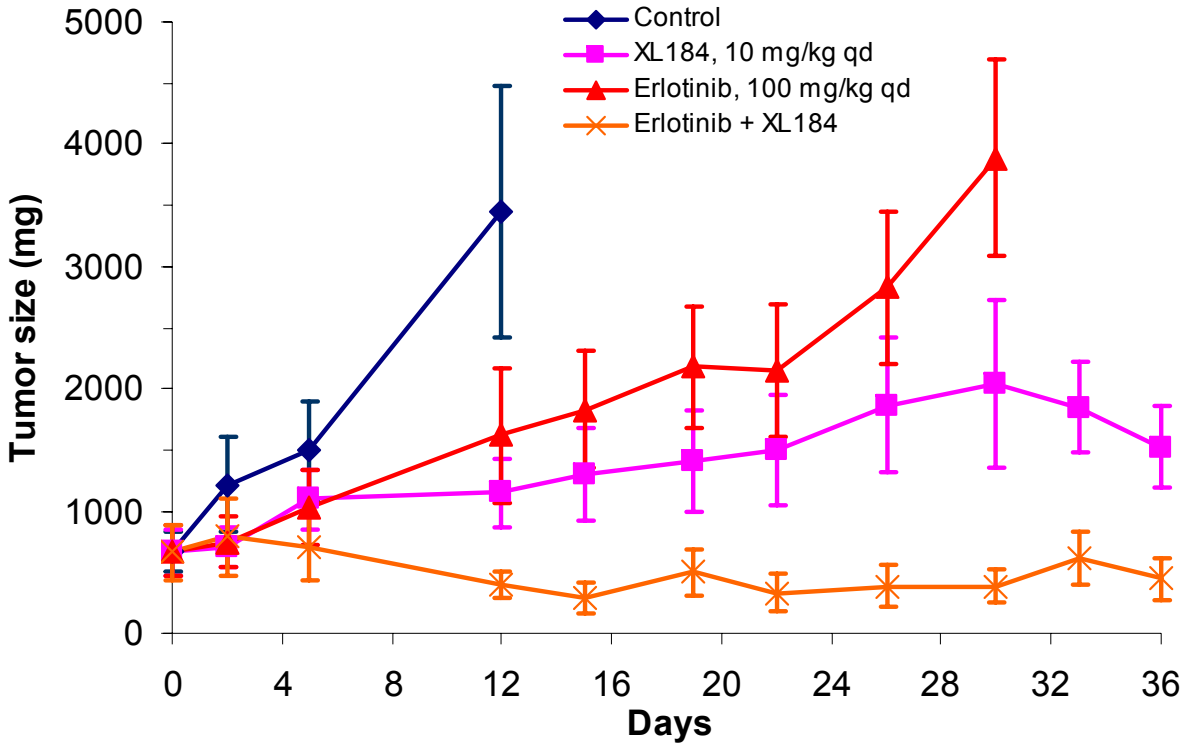
Combination of EGFRI and MET/VEGFR2 Inhibitor – XL184

NSCLC

- Patients who have progressive disease on erlotinib
- Phase 1 run in study to evaluate safety and PK of combining erlotinib and XL184
- Phase 2 Randomized to receive XL184 alone or erlotinib plus XL184
  - 1^{ary} endpoint: RR
  - 2^{ndary} endpoints: safety, PFS, OS, pharmacodynamics
- Correlative studies
  - EGFR mutational analysis
  - MET amplification
  - Pathway molecule status in tumor and surrogate tissue

Reversal of Erlotinib Resistance in NSCLC with XL184

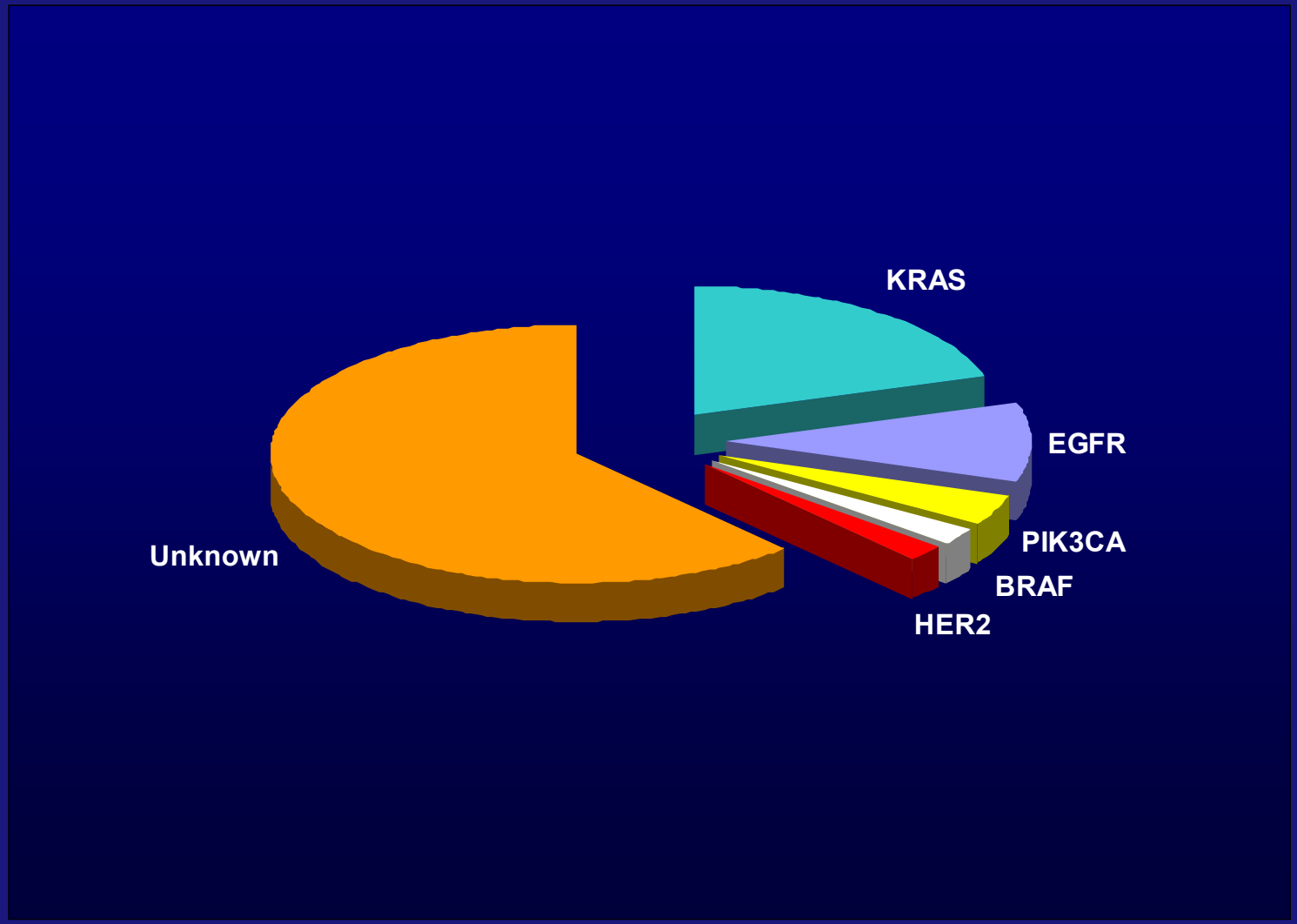
HCC827GR6 xenograft tumors are resistant to gefitinib and erlotinib due to amplification of MET



Data courtesy of J. Engelman, E. Lifshits, and P. Janne, Dana Farber Cancer Inst.

Substantial improvement in anti-tumor activity with the combination of XL184 and erlotinib

"The Lung Cancer Oncogenome Project"



XL647 –Development Plan in NSCLC

Potential to address 1st, 2nd & 3rd line of therapy

- Clinical activity in 1st line NSCLC
- Comparison to erlotinib
- Preclinical activity against resistance conveying EGFR T790M mutation; Clinical evaluation ongoing

Opportunity for combination with targeted agents

- PI3K inhibitors (XL147, XL765)
- Met inhibitors (XL880, XL184)
- Raf inhibitors (sorafenib, XL281)

Anticipate initiation of pivotal studies in H1 2008

3rd /4th line NSCLC

Relapse with documented progressive disease after benefit from erlotinib or gefitinib

- 50% of patients are expected to show T790M mutation

→ Patient population with unmet medical need

- Experimental therapy (Phase 1 with new agents)
- Palliative (best supportive care)

Demonstration of clinical benefit: Improved overall survival, progression free survival and response rate compared to best supportive care

2nd/3rd line NSCLC

Erlotinib as 2nd or 3rd line treatment

- Overall survival 6.7 months (Shepherd et al, 2005)

XL647 may offer advantages

- Target profile: EGFR, HER2, VEGFR2
- Active against T790M
- Tolerability

Possible opportunities for XL647 in a randomized controlled setting

- Improved overall survival
- Improved tolerability

1st line NSCLC

XL647 is active in the first-line setting in clinically selected patients (XL647-201)

- Response rate of about 30% (10/34)
- Clinical benefit of 68% (including PR; and SD and on study for ≥ 3 months)
- Time on study ranging up to 12+ months
- PFS and OS data are maturing

The response rate in unselected patients to first-line doublet chemotherapy is 17-21% (Schiller, 2002)

The response rate to first-line doublet chemotherapy in subjects with EGFR mutations and EGFR wild type is similar (Lee, 2006; Hotta, 2007; TRIBUTE)

Single agent XL647 may offer the opportunity for

- Improved efficacy
- Improved tolerability

XL647 – Broad Development Beyond NSCLC

Target spectrum and preclinical activity support broad development in a variety of indications:

Breast Cancer

- Trastuzumab and lapatinib resistant population, single agent (phase 2)

Head and Neck Cancer

- Single agent, or combined with radiotherapy

Glioblastoma

- Single agent and combination with PI3K inhibitors

Combination with targeted agents

- PI3K inhibitors (XL147, XL765)
- Met inhibitors (XL880, XL184)
- Raf inhibitors (sorafenib, XL281)

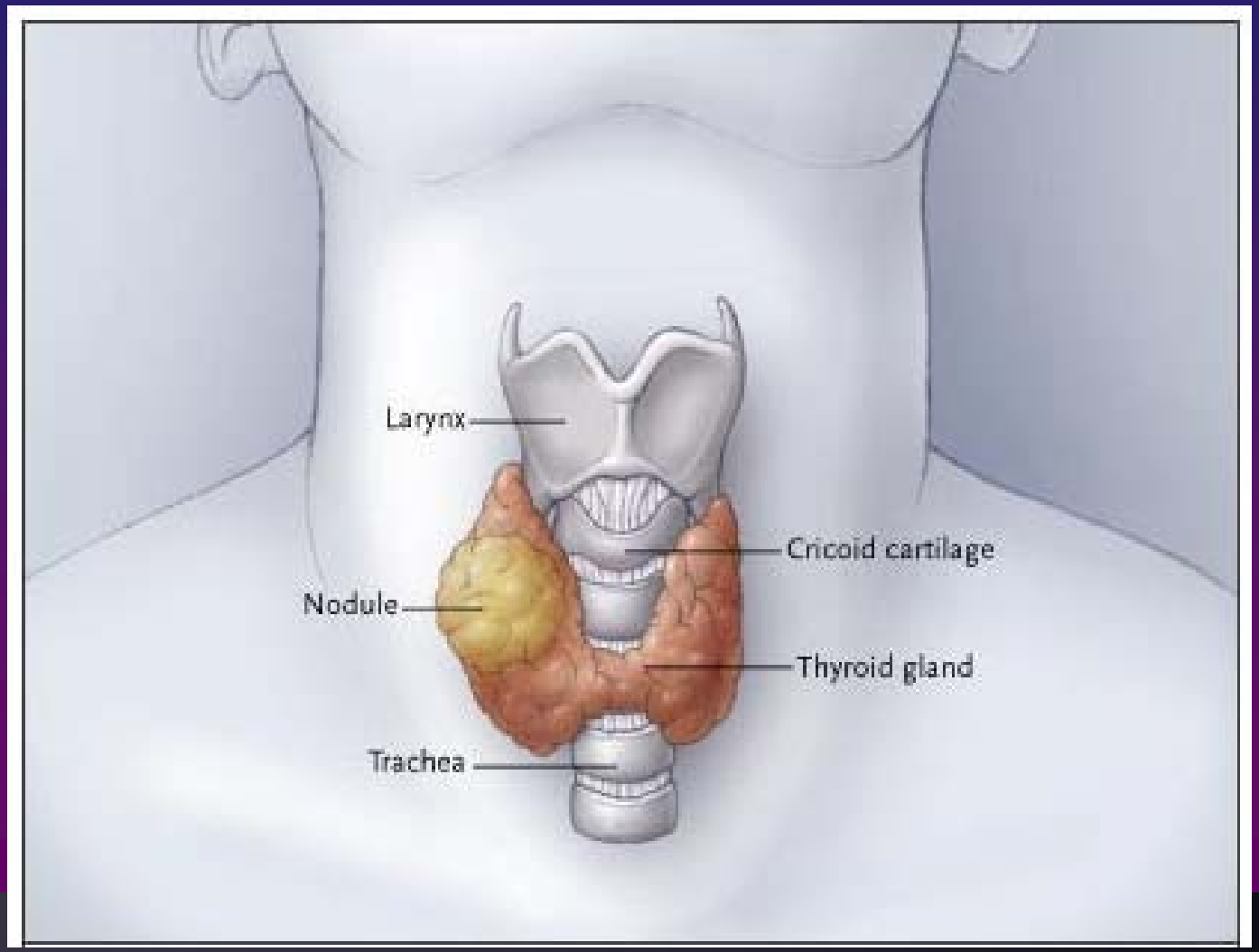
XL184 to Target Multiple Kinases in the Treatment of Metastatic Thyroid Carcinoma

Steven I. Sherman, M.D.

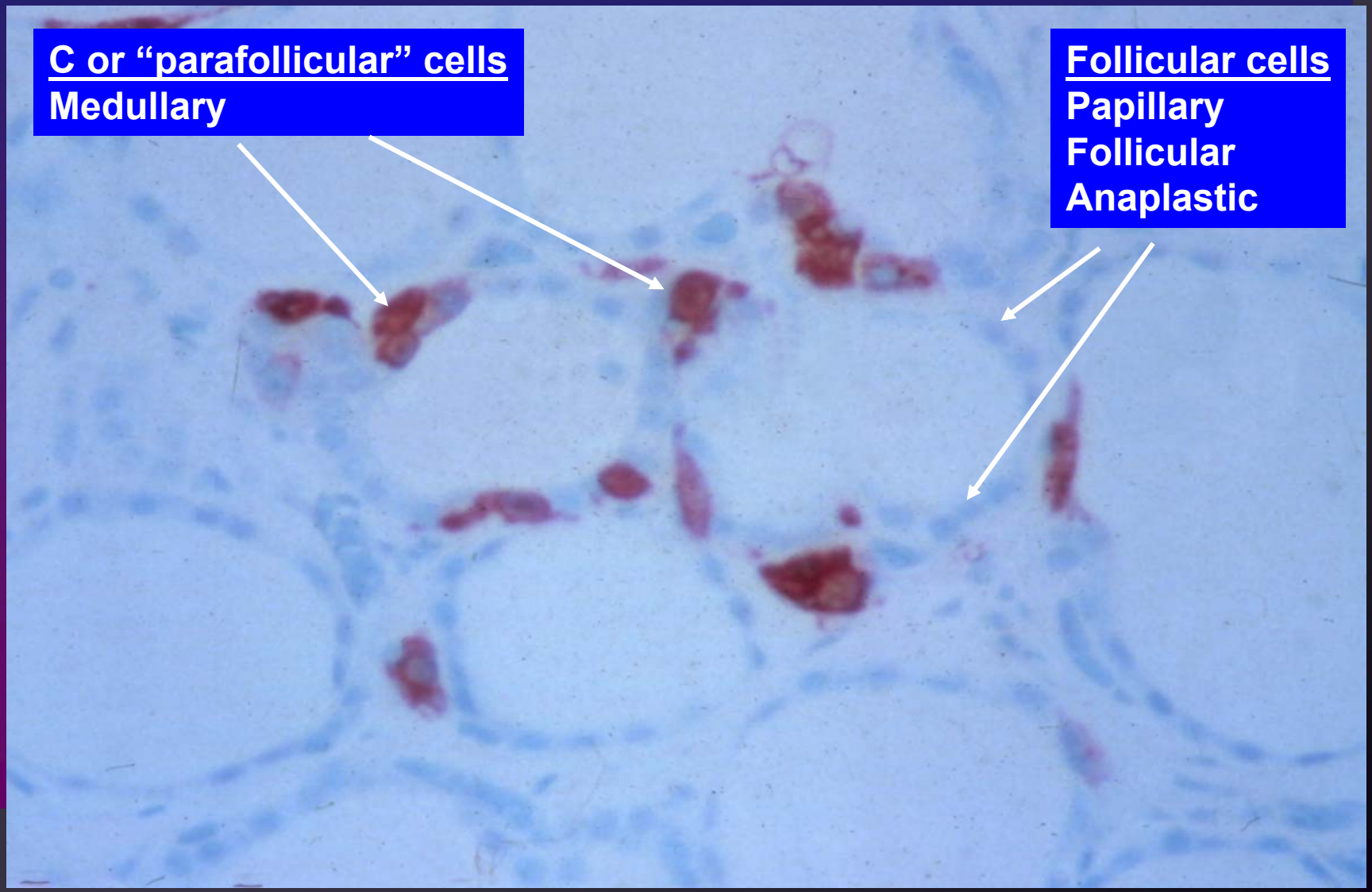
Department of Endocrine Neoplasia and Hormonal Disorders

THE UNIVERSITY OF TEXAS
MD ANDERSON
CANCER CENTER
Making Cancer History™

Thyroid gland and nodule



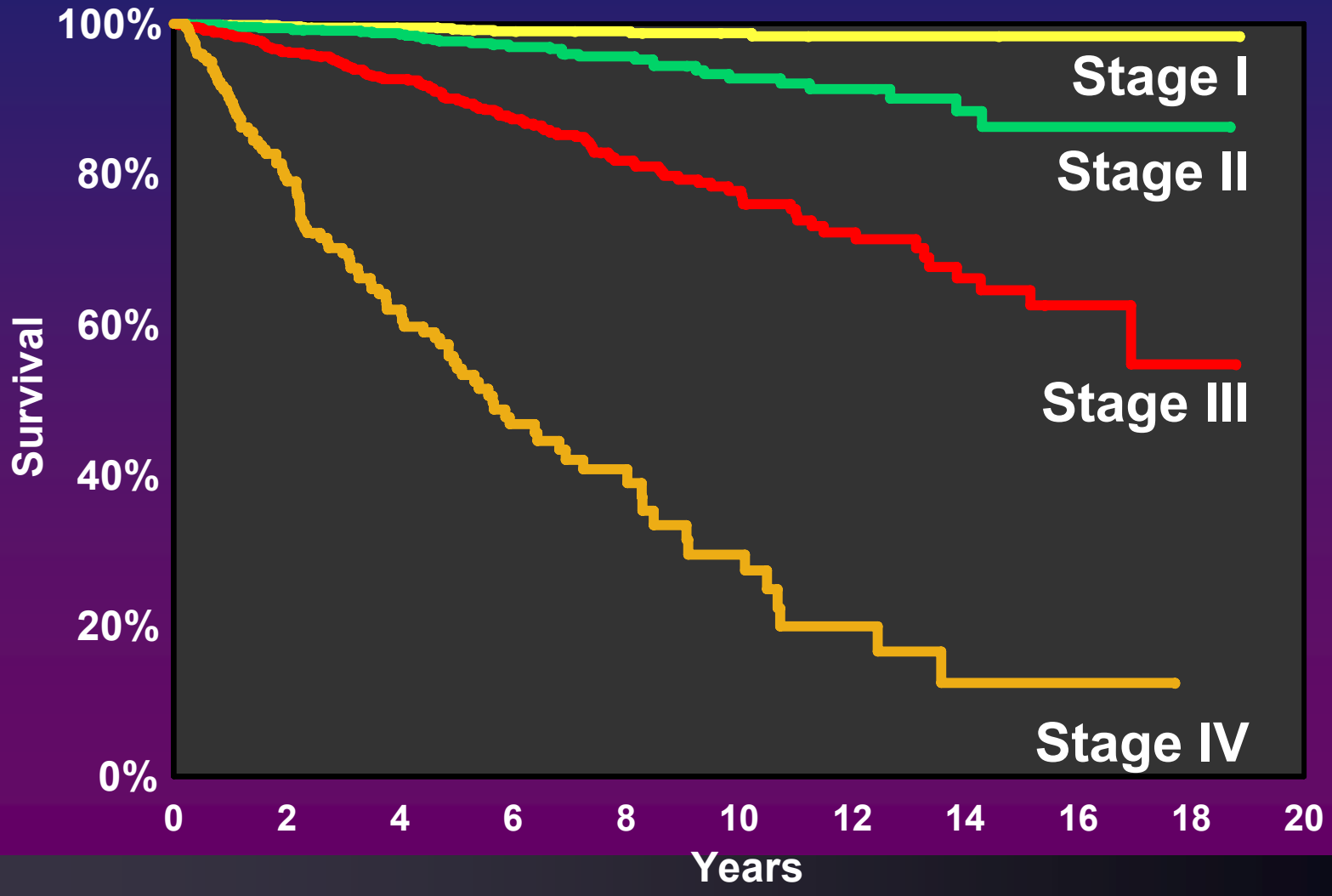
Cells of origin of thyroid carcinomas



Thyroid carcinoma: 2007 estimates

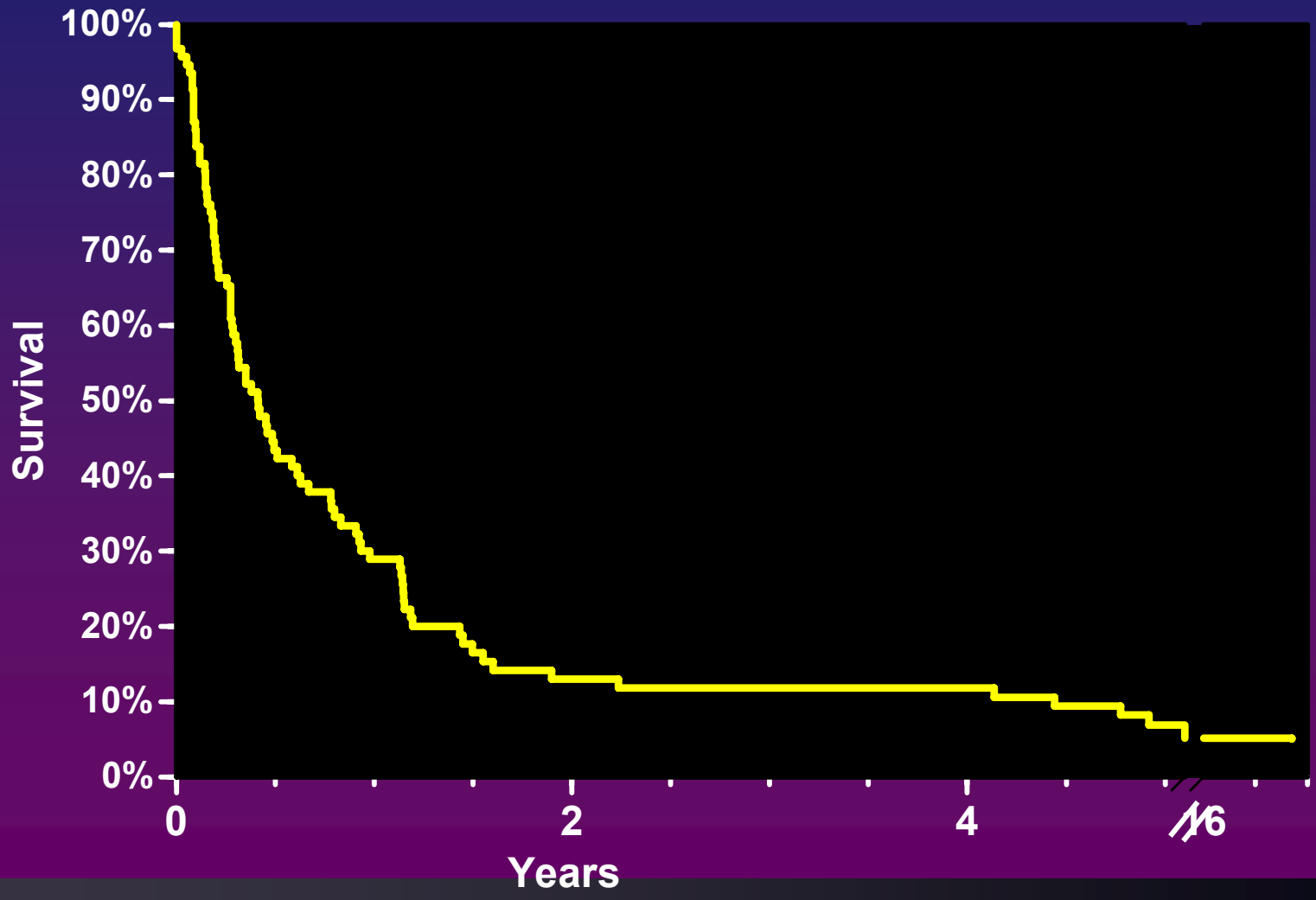
- Incidence 33,550
  - Differentiated (DTC) 95%
  - Medullary (MTC) 4-5%
  - Anaplastic 1%
    - www.cancer.org (as of 2/2007)
- Prevalence (1/2004) 366,000
- 4.8% of all female cancer survivors!
  - www.seer.cancer.gov (as of 11/2007)

Poor outcomes in metastatic differentiated thyroid cancers



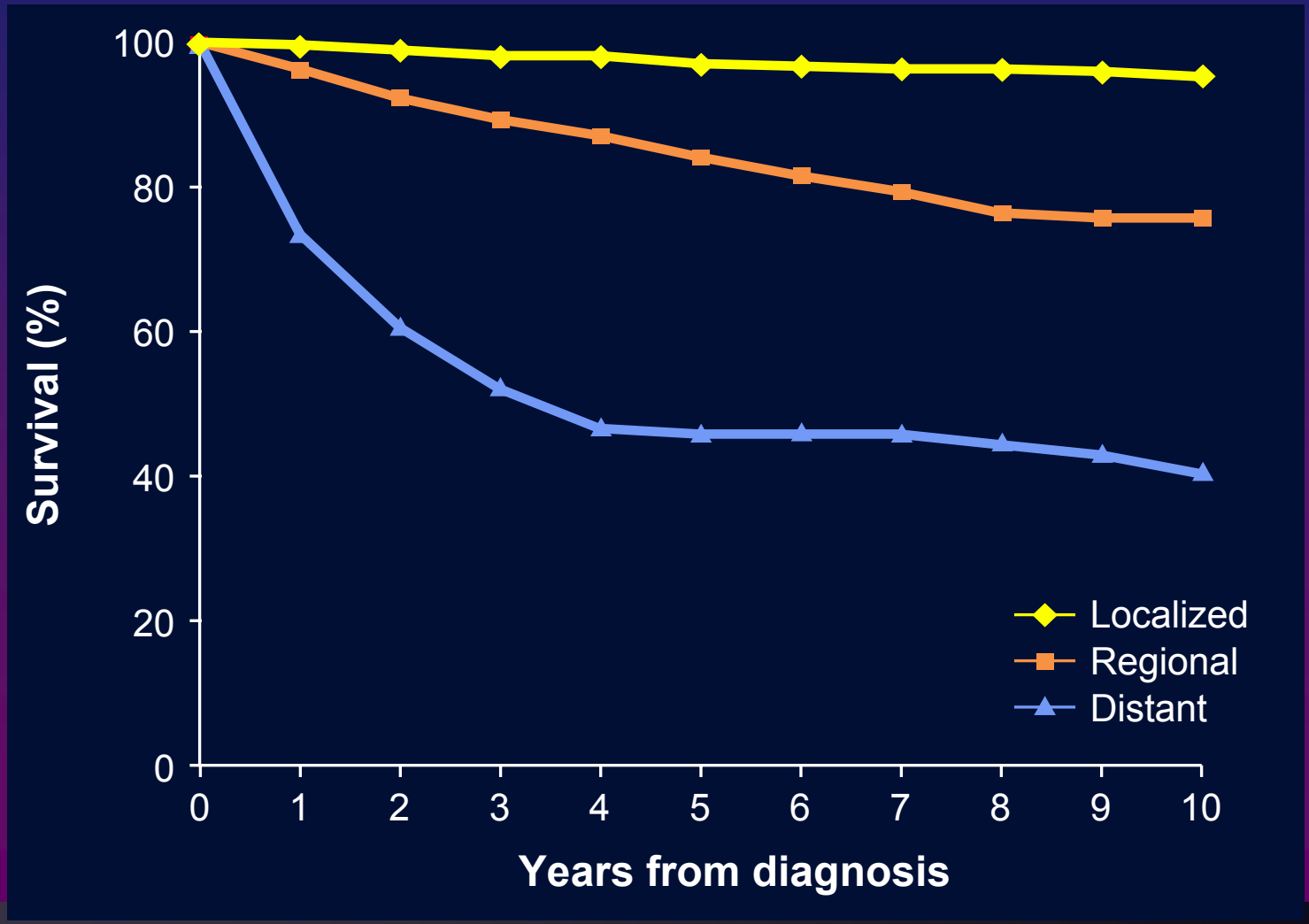
NTCTCSG, 2006 data

Worst outcomes in anaplastic thyroid carcinoma



NTCTCSG, 2004

Poor outcomes in metastatic medullary thyroid carcinoma



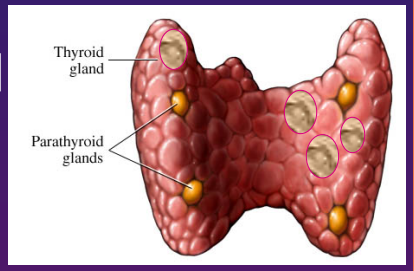
Roman, *et al.*, 2006

Features of familial medullary carcinomas by subtype

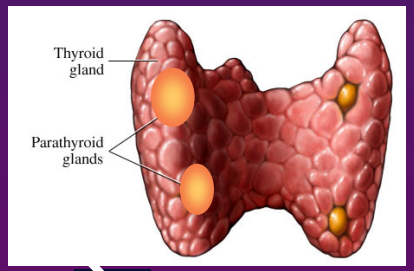
Type 2A

FMTC

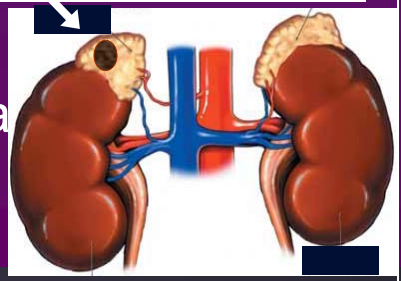
Medullary thyroid carcinoma >90%



Parathyroid hyperplasia 10%-20%

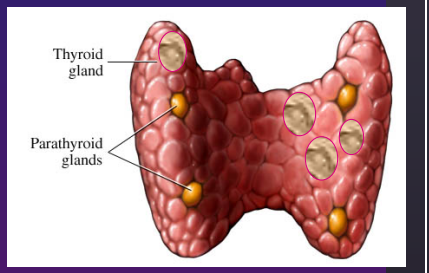


Pheochromocytoma 40%-60%

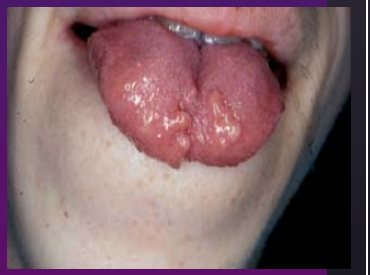


Type 2B

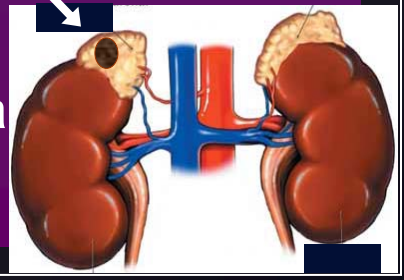
Medullary thyroid carcinoma >98%



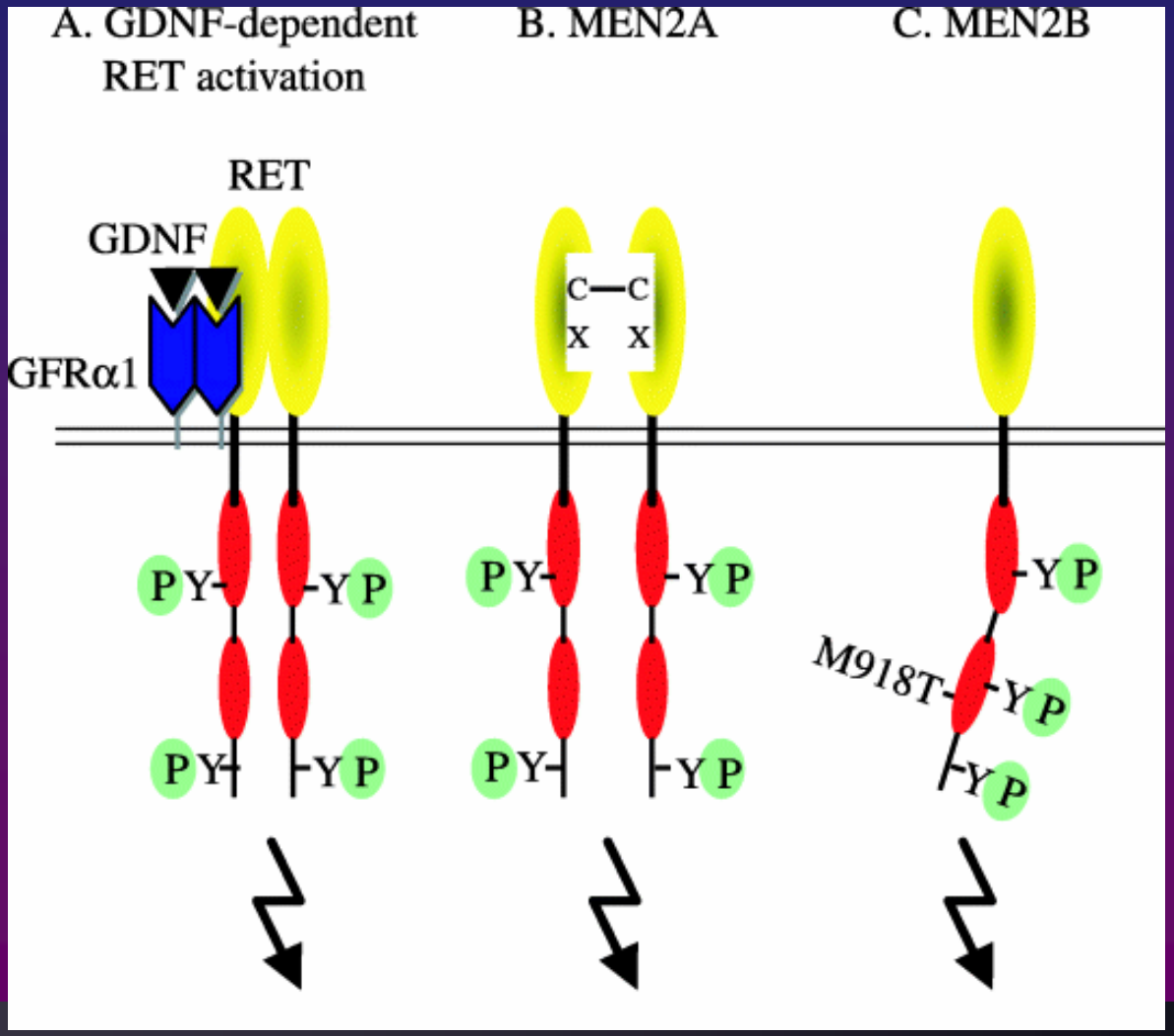
Mucosal neuromas >98%



Pheochromocytoma 40%-60%

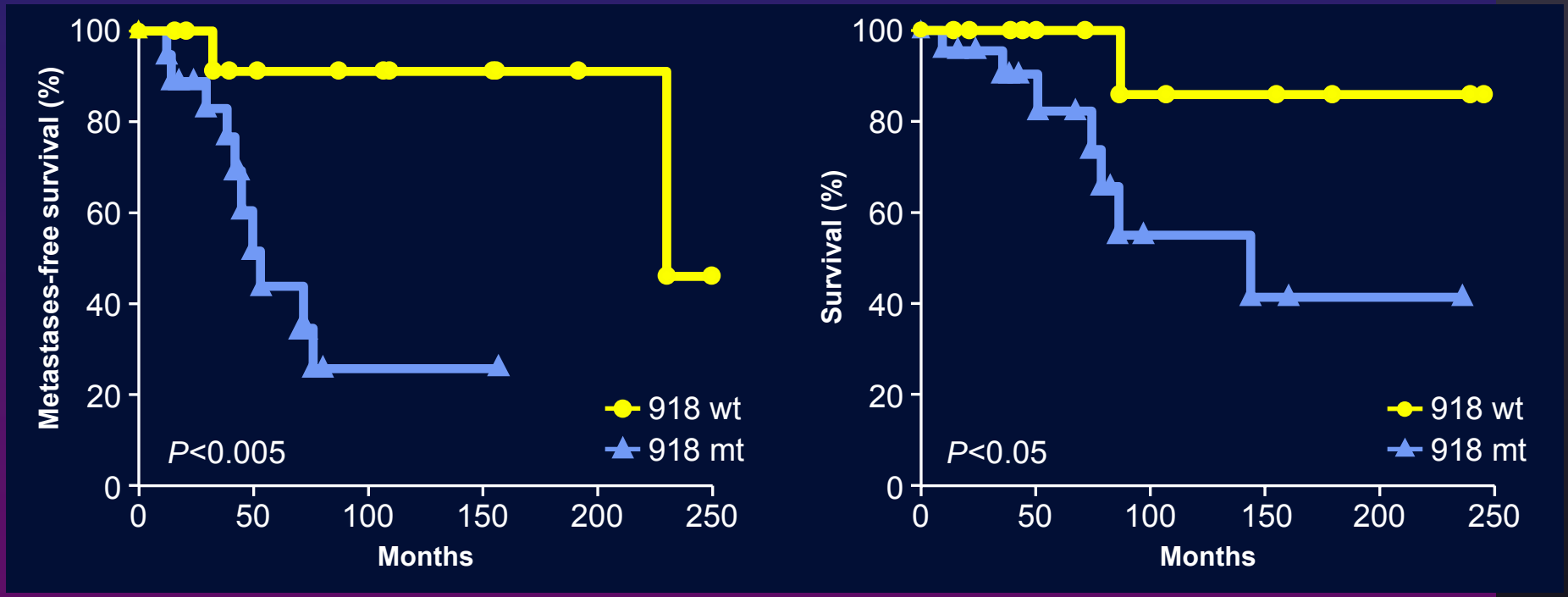


RET mutations in medullary thyroid carcinoma



Kodama, *et al.*, 2005

Role of somatic ret 918 mutation in prognosis



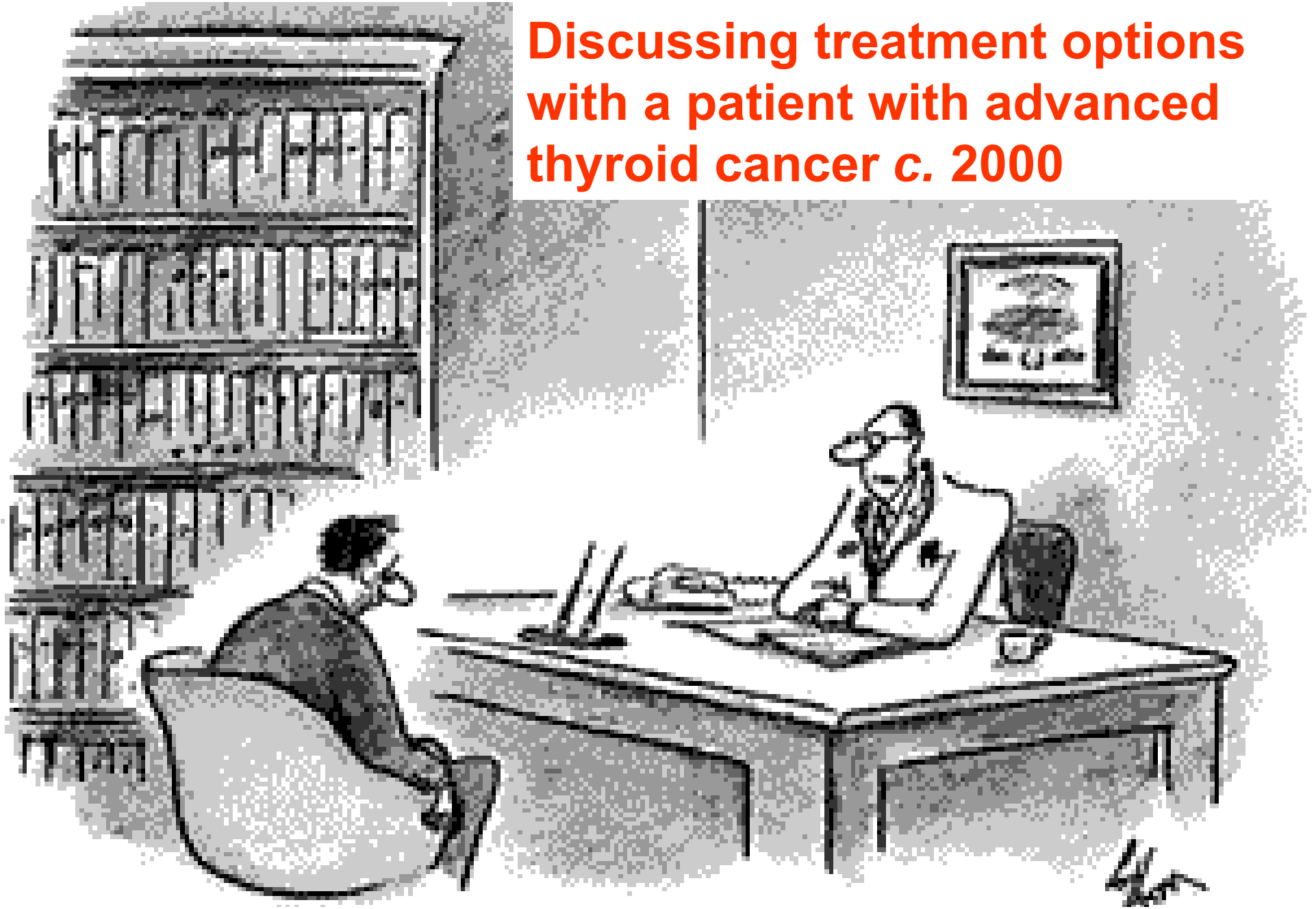
Ret 918 mutation found uniformly in 38%, heterogeneously in 24%

Schilling, *et al.*, 2001

Treating metastatic thyroid cancer

- Traditional
  - Radioiodine (DTC only)
  - Thyroid hormone (DTC only)
  - Cytotoxic chemotherapy
  - Palliation
- New approaches: Targeting molecular abnormalities
  - Etiologic mutations
  - Contributory and secondary mechanisms

Discussing treatment options with a patient with advanced thyroid cancer *c.* 2000



"Unfortunately, there's no cure – there's not even a race for a cure!"

Chemotherapy regimens for medullary carcinoma

Schedule	No. of patients		
	CR	PR	SD + PD
ADM (60 mg/m^2/day)	0	1	3
ADM (60 mg/m^2/day) + CDDP (40 mg/m^2/day)	0	2	4
ADM (15 mg/m^2/week)	1	0	0
BLM (Days 1–3: 30 mg/day + ADM (Day 5: 60 mg/m^2) + CDDP (Day 5: 60 mg/m^2)	0	3	5
ADM (50 mg/m^2) + CDDP (60 mg/m^2) + VDS (3 mg/m^2)	0	1	9
CTX (Day 1: 750 mg/m^2) + VCR (Day 1: 1.4 mg/m^2) + DAC (Days 1–2: 600 mg/m^2/day)	0	2	5
DAC (250 mg/m^2/day) + 5-FU (450 mg/m^2/day)	0	3	2
DAC (200 mg/m^2/day) + 5-FU (400 mg/m^2/day) → STZ (500 mg/m^2/day) + 5-FU (400 mg/m^2/day)	0	3	17
DHAD (12 mg/m^2/day)	0	1	4
TAM (20 mg x 2/day)	0	0	2

ADM: adriamycin; CDDP: cisplatin; BLM: bleomicin; VDS: vindesine; CTX: cyclophosphamide; VCR: vincristine; DAC: dacarbazine; 5-FU: 5-fluorouracil; STZ: streptozocin; DHAD: mitoxantrone; TAM: tamoxifen; CR: complete remission; PR: partial remission; SD + PD: stable disease + progressive disease

Combined CR + PR: 25%

Vitale, *et al.*, 2001

Medullary Carcinoma

RECURRENT OR PERSISTENT DISEASE

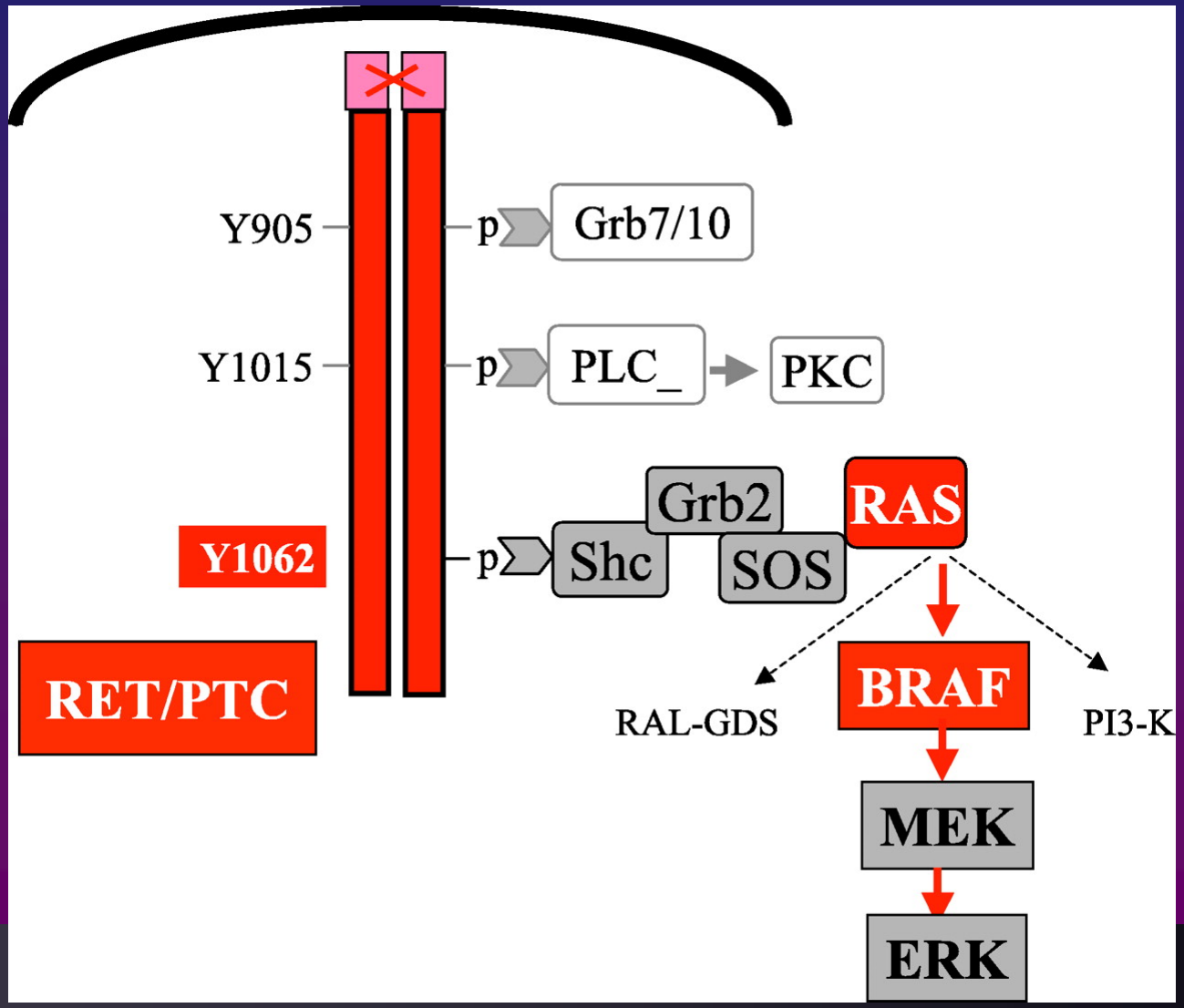
Locoregional →
- Surgical resection ± postoperative RT
- or
- RT, if symptomatic progressive disease or unresectable

Symptomatic, distant metastases →
- Consider palliative resection, ablation (eg, radiofrequency [RFA], embolization or other regional therapy) or other regional treatment

Asymptomatic, distant metastases →
- Observe
- or
- Consider resection, if possible, or ablation (eg, RFA, embolization or other regional therapy) especially if progressive disease

→ **Disseminated symptomatic disease** →
- Clinical trial (preferred)
- or
- RT for focal symptoms
- or
- DTIC-based chemotherapy
- Consider bisphosphonate therapy for bone metastases
- Best Supportive Care

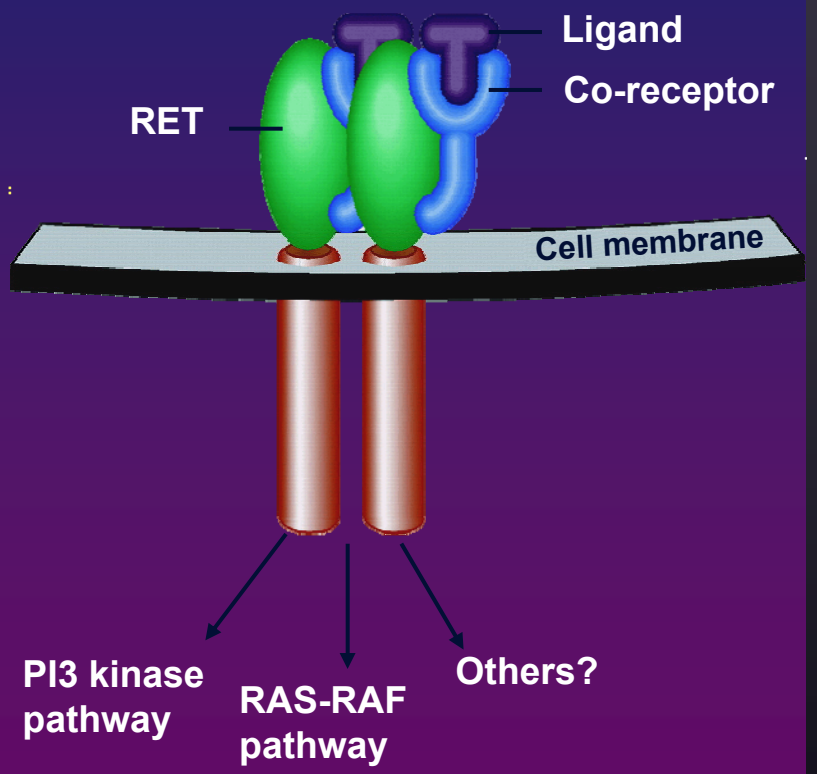
Signaling pathways in DTC: Etiologic signaling activation



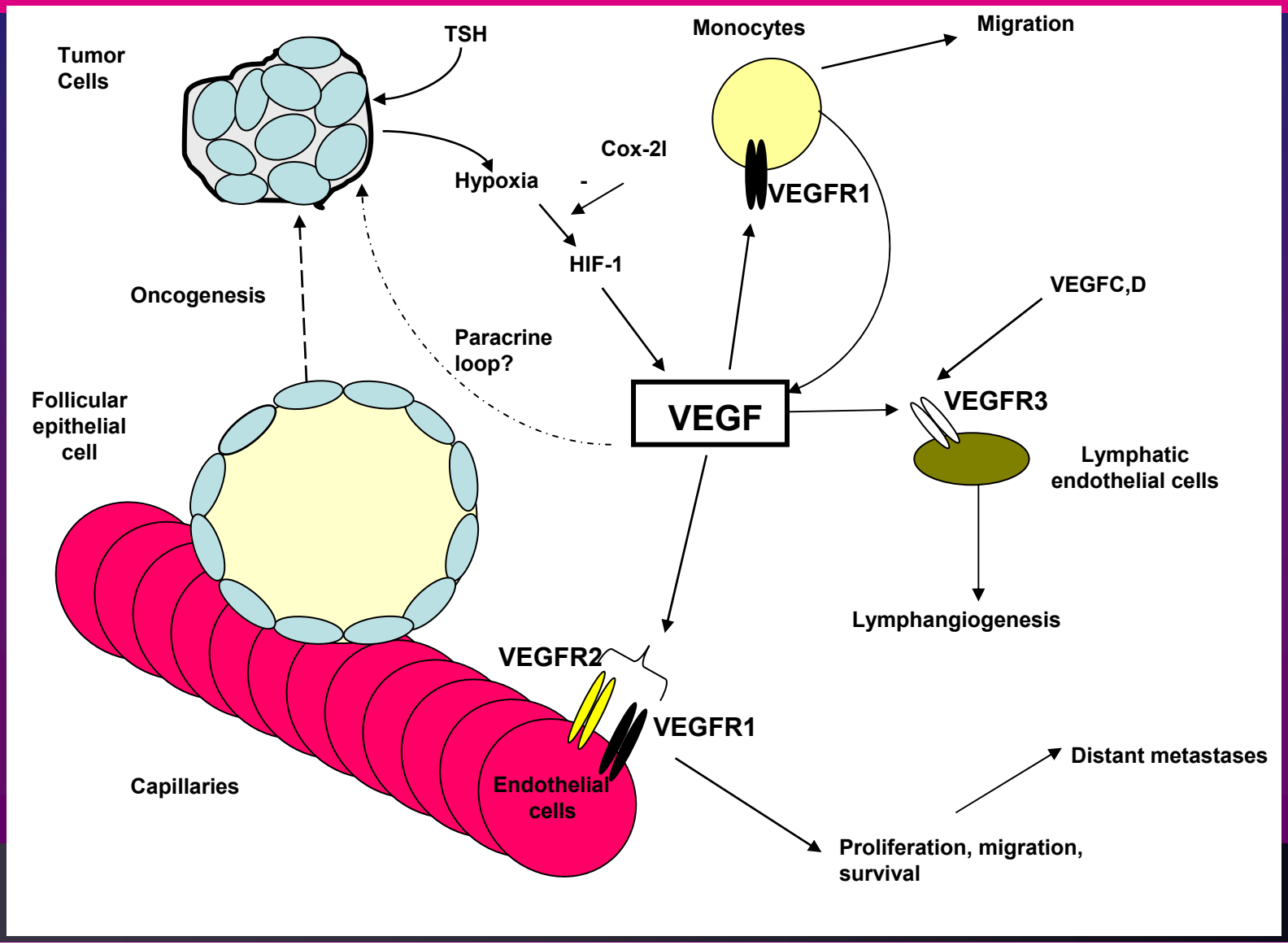
Fagin, 2004

Signaling pathways in MTC: Etiologic signaling activation

- Inhibition of tumor cell growth:
  - Transduction pathways activated by RET
  - Other targets, pathways?
- Inhibition of angiogenesis:
  - VEGF, VEGFR



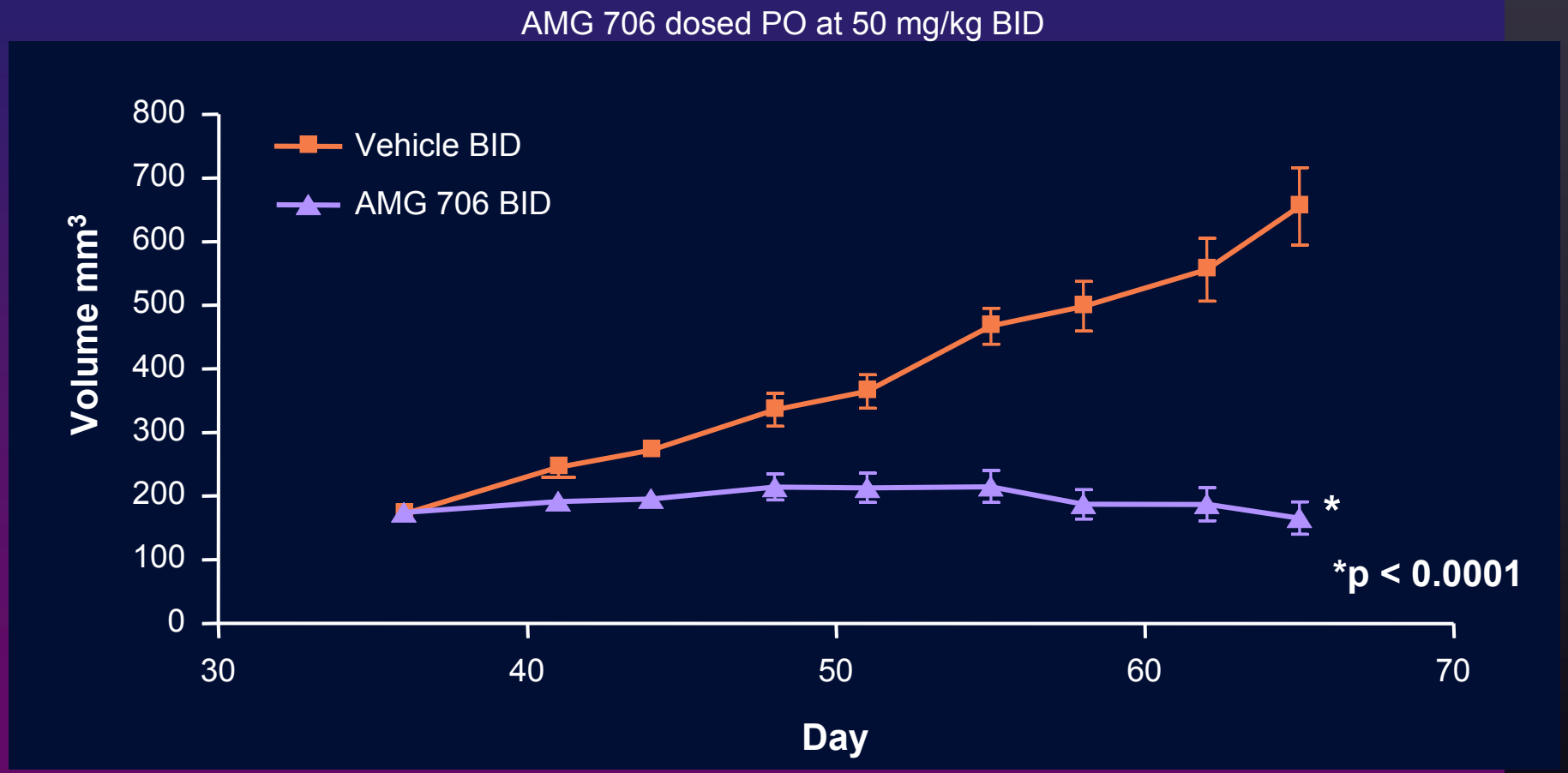
Signaling pathways: Facilitative kinase activation



Kinase inhibitor activities relevant to thyroid carcinomas

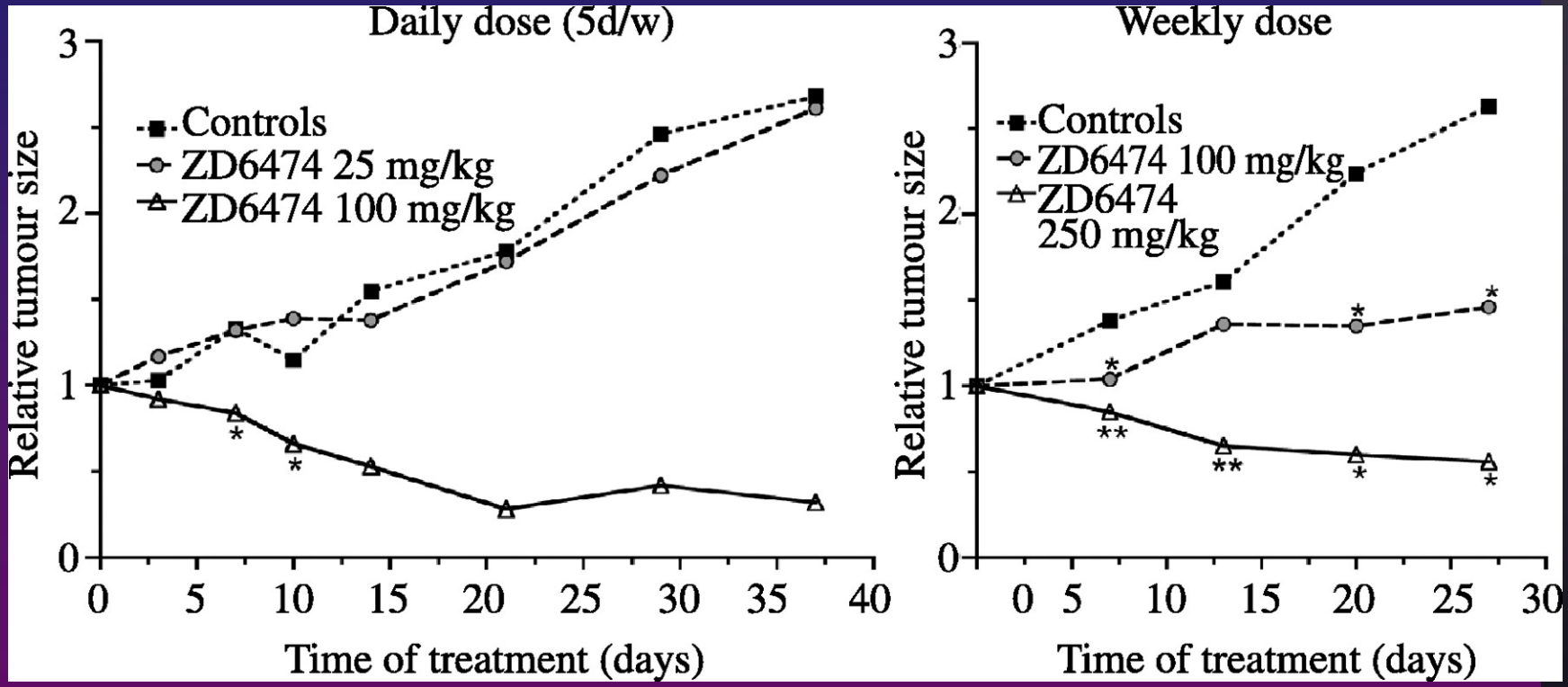
Drug	IC_{50} (nm)						
	VEGFR1	VEGFR2	VEGFR3	RET	RET/PTC3	RAF	C-met
Axitinib	1.2	0.25	0.29	-	-	-	-
Vandetanib	1600	40	110	100	50-100	-	-
Motesanib diphosphate	2	3	6	59	-	-	-
Sunitinib	2	9	17	41	224	-	4000
Sorafinib	-	90	20	49	50	6	$>10^5$

Motesanib inhibits growth of MTC cell line xenografts in mice



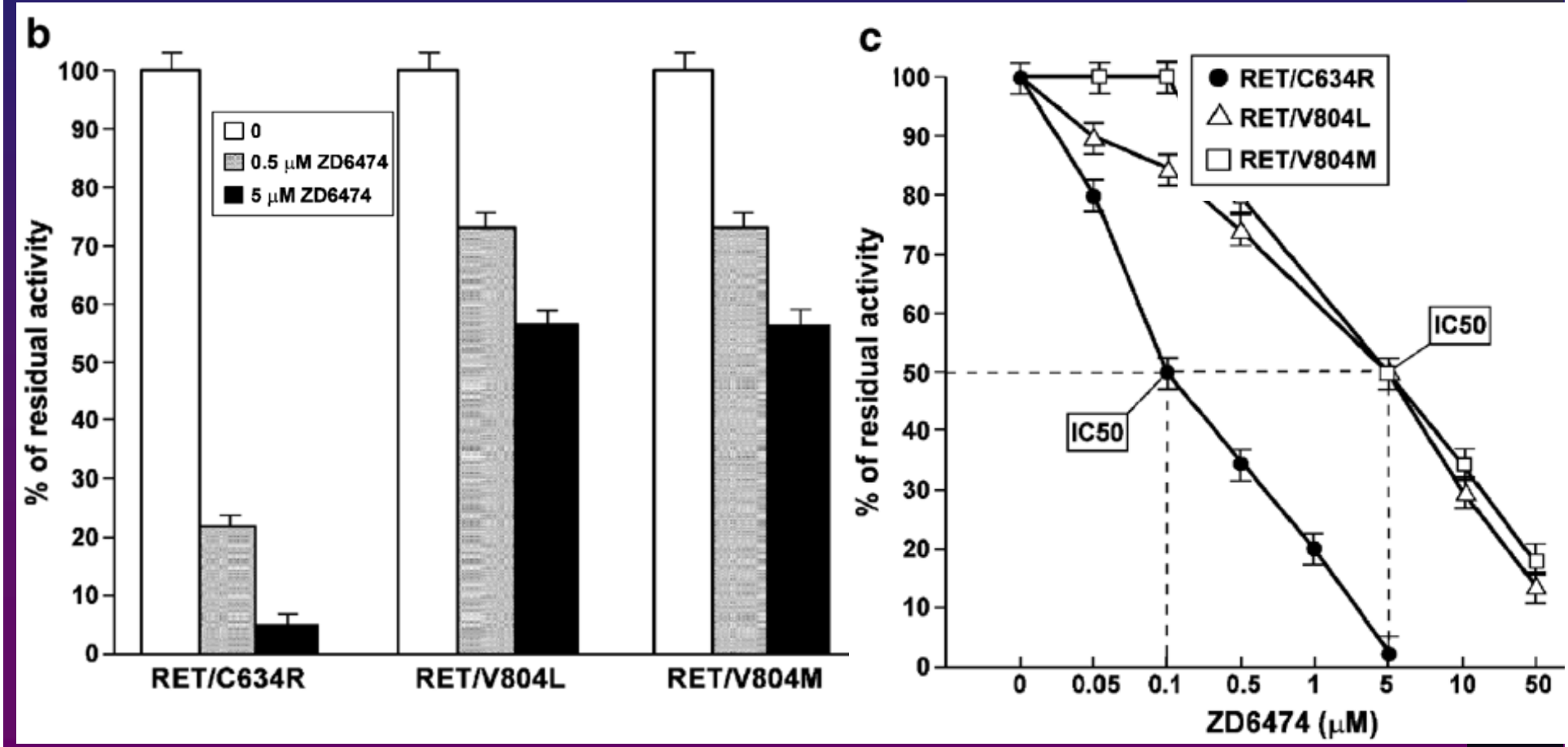
Polverino, *et al.*, ATA 2006

Vandetanib inhibition of growth of human MTC xenograft



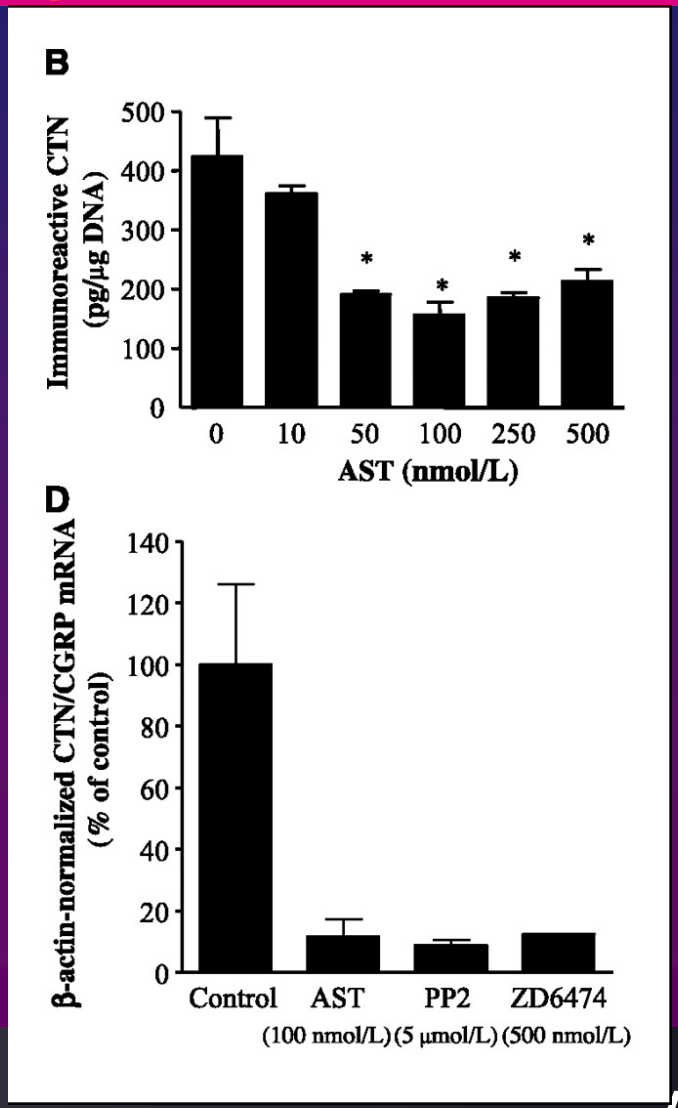
Johanson, *et al.*, 2007

RET V804 mutations confer resistance to vandetanib



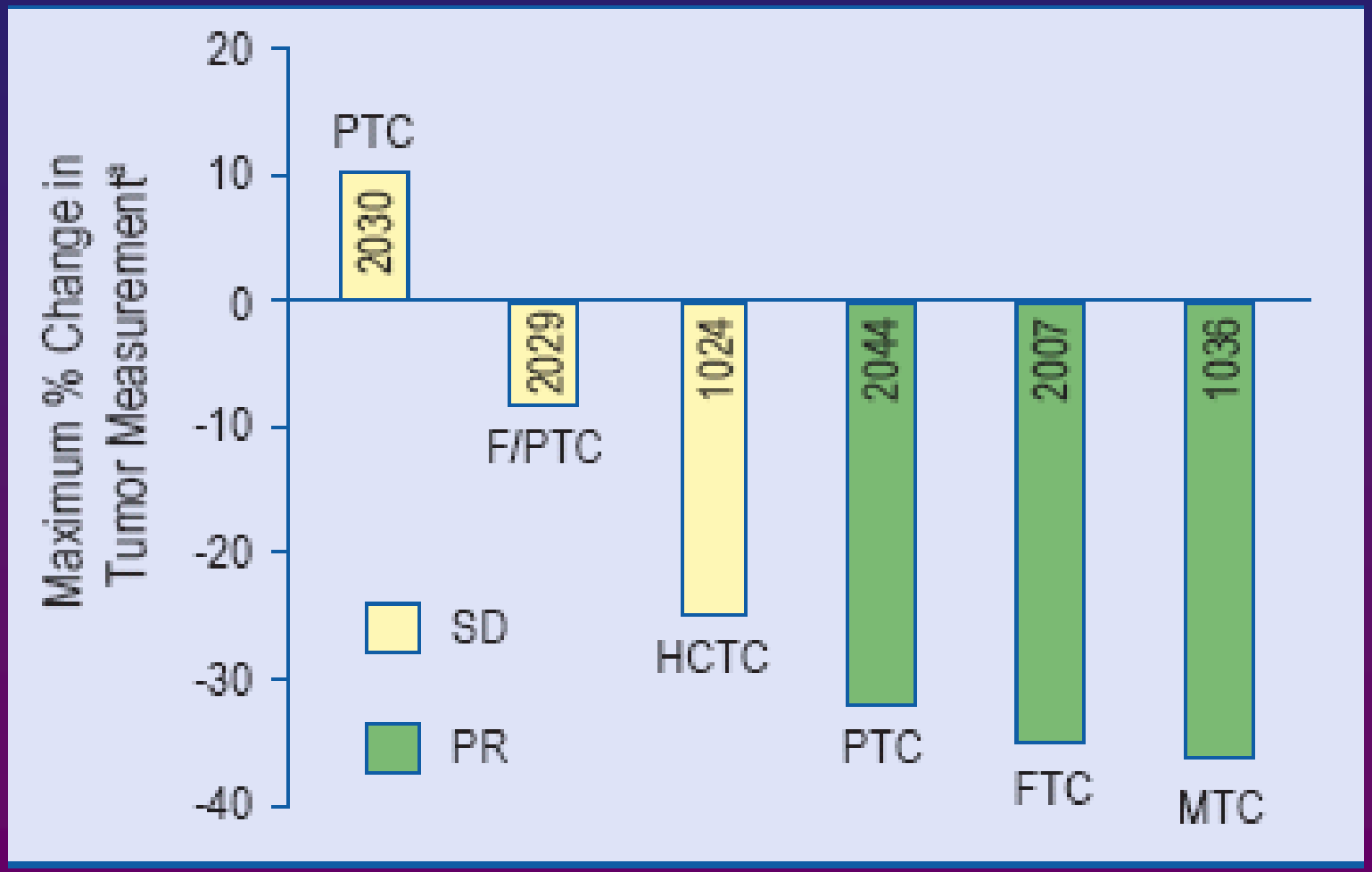
Carlomagno, *et al.*, 2004

RET inhibition may directly affect CT gene transcription



Akeno-Stuart, *et al*., 2007

Motesanib diphosphate Phase I trial



Boughton, *et al.*, ASCO 2006

Phase II trial of motesanib in thyroid carcinoma

Objectives

1. Determine response rates (CR + PR) to motesanib in 2 separate strata:
   - Progressive DTC
   - Progressive or symptomatic MTC

2. Determine duration of response, tumor-related symptoms (MTC), and progression-free survival

Schlumberger, *et al.*, EndoSoc 2007; Sherman, *et al.*, in press

Motesanib: Phase II trial for metastatic MTC

	N (%)
Complete response	0 (0%)
Partial response	2 (2%)
Stable disease	74 (81%)
Clinical benefit (PR + SD ≥24 weeks	45 (49%)
Progressive disease	7 (8%)
Unevaluable/not done	8 (9%)

Schlumberger, *et al.*, EndoSoc 2007

Motesanib: Phase II trial for progressive & metastatic DTC

	N (%)
Complete response	0 (0%)
Partial response	13 (14%)
Stable disease	62 (67%)
Clinical benefit (PR + SD ≥24 weeks)	46 (49%)
Progressive disease	7 (8%)
Unevaluable / not done	11 (12%)

Sherman, *et al.*, in press

Phase II trial of vandetanib in medullary thyroid carcinoma

Objective

Determine response rates (CR + PR) to vandetanib in patients with metastatic inherited MTC

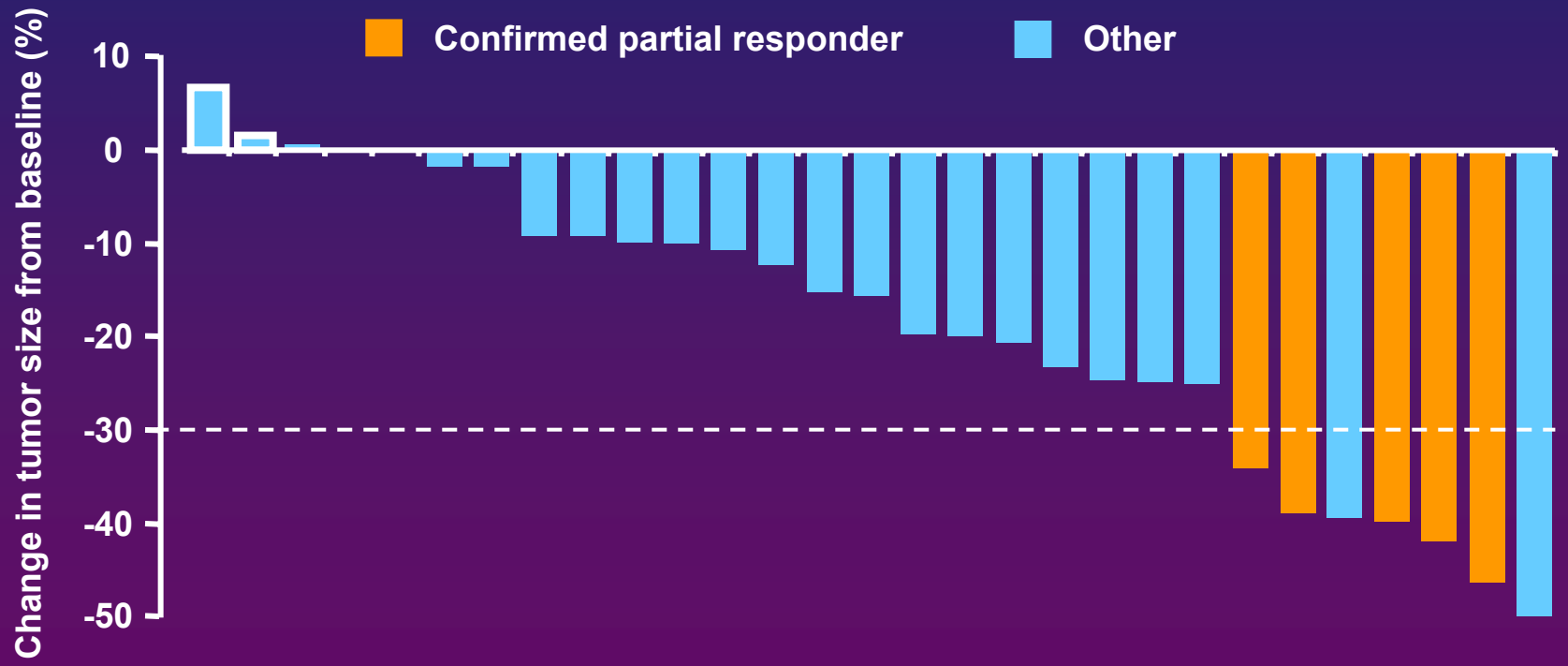
Wells, *et al*., ASCO 2007

Vandetanib: Phase II trial for metastatic inherited MTC

	N (%)
Complete response	0 (0%)
Partial response	5 (17%)
Stable disease	22 (73%)
Clinical benefit (PR + SD ≥24 weeks	15 (50%)
Progressive disease	2 (7%)
Unevaluable/not done	1 (3%)

Wells, *et al.*, ASCO 2007

Best objective tumor response per RECIST

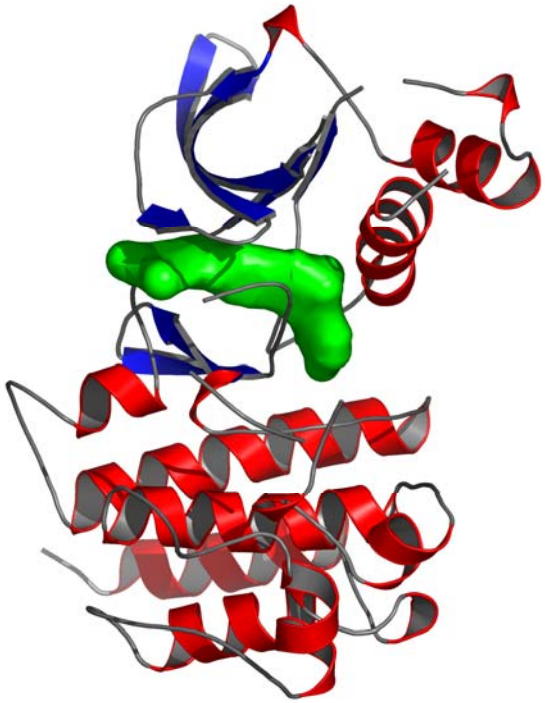


Wells, *et al.*, ASCO 2007

XL184

XL184 is a potent inhibitor of MET, VEGFR2 and RET

- Low nM activity vs. KIT, Flt3
- Lacks the PDGFRβ activity of XL880



XL184 has excellent pharmacokinetic properties

- High exposure and excellent bioavailability

Extremely potent & efficacious in xenograft models

- Potent anti-tumor activity in broad array of tumor xenografts
- Single dose efficacy
- Excellent tolerability in preclinical models

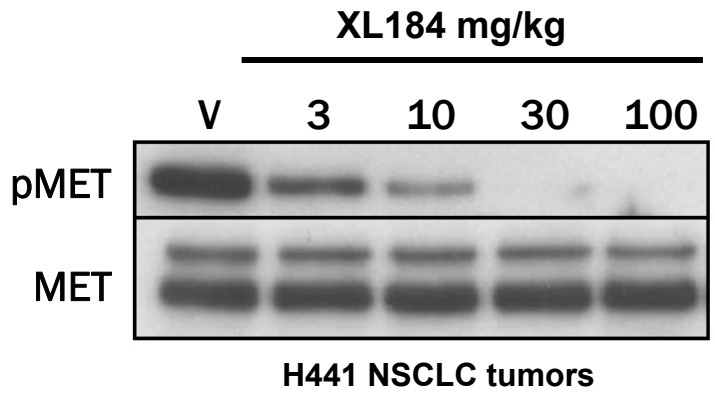
Kinase inhibitor activities relevant to thyroid carcinomas

Drug	IC_{50} (nm)						
	VEGFR1	VEGFR2	VEGFR3	RET	RET/PTC3	RAF	C-met
Axitinib	1.2	0.25	0.29	-	-	-	-
Vandetanib	1600	40	110	100	50-100	-	-
Motesanib diphosphate	2	3	6	59	-	-	-
Sunitinib	2	9	17	41	224	-	4000
Sorafinib	-	90	20	49	50	6	$>10^5$
XL184	-	0.035	-	4	-	-	1.8

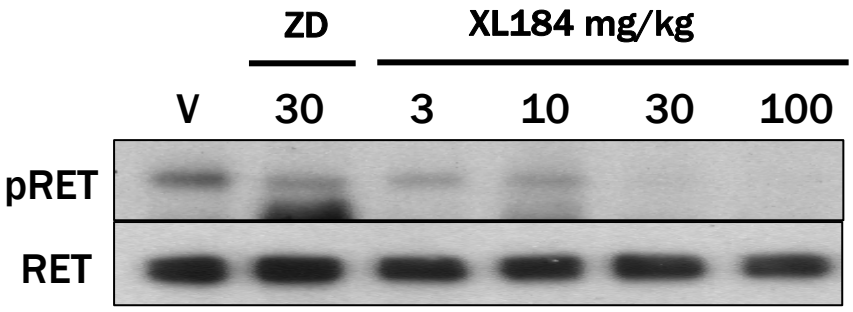
XL184 PD Activity

XL184 is a balanced inhibitor of MET, VEGFR2 and RET in preclinical tumor models

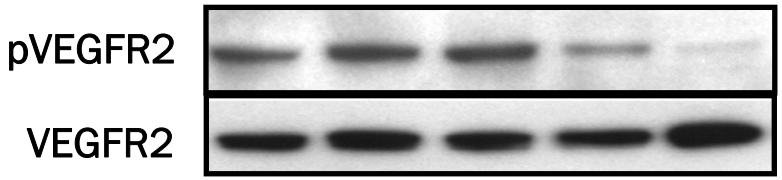
- Biochemical potencies: MET 1.8 nM, VEGFR2 0.035 nM, RET 4 nM, PDFGRβ 234 nM



H441 NSCLC tumors



TT thyroid tumors

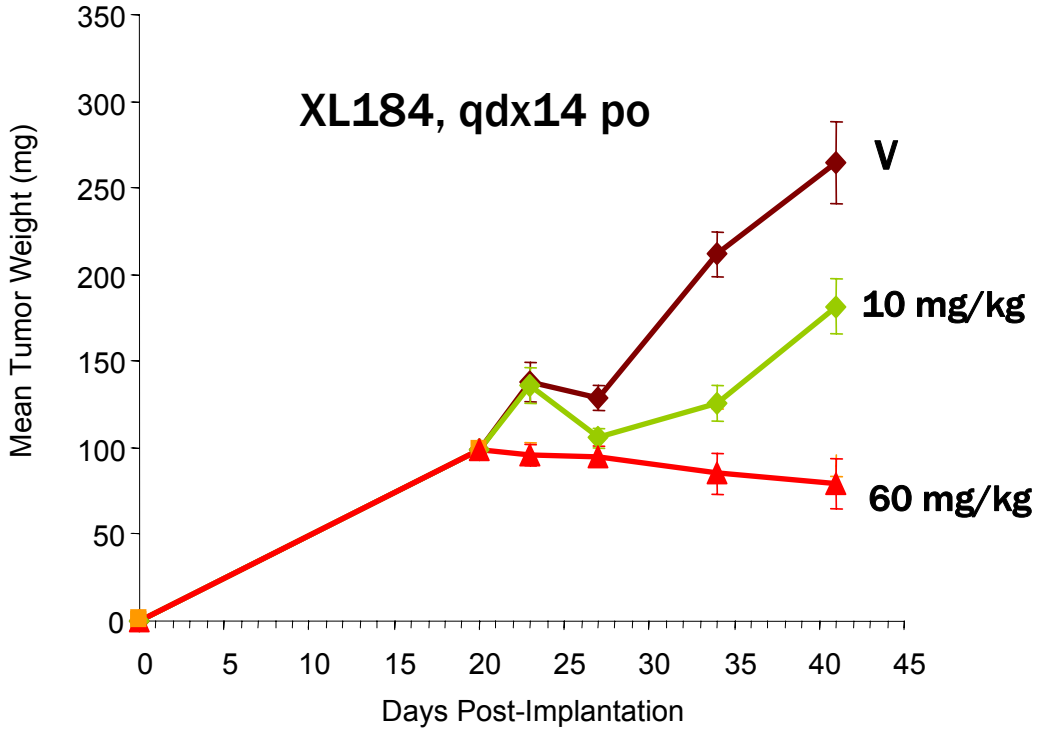


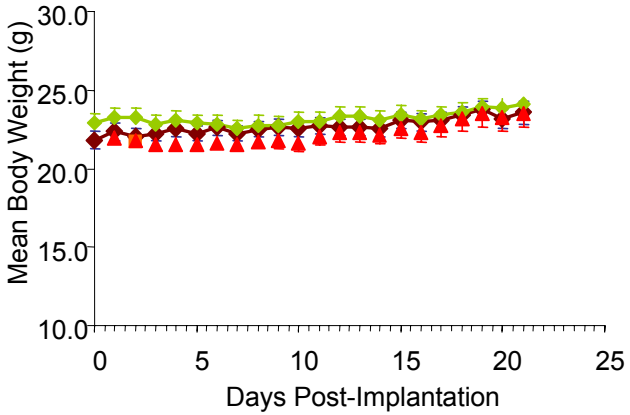
Mouse lung

XL184 Inhibits the Growth of TT Xenograft Tumors

Daily oral dosing of XL184 causes substantial inhibition of TT tumor growth

- TT cells are derived from a thyroid tumor and carry an activating mutation in RET
- Dose response parallels dose response for inhibition of pRET in TT tumors
- Well tolerated – no body weight loss





EXELIXIS™

A Phase 1 Dose-Escalation Study of the Safety and Pharmacokinetics (PK) of XL184, a VEGFR and MET Kinase Inhibitor, Administered Orally to Patients (Pts) With Advanced Malignancies

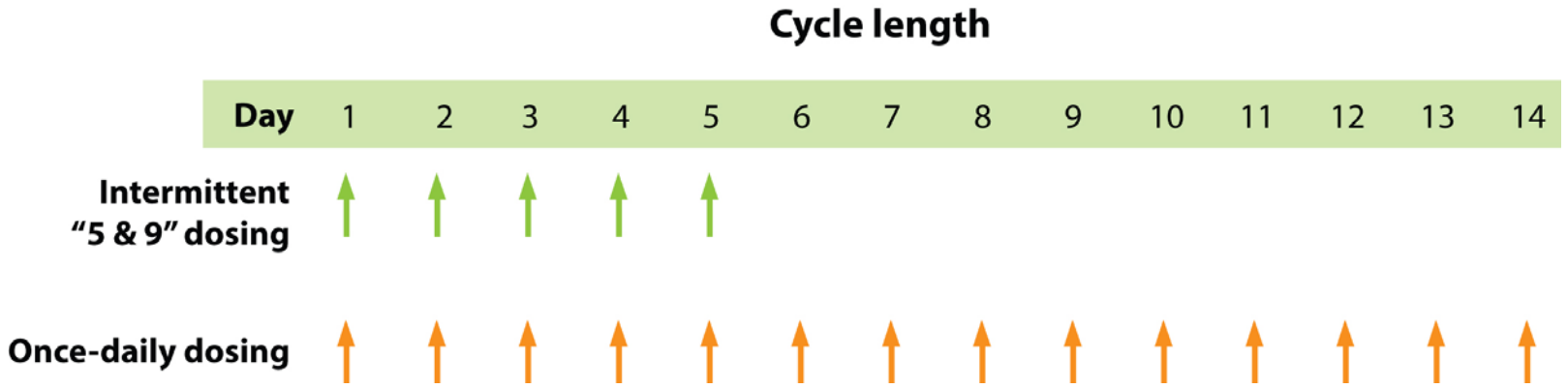
A152

Phase 1 Study
Evaluating Intermittent and Daily Dosing

EXELIXIS™

Objectives:

Evaluate safety, pharmacokinetics, pharmacodynamics and preliminary anti-tumor activity of XL184 in patients with advanced malignancies



Baseline Characteristics

Characteristic	No. of patients (N = 44[a])
Median age (range), years	54 (29-89)
Gender (male/female)[a]	33/11
Race[b]	
Caucasian	35
Black	4
Asian	2
Other	3
Tumor diagnosis/primary site[a]	
Medullary thyroid	10
Colorectal	5
Carcinoid, melanoma, sarcoma	3 each
Papillary renal cell, parotid, pancreatic, gastric, mesothelioma	2 each
Breast, liver, cutaneous T-cell lymphoma, adeno/GE junction, head and neck, appendiceal, laryngeal, neuroendocrine, SCC/tongue, adenocystic	1 each
ECOG performance status[a]	
0	18
1	23
2	2
Unknown	1
Prior chemotherapy[b]	
Median number of regimens (range)	3 (0-10)

[a]Includes data from 38 patients reported in the September 4, 2007, data transfer and 6 additional patients not included in the September 4, 2007, data transfer.
[b]Includes data from 37 patients reported in the September 4, 2007, data transfer.

Summary of Treatment Status

Status	Patients, n
On treatment	15
Off treatment	29
Reason for withdrawal[a]	
Progressive disease	24
Investigator decision	2
Other	3

XL184 dose level achieved	Patients, n	Months on study
0.08 mg/kg	4	1-9
0.16 mg/kg	4	1-20+ (2 active)
0.32 mg/kg	4	1-4
0.64 mg/kg	3	1-4
1.28 mg/kg	3	2-13+ (1 active)
2.56 mg/kg	3	3-7
5.12 mg/kg	5	1-9+ (3 active)
7.68 mg/kg	3	1-4
11.52 mg/kg	3	1-5
175 mg daily	3	1-3+ (2 active)
265 mg daily	9	0-1+ (8 active)

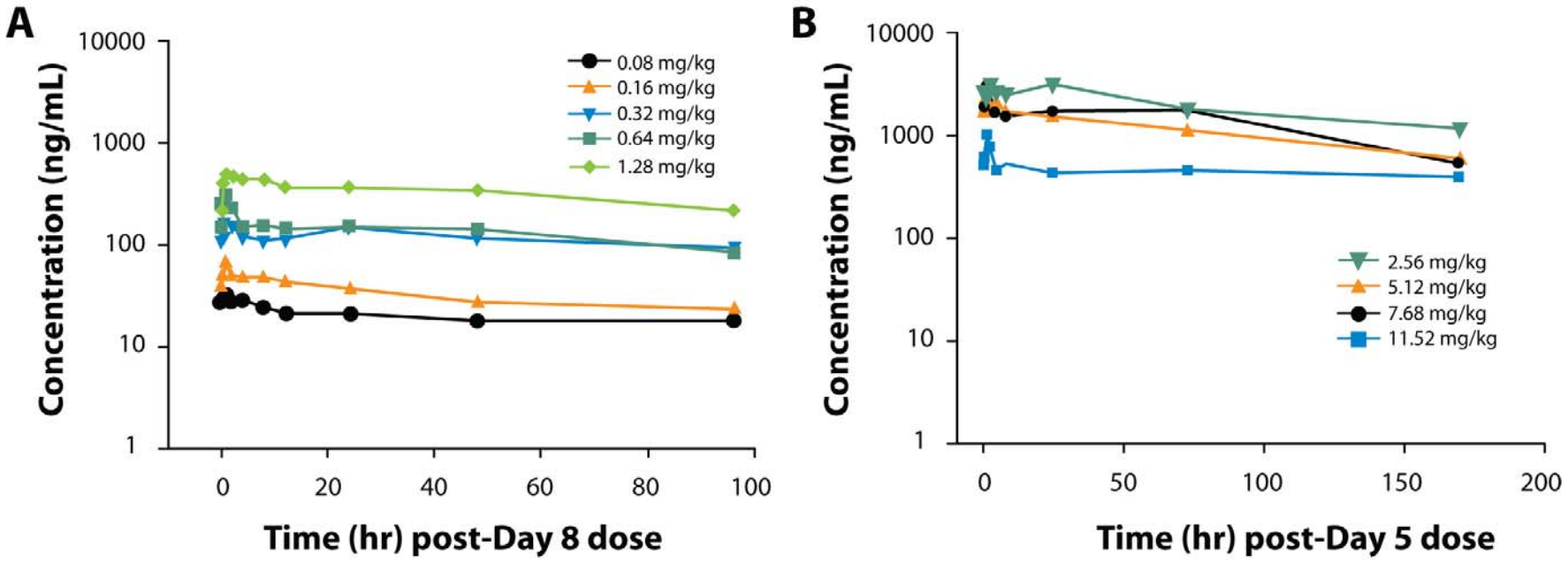
[a]Data available for 38 patients reported in the September 4, 2007, data transfer. This includes patients with date of withdrawal. Data are preliminary because study is ongoing.

Most Frequently Reported (≥10% of Patients) Adverse Events Considered Possibly or Probably Related to Study Treatment

Adverse event[a]	n (%) of patients (N = 38)	
	Grade[b] 1 or 2	Grade 3 or 4
Diarrhea	9 (24)	1 (3)
Fatigue	6 (16)	1 (3)
Increased AST	4 (11)	1 (3)
Mucosal inflammation	5 (13)	0
Nausea	5 (13)	0
Increased ALT	3 (8)	1 (3)
Vomiting	4 (11)	0

ALT, alanine aminotransferase; AST, aspartate aminotransferase
[a]Adverse event data include 1 patient who elected to withdraw from the study and later re-enrolled under a unique patient study number.
[b]According to the Common Terminology Criteria for Adverse Events Version 3.0.

Mean XL184 Plasma Concentration-Time Profiles



Plasma samples were analyzed for XL184 using a validated liquid chromatography method with tandem mass spectrometric detection method.
The lower limit of quantitation for XL184 in plasma was 0.5 ng/mL based on a plasma volume of 50 µL.
PK data include 1 patient who elected to withdraw from the study and later re-enrolled under a unique patient study number.

Data represent mean values following 5 consecutive daily doses of XL184.
(A) C1-C5; (B) C6-C9.

Response to Treatment

Response	Patients, n	Primary cancer diagnosis	Duration of stable disease (≥3 months)
Confirmed partial response	2	Medullary thyroid	
Unconfirmed partial response	2	Medullary thyroid, Neuroendocrine	
Stable disease	1	Cutaneous T-cell lymphoma	20+
	2	Carcinoid	8, 19+
	1	Parotid	4
	3	Colorectal	3, 7, 7
	1	Appendiceal	4
	2	Medullary thyroid	9+, 13+
	2	Sarcoma	6, 6
	1	Papillary renal cell	9
	1	Melanoma	5+
	1	Mesothelioma	3+
Progressive disease	21	Various	
Unevaluable/premature	8	Various	

Response of MTC patients to XL184

Patients Have Undergone at Least 1 Evaluation

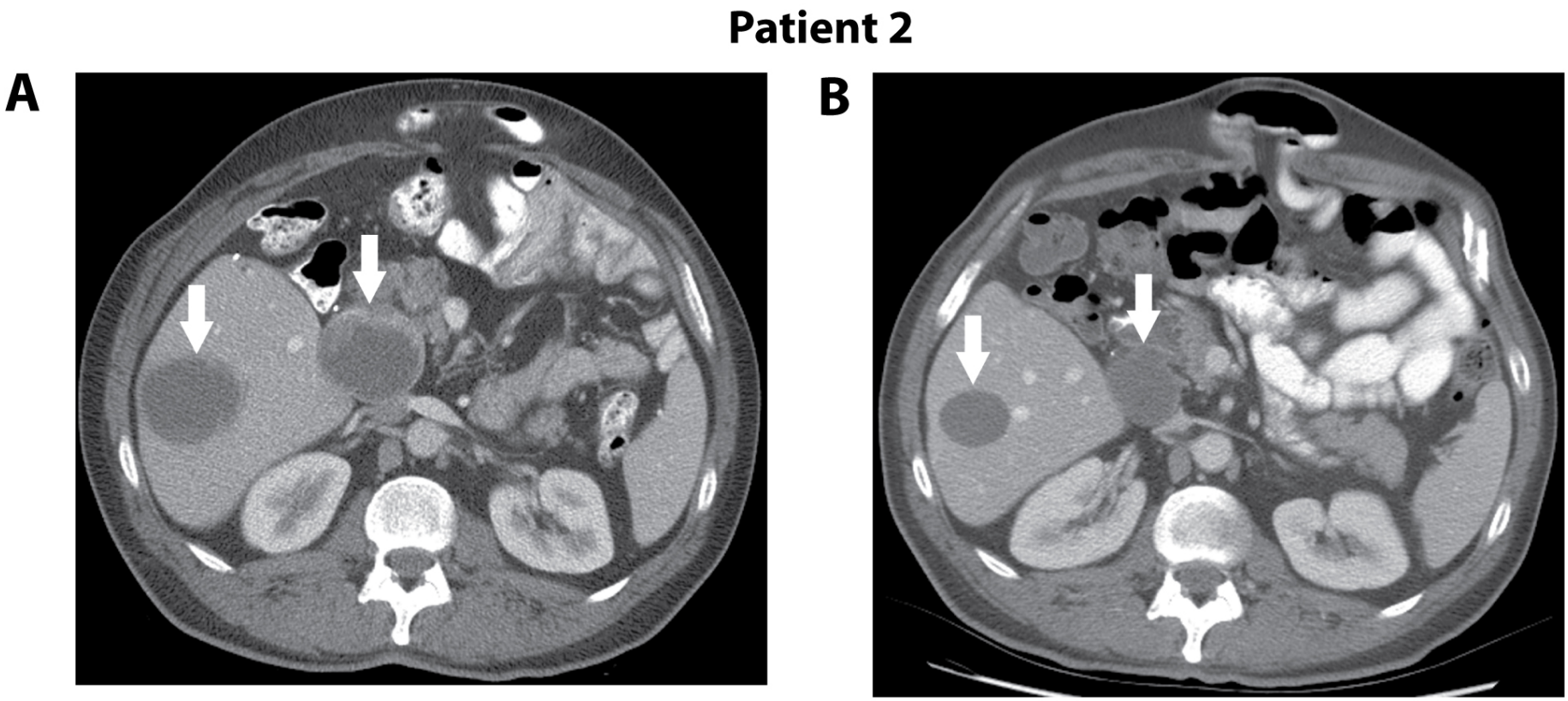
	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7
Months on study	13+	10+	9+	8+	2+	1+	1+
Tumor measurement[a]	−16	−39	NM	−48	−11	−16	−30
Best response	SD	PRc	SD	PRc	TEE	TEE	PRu
Calcitonin	−52	−95[b]	−91	−89[b]	−52	−66	−73
CEA	−42	−82	−51	−69	−5	−10	1

NM, nonmeasurable disease; SD, stable disease (≥3 months); PRc, confirmed partial response; PRu, unconfirmed partial response; TEE, too early to evaluate

[a]Measurable lesions were assessed using modified RECIST and percent change from baseline was rounded to the nearest integer.

[b]See Figure 5 for scans of these patients.

Scans of a Patient With MTC



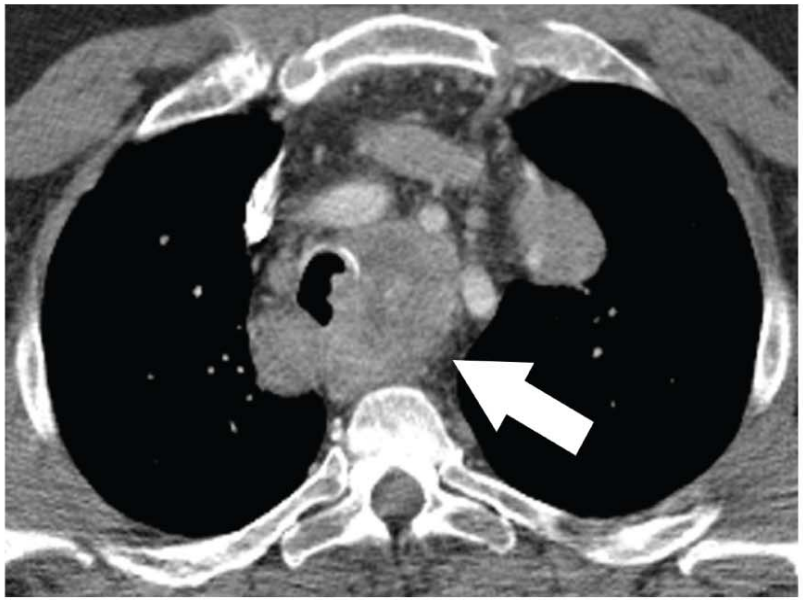
54-year-old male. Liver and periportal nodal metastatic disease at baseline (A) and after 7.5 months (B) of treatment with XL184.

Scans of a Patient With MTC

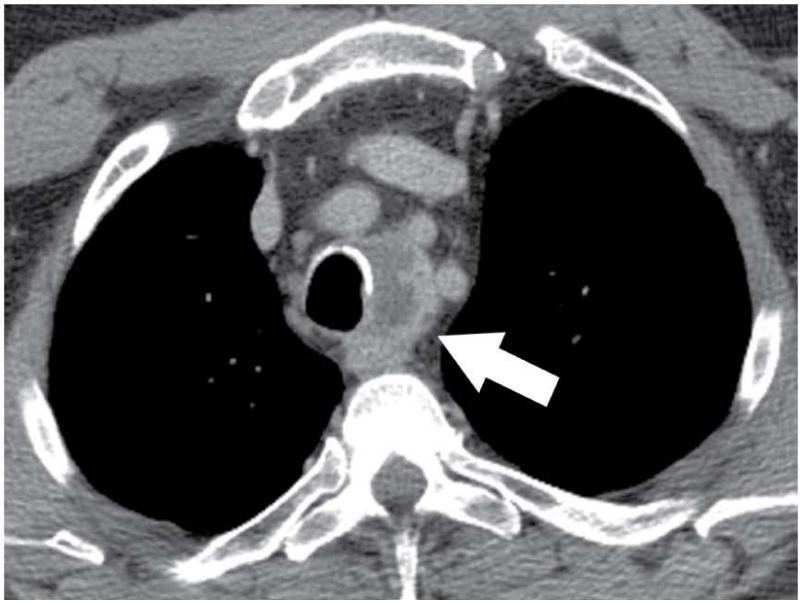
EXELIXIS™

Patient 4

C



D



55-year-old male. Metastatic paratracheal nodal disease involving the trachea at baseline (C) and after 4 months (D) of treatment with XL184.

Conclusions XL184-001

XL184 is generally well tolerated at doses tested to date.

Preliminary analysis of pharmacodynamic biomarkers in plasma shows changes in VEGF- A, sVEGFR2, and PIGF consistent with effects observed with other anti-angiogenic agents.

Antitumor activity has been observed at doses not associated with significant toxicity and in patients with various cancers to date:

- 4 PRs (2 PRc, 2 PRu) and 15 patients with stable disease (SD) >3 months (3+ to 20+ months

All 7 patients with MTC had signs of anti-tumor activity:

- 6 pts had tumor shrinkage
- 3 pts had PRs (2 confirmed)
- 2 pts had SD >9 months

XL184 Development Plans - MTC

Anti-tumor activity in Phase 1 – medullary thyroid cancer

Expanded cohort of MTC patients (n = 20) in Phase 1 at the maximum tolerated dose

Current Standard of Care

- Surgery is current treatment for localized disease – no standard treatment for metastatic disease

Possible opportunity for demonstrating clinical benefit of XL184 in comparison to best supportive care

- Progression free survival

XL184 Phase 2 Program

Glioblastoma Multiforme

- Encouraging activity of targeting angiogenesis in GBM
- MET and KIT are implicated in GBM
- Phase 2 study to start at MTD

NSCLC

- MET amplification plays an important role in resistance development
- Phase 1 /2 study initiated
- Combination of EGFR inhibitor and XL184 vs. XL184 alone

Variety of other tumors potentially responsive to XL184

- Including breast, colon, and liver cancer, and other tumor types

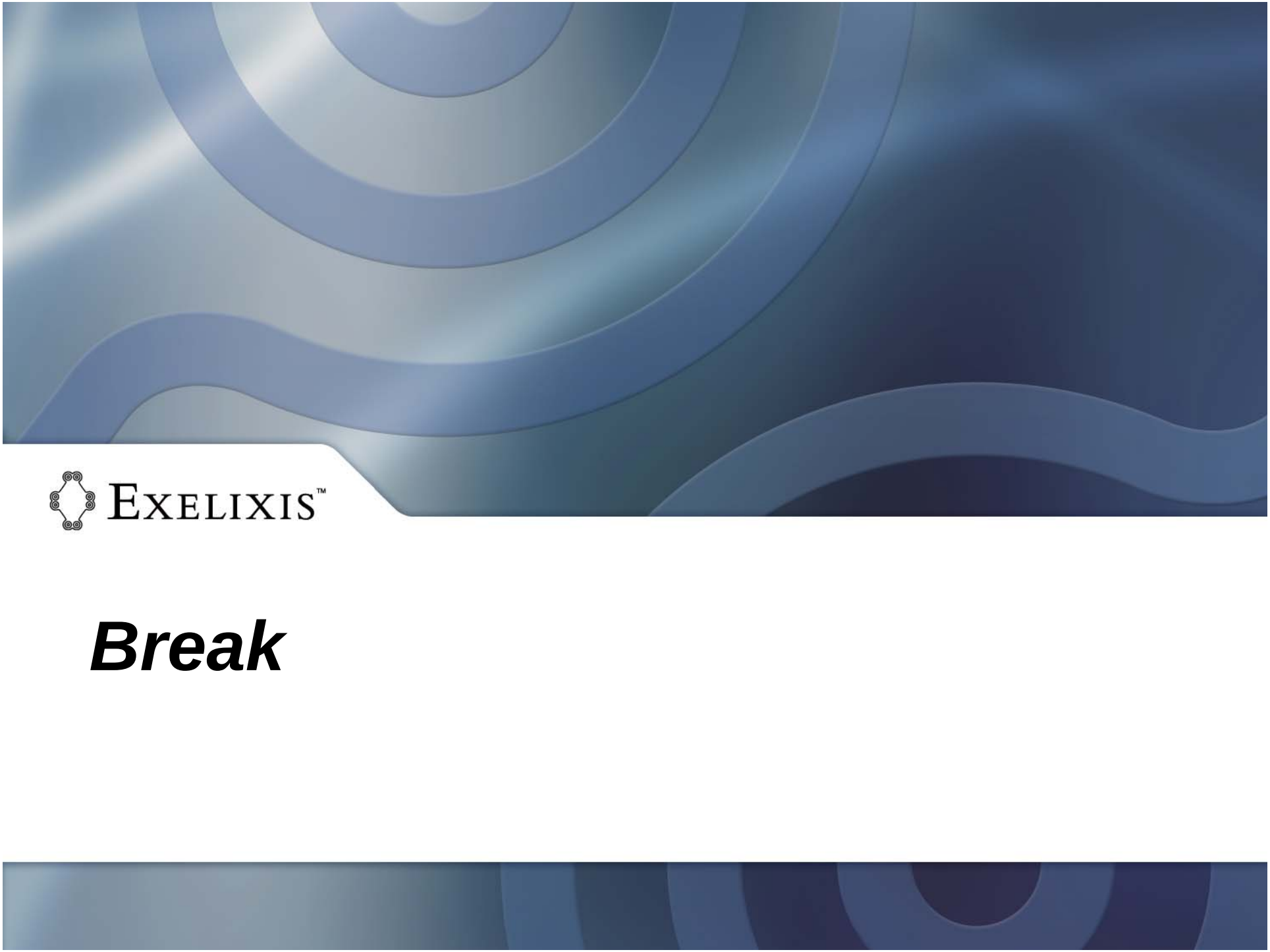
EXELIXIS™

Clinical Q&A Panel

Gisela Schwab, MD, Exelixis

Vincent Miller, MD, Memorial Sloan-Kettering Cancer Center

Steven Sherman, MD, MD Anderson Cancer Center



Break

R&D Overview
Michael Morrissey, President R&D

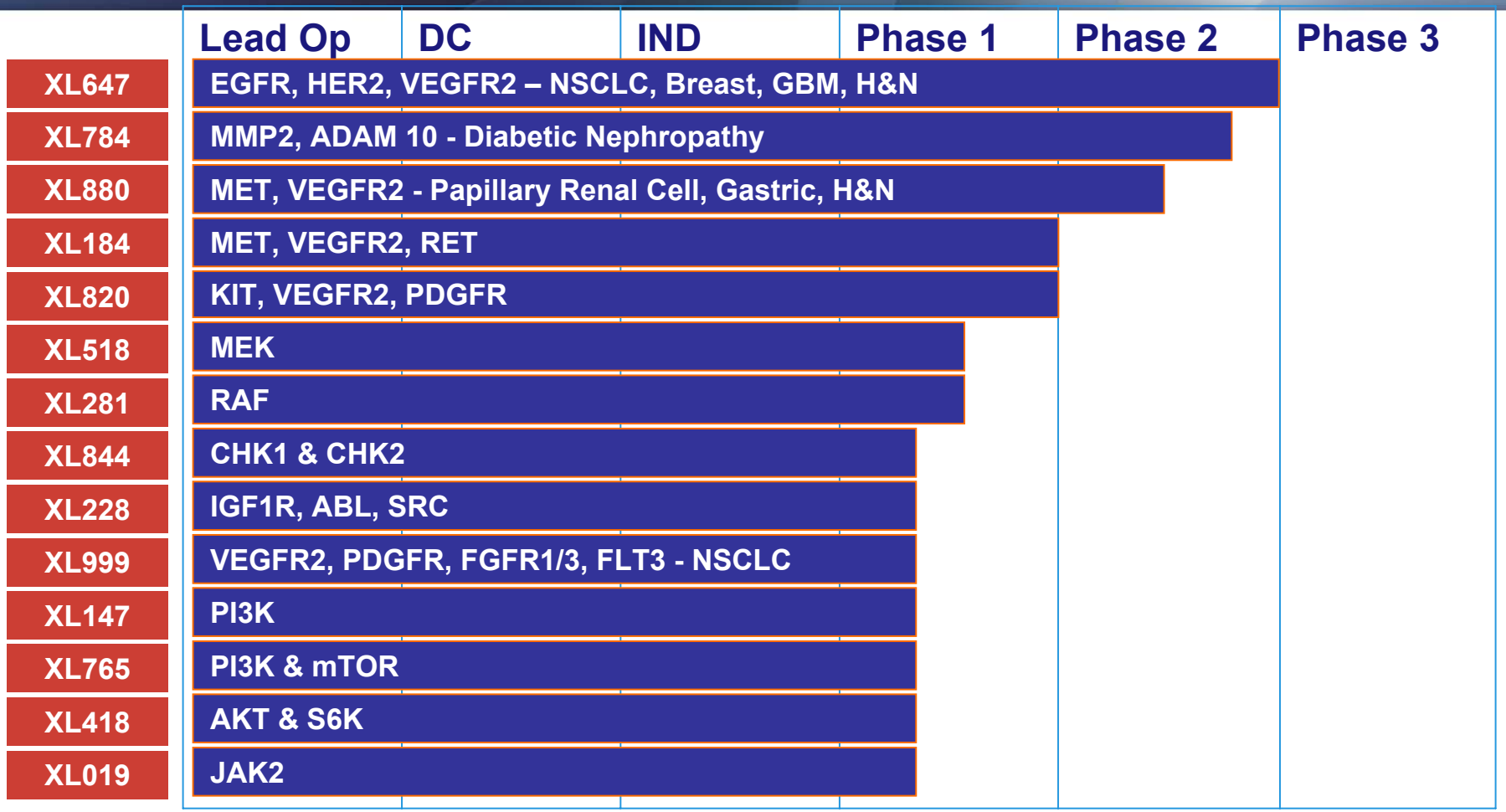
Exelixis Pipeline Update – 2008

- ***Advancement:*** XL compounds moving into Phase 2 & 3
- ***Attrition:*** XL compounds discontinued
- ***Addition:*** New XL DCs and INDs

Key components of portfolio

- Data driven decisions
- Maximize success – not minimize failure
- Product opportunities:
  - Early market entry in niche indications
  - Enhanced commercial potential with broad list of potential tumor types and indications

Exelixis Pipeline - 2007



The compounds XL647, XL784 and XL999 have been out-licensed to Symphony Evolution, Inc. and are subject to a repurchase option. Pursuant to a product development and commercialization agreement between Exelixis and GlaxoSmithKline, GlaxoSmithKline has the option, after completion of clinical proof-of-concept by Exelixis, to elect to develop up to three compounds in Exelixis' product pipeline, including XL784 and XL999, but excluding XL647, XL518, XL147, XL765 and XL019. Finally, the compound XL518 is the subject of a co-development collaboration between Genentech and Exelixis.

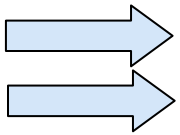
Exelixis Development Portfolio in 2008: Potential Pivotal Trials

XL647	→	Non Small Cell Lung Cancer (NSCLC)
XL784		
XL880		
XL184	→	Medullary Thyroid Cancer (MTC)
XL820		
XL518		
XL281		
XL844		
XL228		
XL999		
XL147		
XL765		
XL418		
XL019	→	Myelofibrosis (MF)

The compounds XL647, XL784 and XL999 have been out-licensed to Symphony Evolution, Inc. and are subject to a repurchase option. Pursuant to a product development and commercialization agreement between Exelixis and GlaxoSmithKline, GlaxoSmithKline has the option, after completion of clinical proof-of-concept by Exelixis, to elect to develop up to three compounds in Exelixis' product pipeline, including XL784 and XL999, but excluding XL647, XL518, XL147, XL765 and XL019. Finally, the compound XL518 is the subject of a co-development collaboration between Genentech and Exelixis.

Exelixis Development Portfolio in 2008: Potential Opt-in Compounds

- XL647
- XL784
- XL880
- XL184
- XL820
- XL518
- XL281
- XL844
- XL228
- XL999
- XL147
- XL765
- XL418
- XL019



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Exelixis Development Portfolio in 2008: Potential Phase 2 Compounds

XL647	Breast, GBM, H&N
XL784	
XL880	PRC, Gastric, H&N
XL184	NSCLC, GBM
XL820	GIST, Melanoma, AML
XL518	
XL281	Solid tumor (potentially CRC, NSCLC, melanoma)
XL844	Pancreatic + Gem
XL228	CML and/or solid tumor
XL999	
XL147	Single Agent & Combination Phase 2 with Chemotherapeutics & Targeted Agents
XL765	
XL418	
XL019	Polycythemia Vera

The compounds XL647, XL784 and XL999 have been out-licensed to Symphony Evolution, Inc. and are subject to a repurchase option. Pursuant to a product development and commercialization agreement between Exelixis and GlaxoSmithKline, GlaxoSmithKline has the option, after completion of clinical proof-of-concept by Exelixis, to elect to develop up to three compounds in Exelixis' product pipeline, including XL784 and XL999, but excluding XL647, XL518, XL147, XL765 and XL019. Finally, the compound XL518 is the subject of a co-development collaboration between Genentech and Exelixis.

Exelixis Development Portfolio in 2008: Compounds Discontinued at YE2007

- XL647
- XL784 → **Did not meet endpoint in Phase 2: Considering options**
- XL880
- XL184
- XL820
- XL518
- XL281
- XL844
- XL228
- XL999 → **CV tox at low dose – MOA similar to sunitinib**
- XL147
- XL765
- XL418 → **Low exposure in Phase 1: PI3K inhibitors promising**
- XL019

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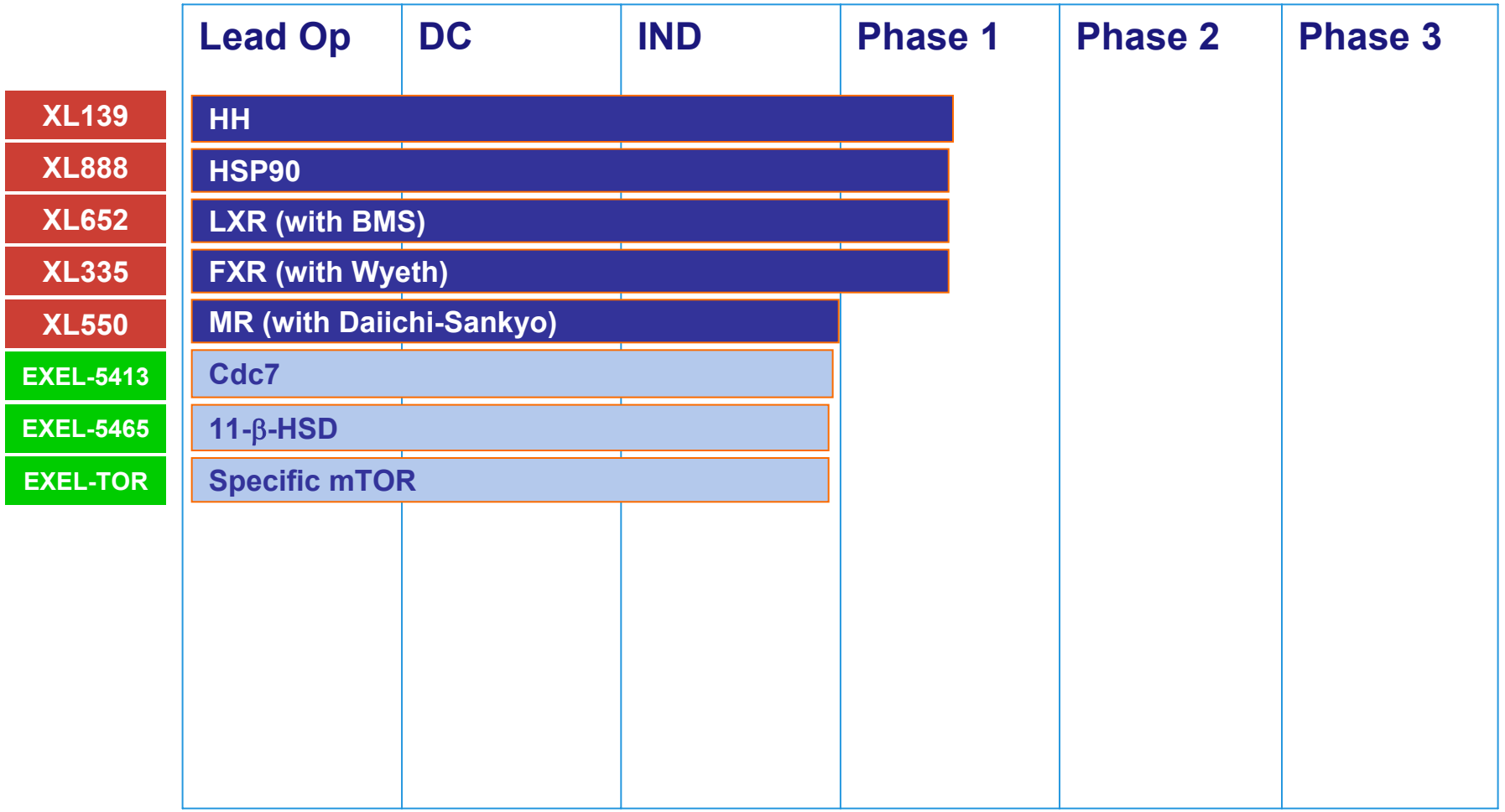
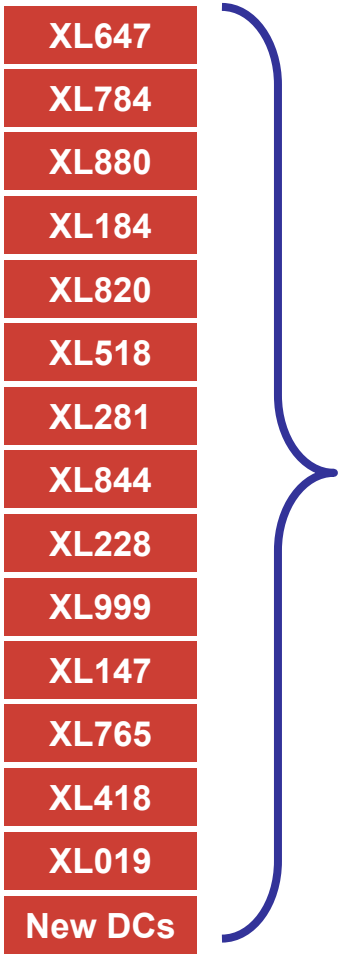
Exelixis Development Portfolio in 2008: New Projected DCs and INDs



Exelixis Development Portfolio: Evolving Landscape in 2008



Projected advancement in 2008

- 3 XLs advancing into pivotal trials
- Up to 5 XLs submitted for opt-in decisions
- 9 XLs in phase 2
- 8 XLs complete phase 1
- 2 XLs discontinued, 1 XL on hold
- 3 new XL INDs
- 3 new XL DCs

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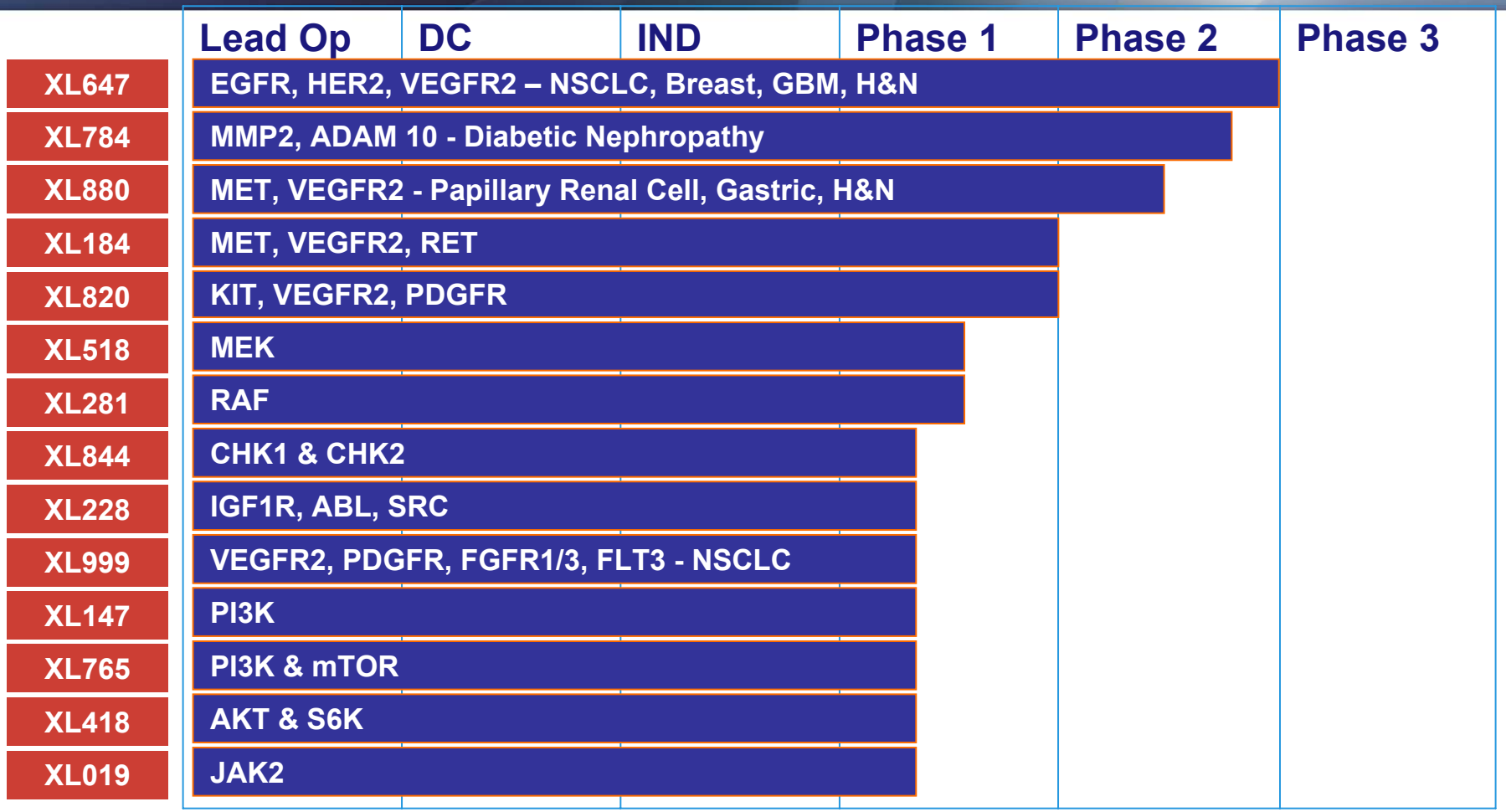
EXELIXIS™

Clinical Pipeline Update

Gisela Schwab, MD

SVP and Chief Medical Officer

Exelixis Pipeline



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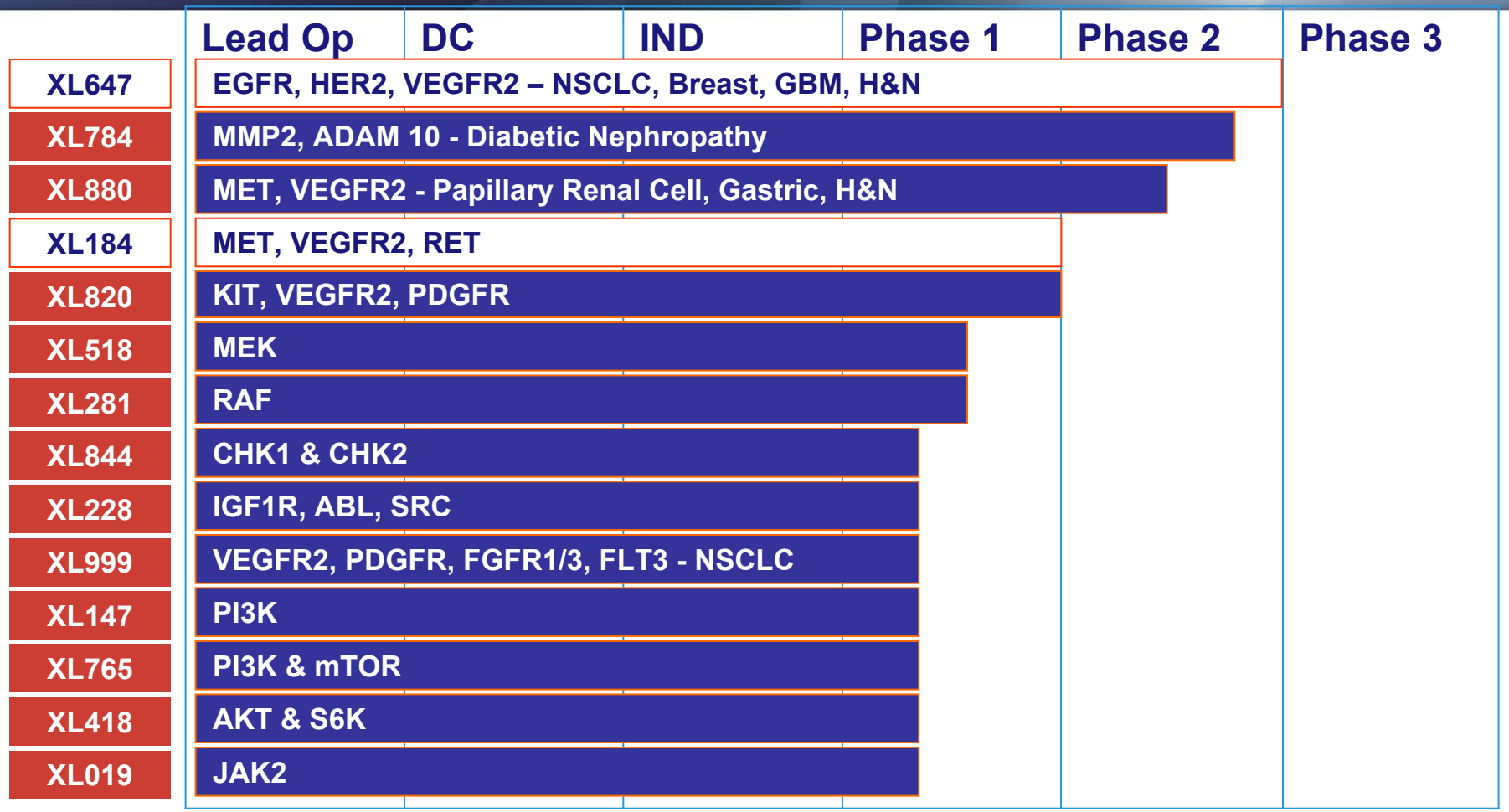
Exelixis Pipeline



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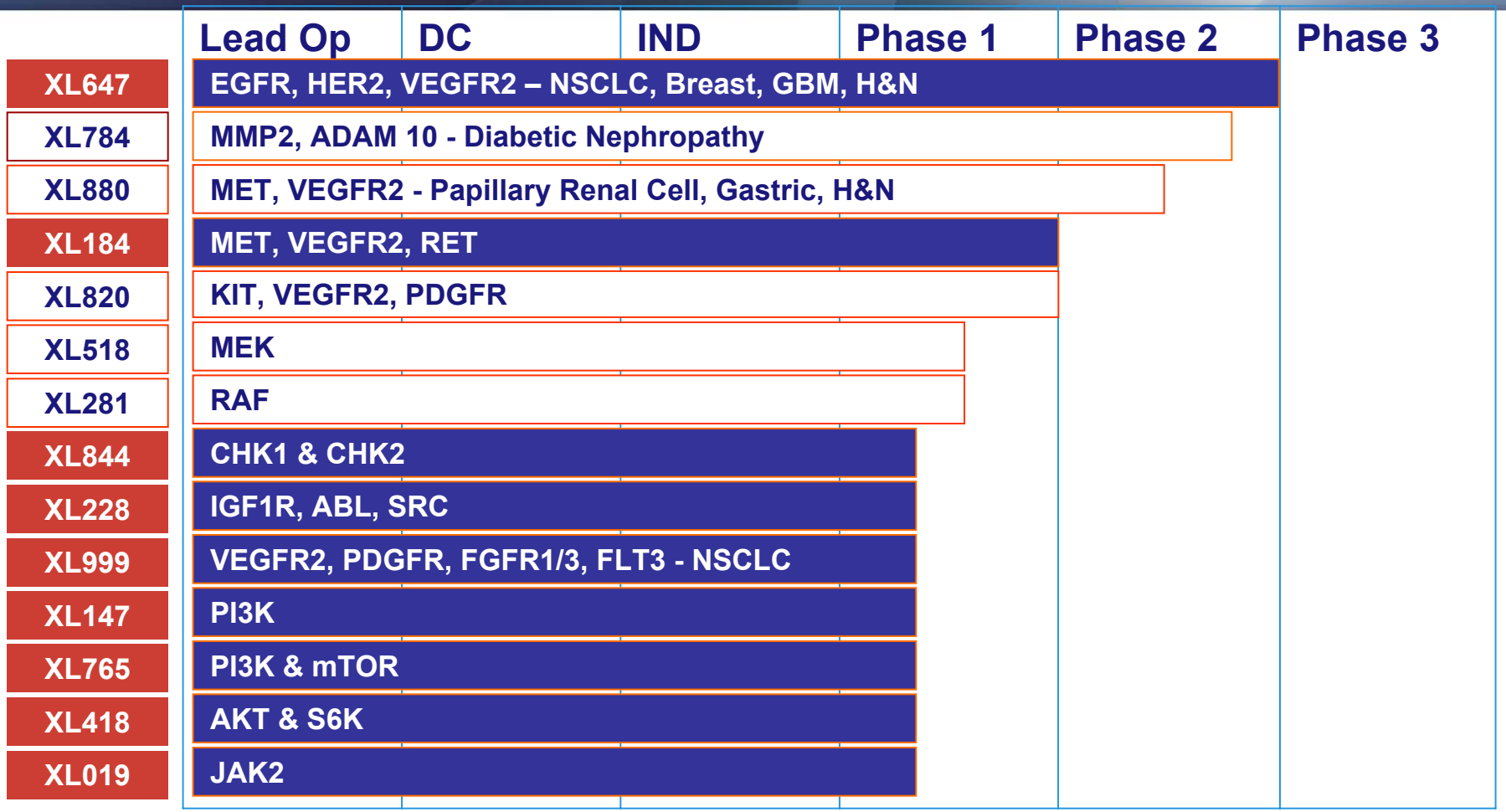
Exelixis Pipeline



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XL880 Overview

Balanced inhibitor of MET and VEGFR2

- Sub-nM inhibition, slow off rate
- Potent inhibition of activated MET mutants

Excellent pharmacodynamic properties

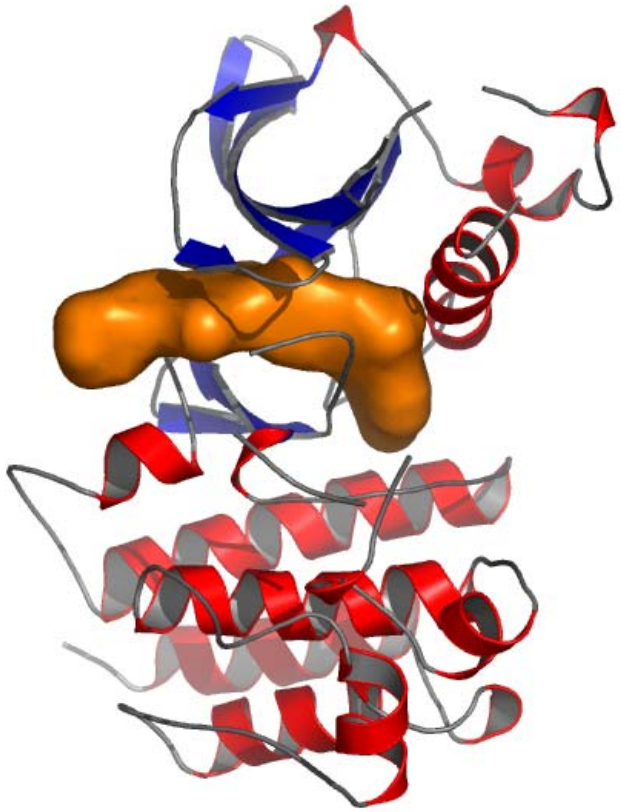
- Potent in vivo inhibition of MET and VEGFR2
- Long duration of action

Potent oral activity in multiple xenograft models

- Regression of large tumors
- Intermittent/single dose efficacy

Clinical Profile

- Clear anti-tumor activity in Phase 1 and 2
- Generally well tolerated
- Potent in vivo inhibition of MET and VEGFR2 (paired tumor and plasma samples)
- Evaluated intermittent (5&9) and daily dosing



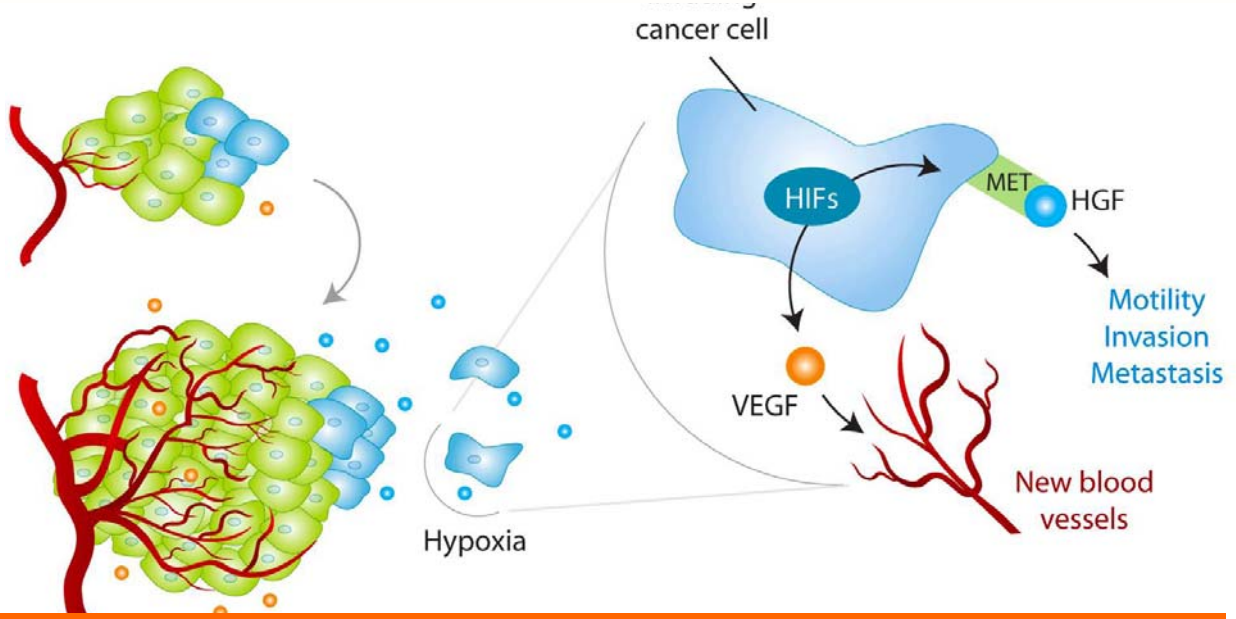
MET and VEGFR Cooperate to Promote Tumor Survival

VEGF induction promotes angiogenesis

- Improved tumor blood flow, better oxygenation

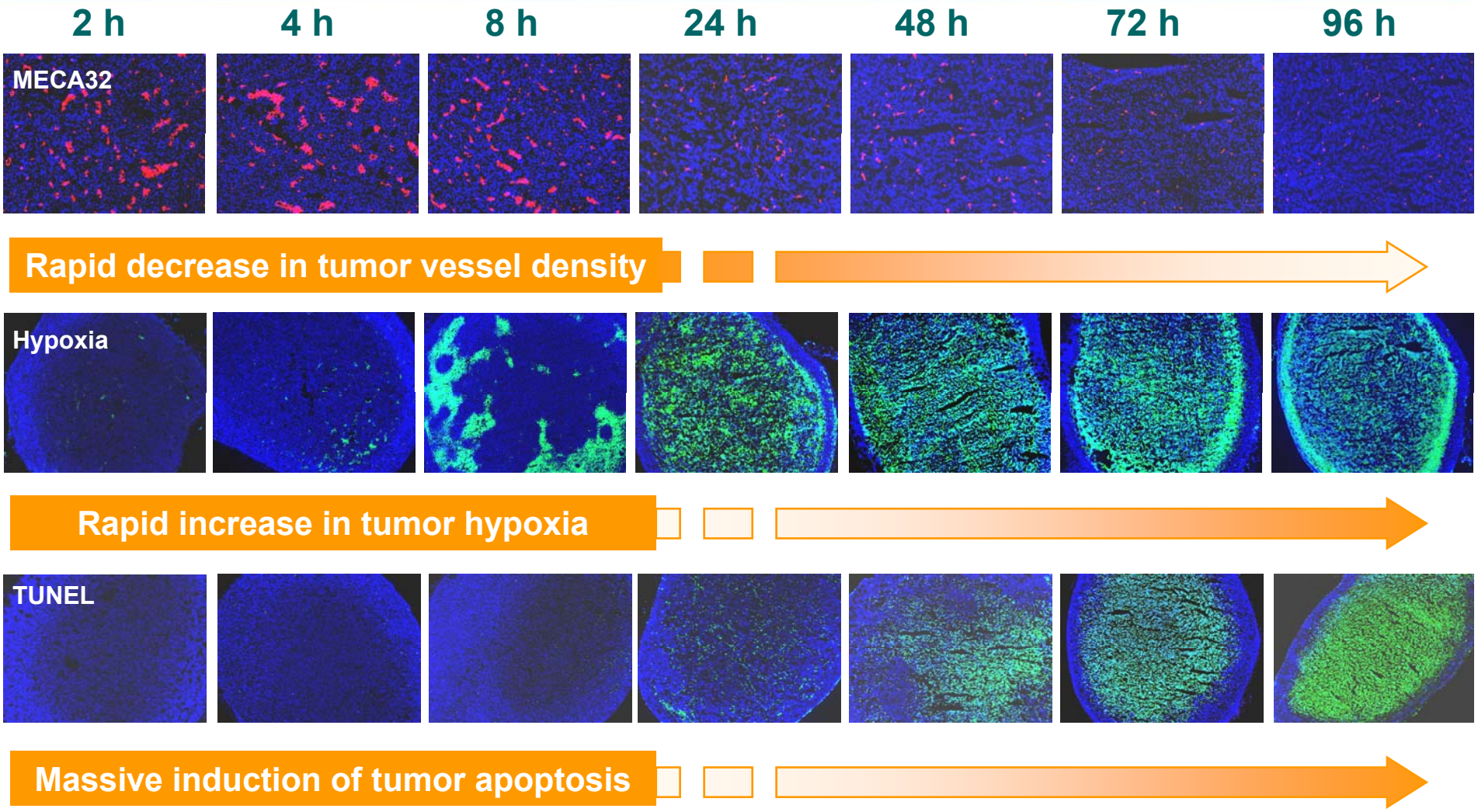
MET expression promotes survival, migration and invasion

- Migration to better oxygenated tissue



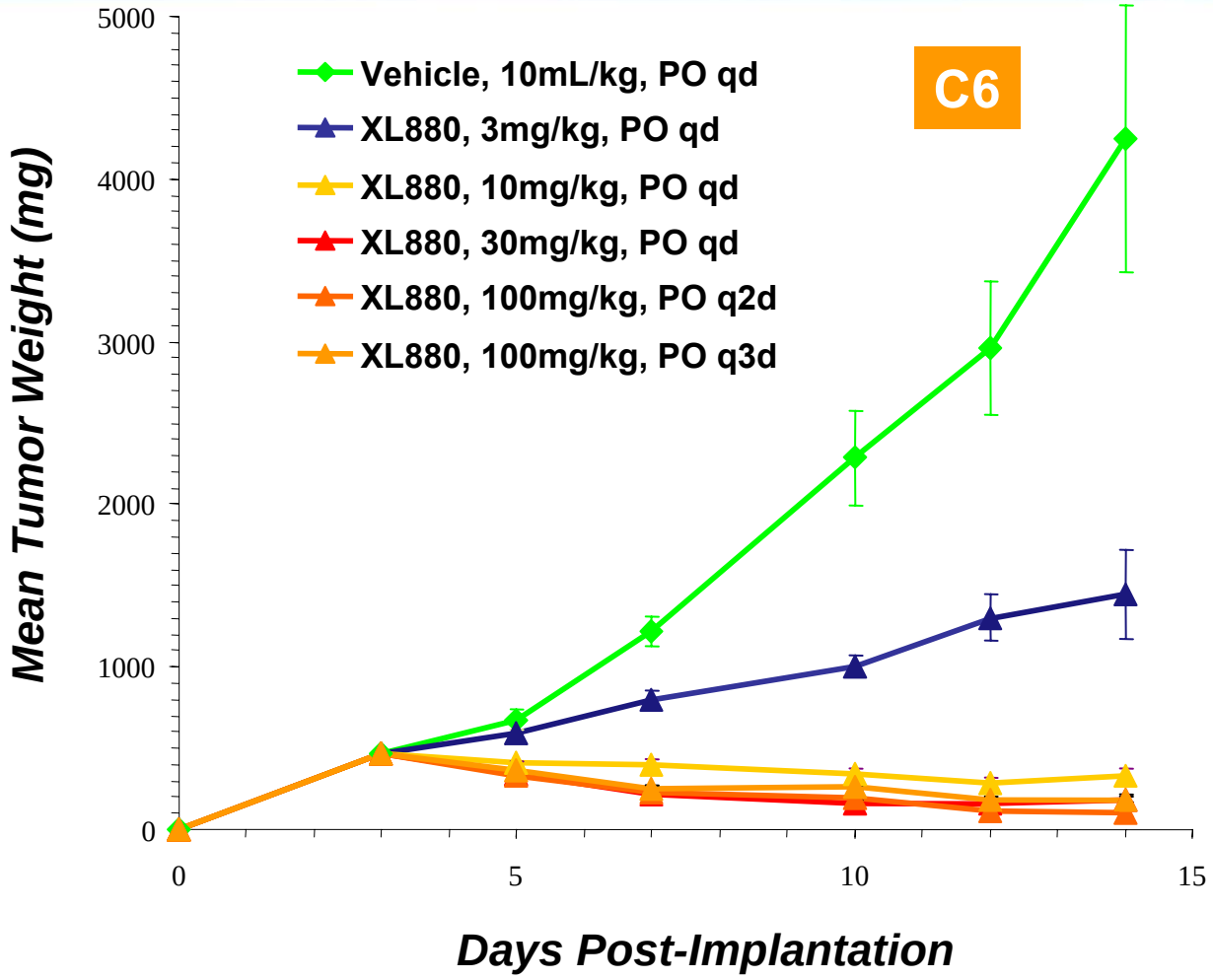
Dual inhibition of MET and VEGFR blocks tumor escape mechanisms used to overcome hypoxia

XL880: Acute Effects on Tumors



MDA-MB-231 xenograft tumors

XL880 Tumor Xenograft Efficacy

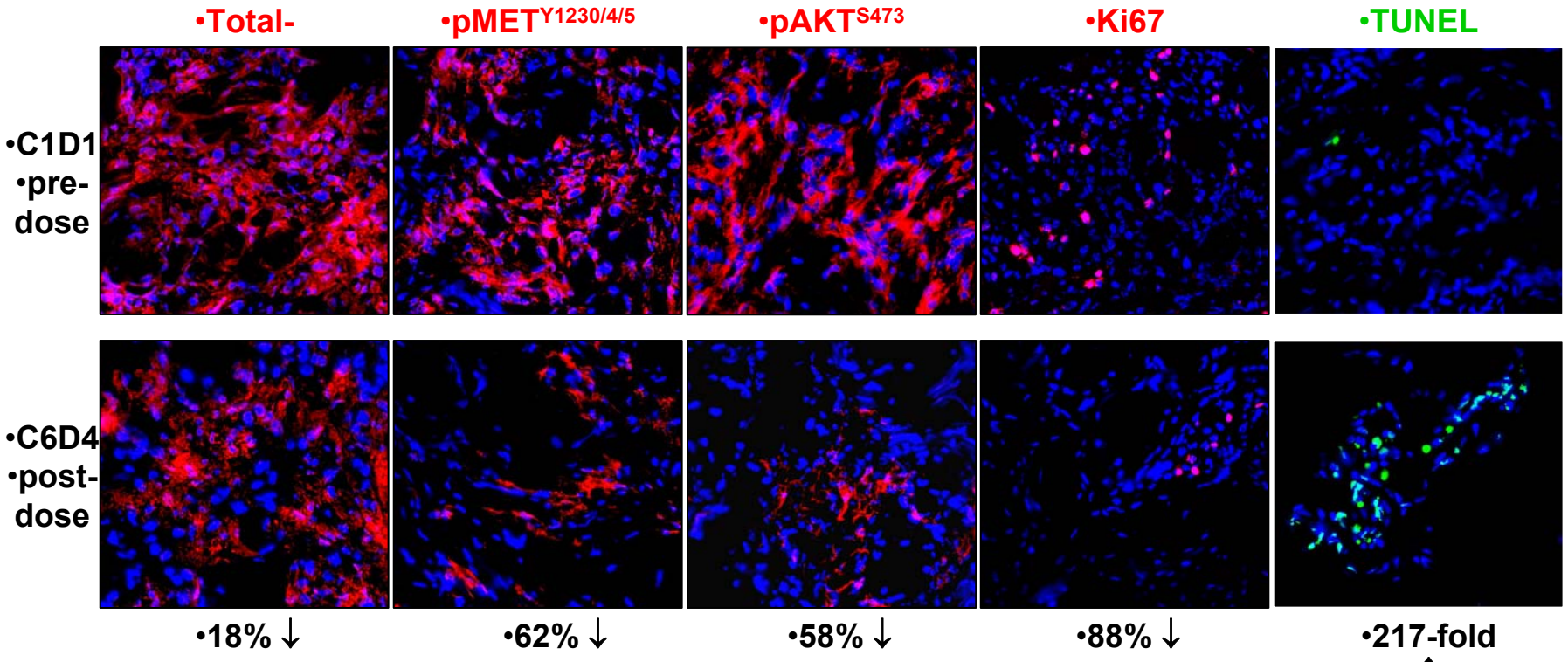


Model	ED50 mg/kg
MBA-231*	4
HT29	7
H441*	7
U87	2
C6*	2

*Regression of large (0.5-1.0g) tumors

No discernable tumors in some animals

Biological Activity of XL880 in Patients Phase 1

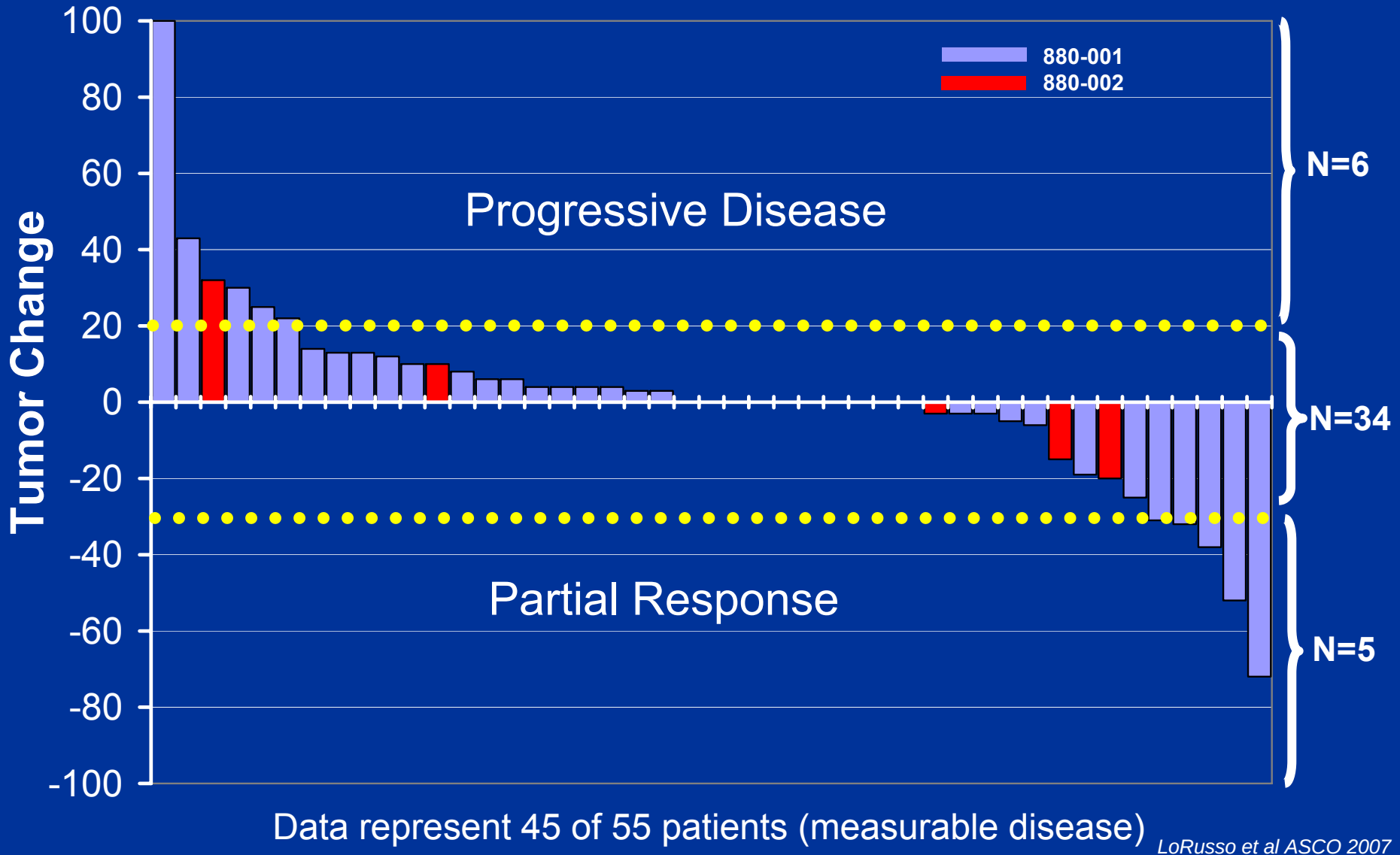


XL880 inhibits MET and associated downstream signaling in tumors

Similar effects observed in tumor biopsies from 4 patients (melanoma, breast, thyroid, and PRC), but not in biopsies of normal skin

Clinical observations of hypertension and proteinuria indicative of VEGFR2 inhibition

Best XL880 Tumor Response



Data represent 45 of 55 patients (measurable disease)

LoRusso et al ASCO 2007

Phase 1 Efficacy Summary of XL880

Outcome	XL880-001	XL880-002	Total
	Tumor (duration in months)	Tumor (duration in months)	
Partial Response (RECIST)	3 PRC (5, 10+, 23+) MTC (10+) Hurthle Cell Thyroid (11)		5
Minor Response (decrease <30%)	RCC (5) Carcinoid (11) Melanoma (4) CRC (6) MTC (4) Urethral (5)	Nasopharyngeal (6) Papillary thyroid (6+) CRC (3+)	9
Stable Disease (>3 months)	4 CRC (6, 4, 4, 8) Biliary (5) Ovarian (8+)	NSCLC (4) Chondrosarcoma (3) RCC (4+) Mesothelioma (3+) CRC (4)	11

PRC-Papillary Renal Cell, CRC- Colorectal Cancer, RCC- renal cell carcinoma,
MTC- Medullary Thyroid carcinoma, (n=55 subjects)

LoRusso et al ASCO 2007

XL880 - Phase 2 Program

Papillary Renal Cell Cancer

- Stratified phase 2 study
  - MET activated or not

Gastric Cancer

- Poorly differentiated
- 7q31 amplification

Head and Neck Cancer

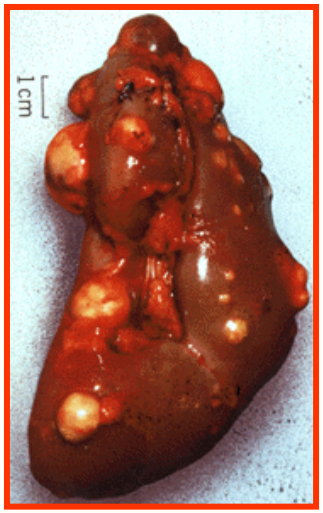
- MET often over-expressed

Good rationale for other indications including breast cancer, NSCLC, CRC, pancreatic cancer

Papillary Renal Cancer

Papillary carcinomas represent ~ 15% of all renal cancers

- Typically bi-lateral, multi-focal tumors
- Only treatment is surgery (nephrectomy)



Both sporadic and hereditary forms are known

- Hereditary form is caused by germline activating mutations in MET
- Mutations in MET also documented in sporadic form (15%)
- Other genetic abnormalities in MET

Both VEGF and VEGFR2 mRNAs are upregulated in PRC compared to normal kidney

- VEGF pathway inhibition has shown evidence of activity (sunitinib and sorafenib)

A Phase 2 Study of the Dual MET/VEGFR2 Inhibitor XL880 in Patients (pts) With Papillary Renal Carcinoma (PRC)

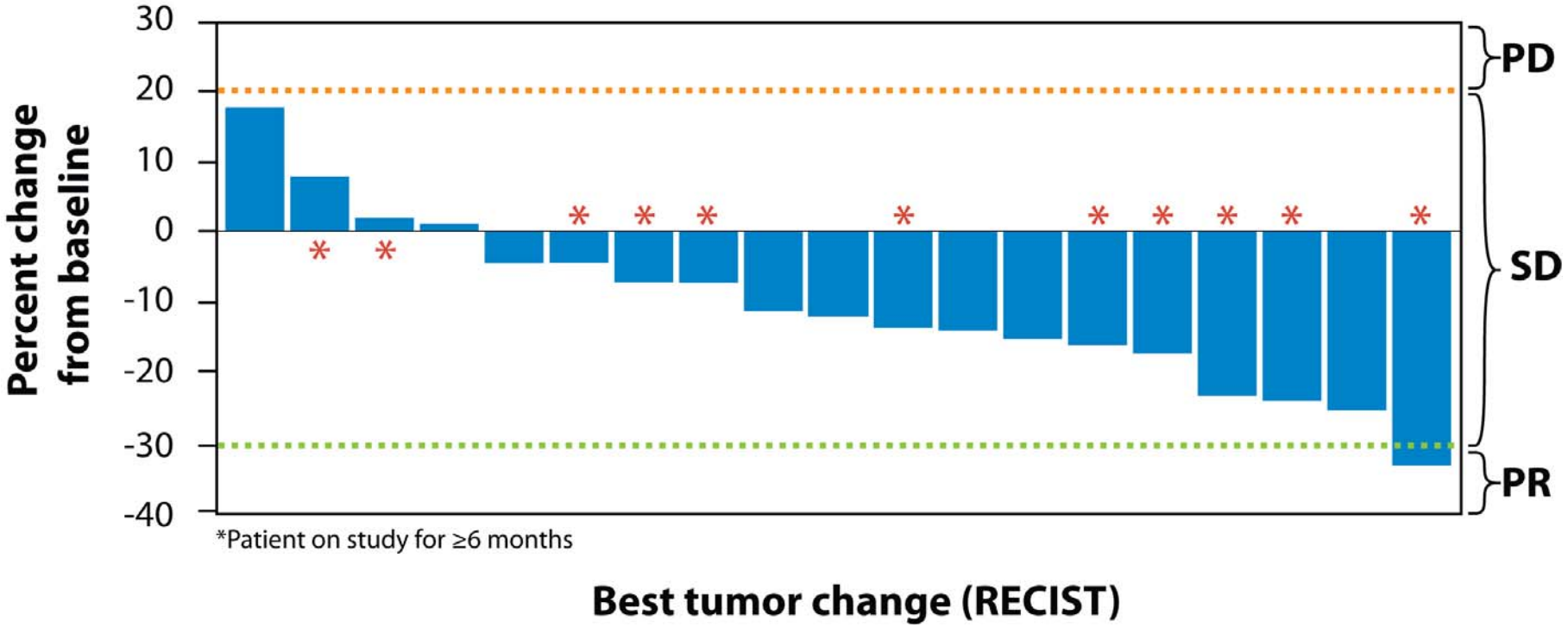
Robert Ross,[1] Ramaprasad Srinivasan,[2] Ulka Vaishampayan,[3] Ronald Bukowski,[4] Jonathan Rosenberg,[5] Peter Eisenberg,[6] Theodore Logan,[7] Sandhya Srinivas,[8] Mark Stein,[9] Thomas Mueller,[10] Harold Keer,[10] on behalf of the XL880-201 Investigators and Exelixis R&D

[1]Dana-Farber Cancer Institute, Boston, MA; [2]National Cancer Institute, Bethesda, MD; [3]Karmanos Cancer Institute, Detroit, MI;
[4]The Cleveland Clinic Foundation, Cleveland, OH; [5]UCSF Urologic Oncology Clinic, San Francisco, CA;
[6]California Cancer Care, Greenbrae, CA; [7]Indiana University, Indianapolis, IN; [8]Stanford University, Stanford, CA;
[9]The Cancer Institute of NJ, New Brunswick, NJ; [10]Exelixis, Inc., South San Francisco, CA

Baseline Characteristics

Characteristic	Value Number of patients (N = 16)
Median age (range), years	56 (25-79)
Sex, M/F (%)	13/3 (81/19)
Race, n (%)	
Black or African-American	1 (6.25)
White	14 (87.5)
Other	1 (6.25)
ECOG status, n (%)	
0	12 (75)
1	4 (25)
Prior cancer therapy alone, n (%)	0
Prior radiation therapy, n (%)	2 (12.5)
No prior therapy reported, n (%)	14 (87.5)

Tumor Response in the XL880-201 Population



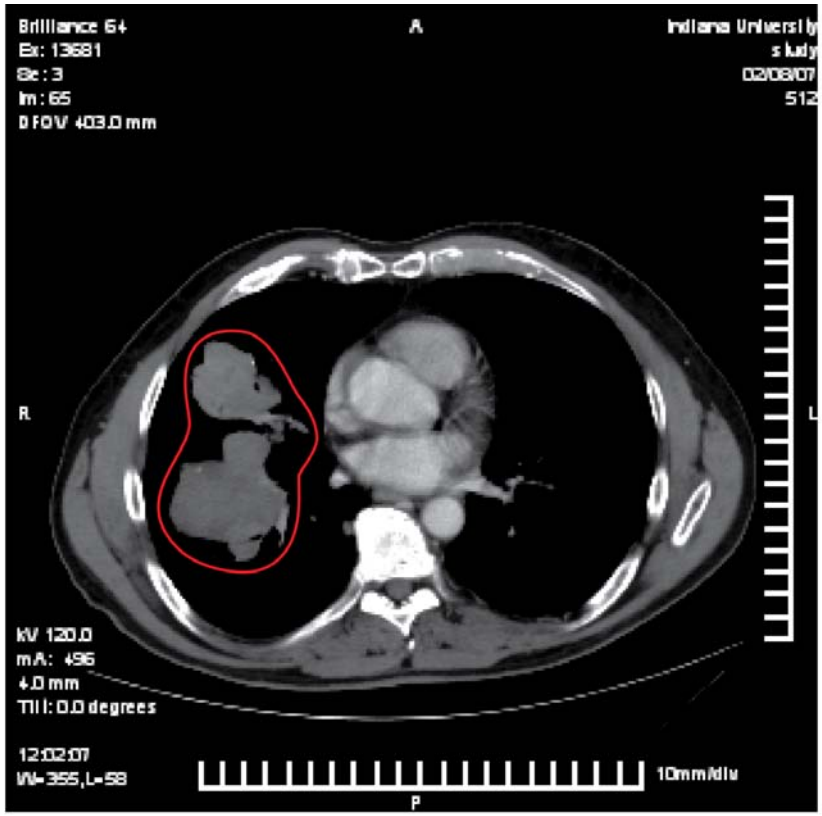
PD, progressive disease; SD, stable disease; PR, partial response.

Time on Study and MET Mutation Status (N = 21)[a]

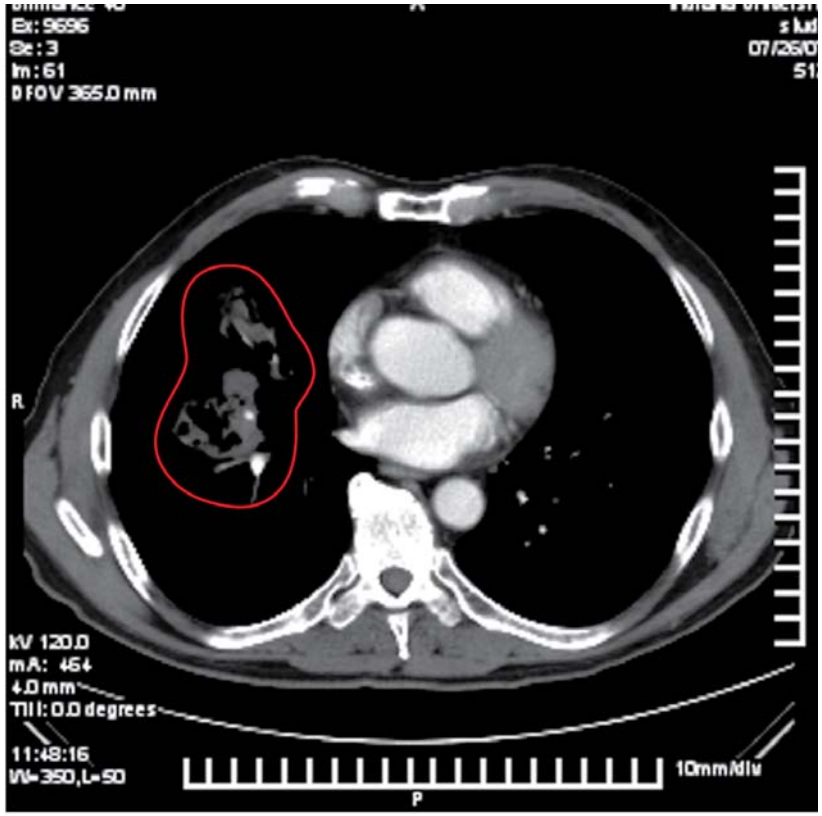
Patient no.	Time on study (months)	Change in tumor (RECIST)	MET status altered	MET sequence (germline)
1	15+	SD (↓23%)	No	WT
2	11+	SD (↑8%)	No	WT
3	10+	SD (↓14%)	No	WT
4	9+	SD (↓7%)	No	WT
5	9+	SD (↓4%)	No	WT
6	9	SD (↓7%)	No	WT
7	8+	SD (↓24%)	No	WT
8	8+	SD (↑2%)	No	WT
9	8+	PR (↓33%)	No	WT
10	7	SD (↓16%)	No	WT
11	6+	SD (↓17%)	No	WT
12	6	SD (↑1%)	No	WT
13	4+	SD (↓14%)	Yes	H1094R (KD) het
14	4	SD (↓11%)	No	WT
15	4	SD (↓12%)	No	WT
16	3+	N/A	Yes	H1094R (KD) het
17	3	SD (↓25%)	Yes Tumor somatic mutation	WT
18	3	SD (↓4%)	No	WT
19	3	SD (↑18%)	No	WT
20	2+	SD (↓15%)	Yes	H1094R (KD) het
21	<1+	TEE	Yes	H1094R (KD) het

+ indicates active on study treatment
SD, stable disease; TEE, too early to evaluate; WT, wild type; PR, partial response
[a]As of October 10, 2007.

Representative CT Scanning Images of the Patient Who Achieved Partial Response

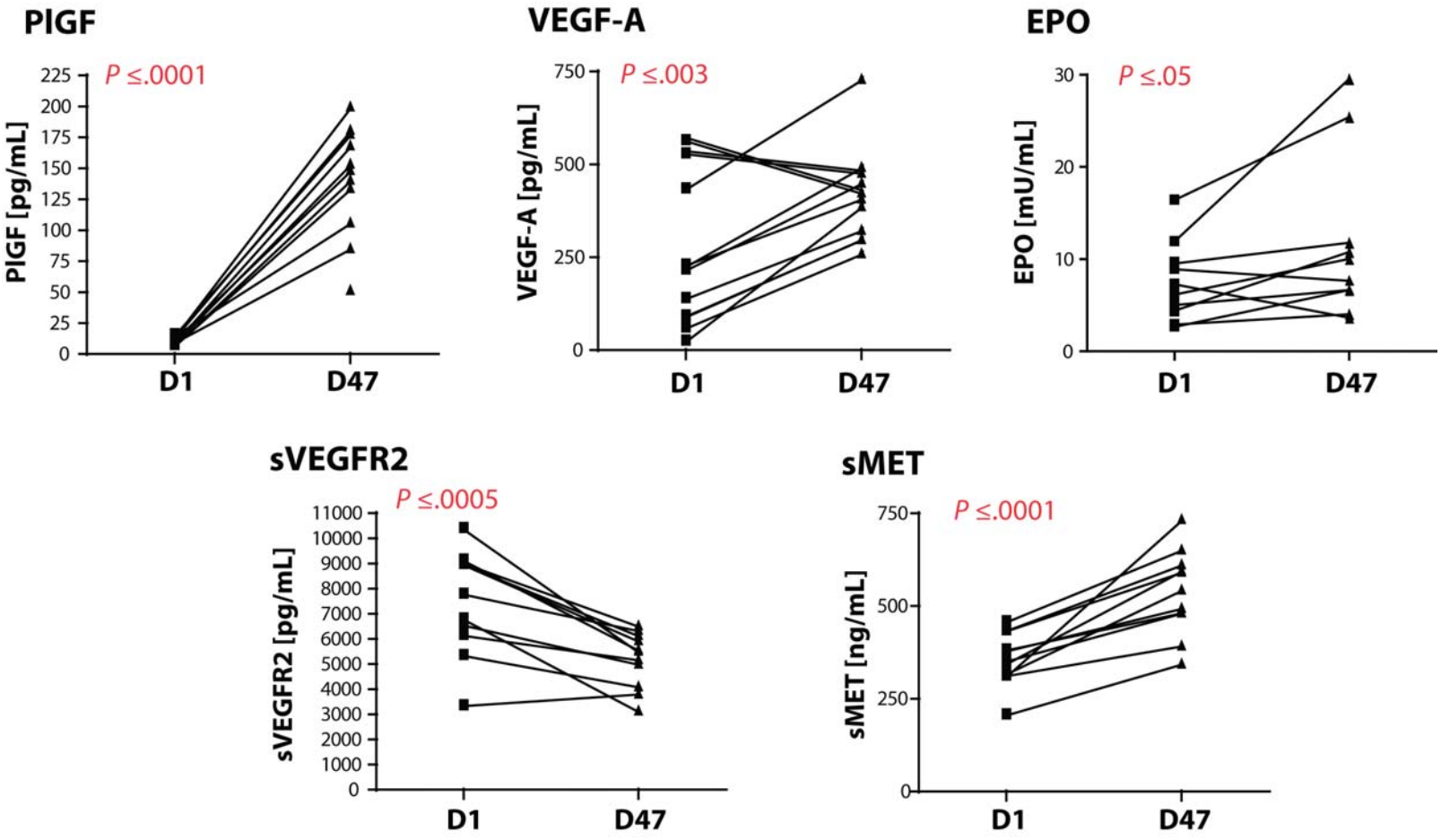


Scan 2/08/07
(baseline)



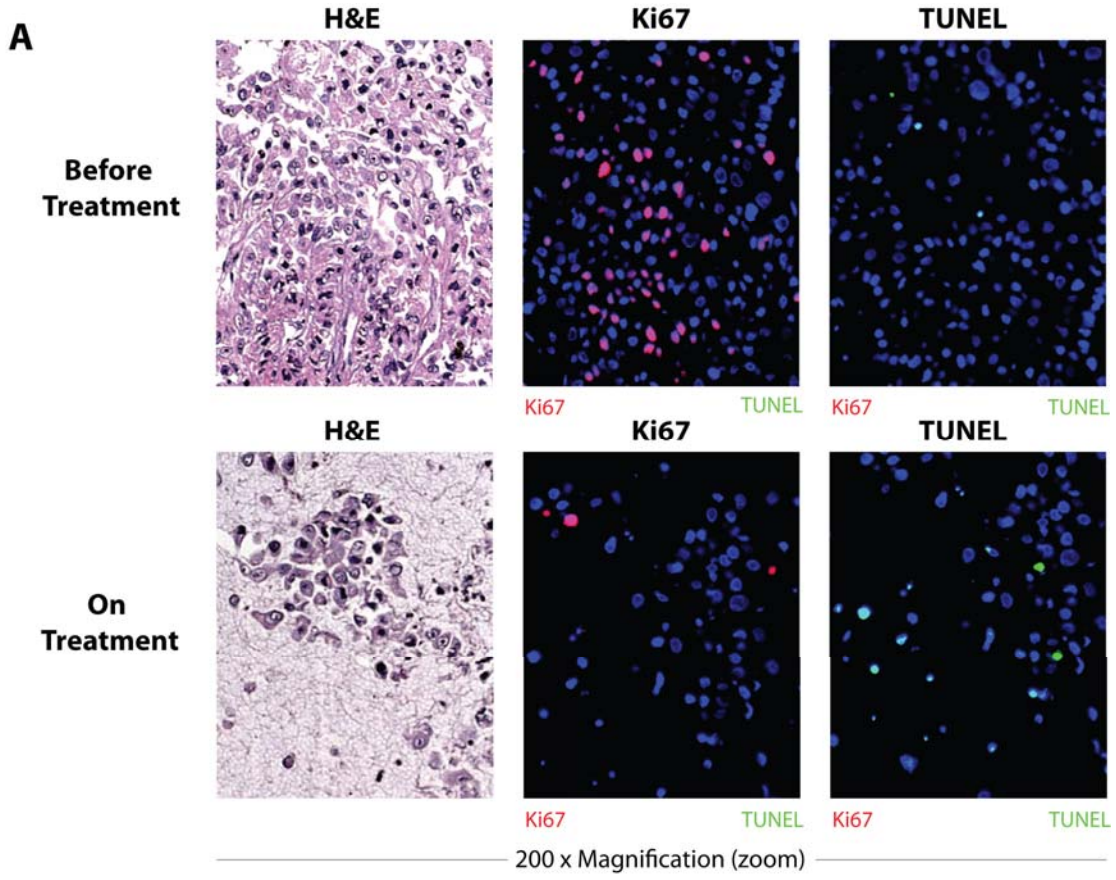
Scan 7/26/07
(confirmed ~30 days later)

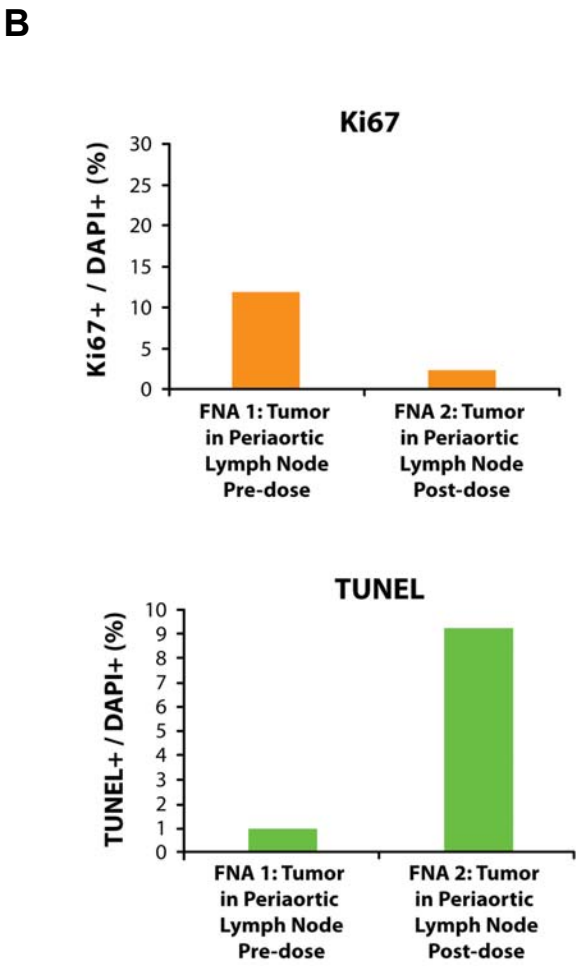
Analysis of Exploratory Biomarkers in Plasma Samples of Patients Enrolled in Study XL880-201



Modulation of soluble plasma biomarkers consistent with the anti-angiogenic activity of XL880

IHC Analysis of Sequential Samples From a Periaortic Lymph Node Metastasis





Adverse Events Reported in ≥10% of Patients

	Total of 16 patients, n (%)	
Preferred term	**Related[a]**	**Regardless of relationship**
Hypertension	7 (44)	7 (44)
Diarrhea	2 (13)	3 (19)
Dizziness	2 (13)	2 (13)
Fatigue	2 (13)	3 (19)
Headache	2 (13)	3 (19)
Dysphonia	0	2 (13)
Nausea	1 (6)	3 (19)
Vomiting	1 (6)	2 (13)

[a]There was only 1 Grade 4 AE (GGT increased) considered related to XL880; no Grade 5 AEs were judged related to XL880 treatment.

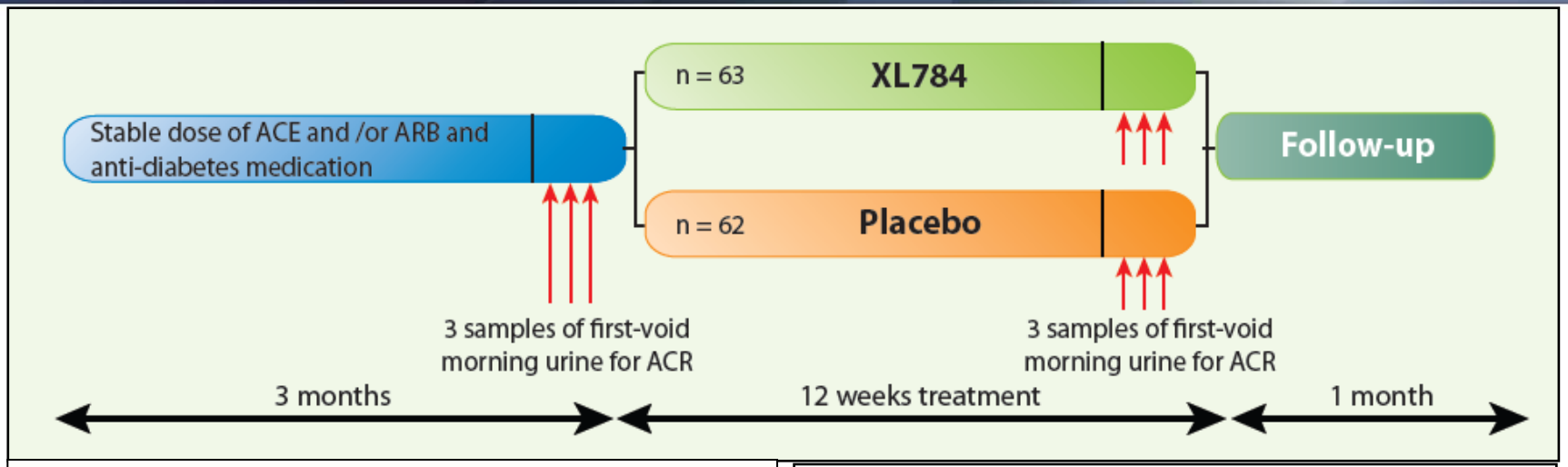
XL880 Conclusions

XL880 demonstrates MET & VEGFR2 inhibition both preclinically and in the clinic

XL880 is generally well tolerated

XL880 demonstrates clear antitumor activity in a variety of tumor types in the Phase 1 and in Phase 2 in PRC

XL784 – Randomized Phase 2 study in Diabetic Nephropathy



Inclusion

ACR ≥ 500 mg/g

GFR ≥ 30 ml/min/1.73m^2

Stable blood pressure

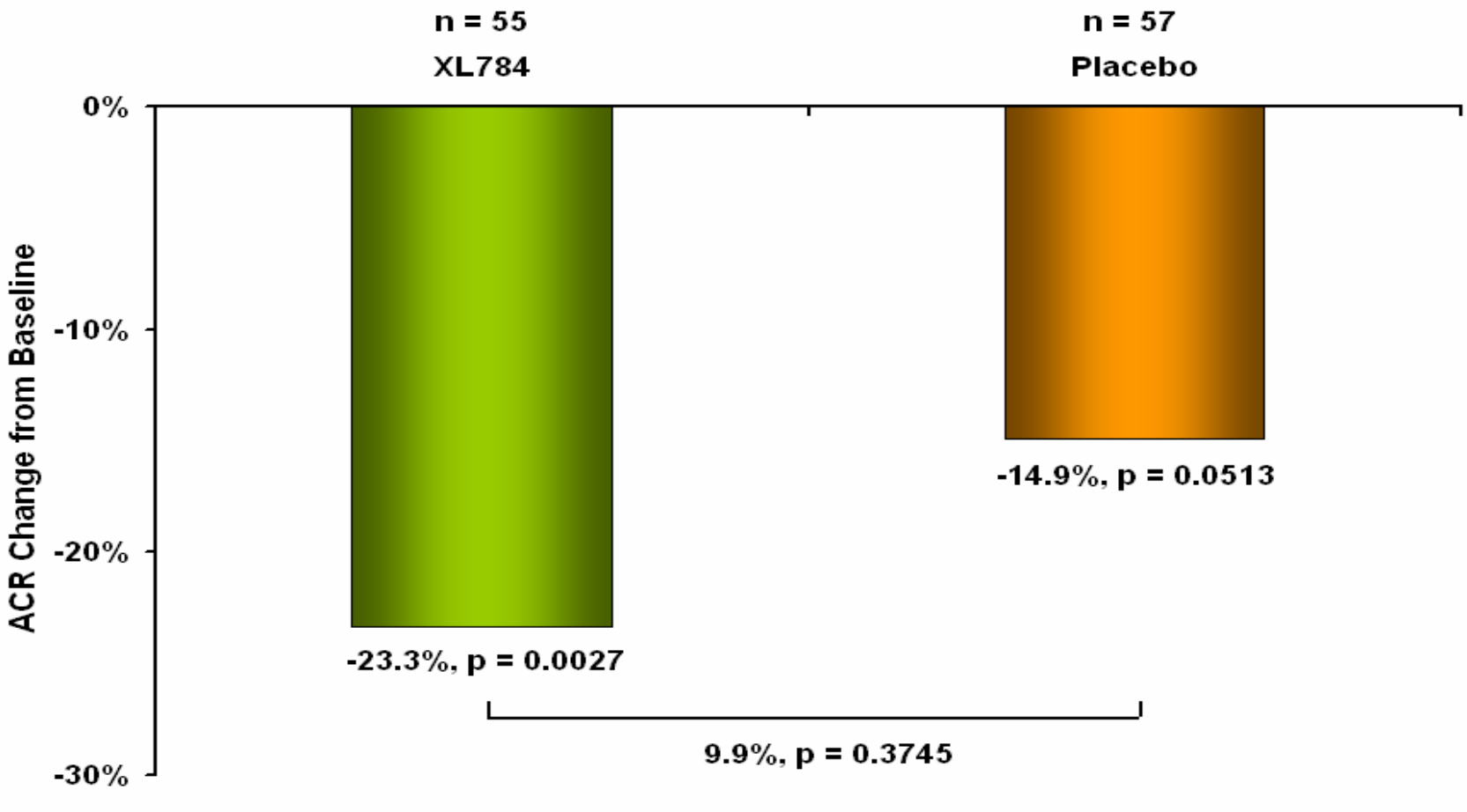
Stable dose and schedule of ACE and / or ARB for 3 months

Stable dose and schedule of antidiabetic medication for 3 months

Exclusion

HbA1c > 10%

Failed to Meet Primary Endpoint (mITT)



95% CI for treatment difference: (-13.6%, 29.5%)

Subgroup Analyses

EXELIXIS™

Planned

Baseline ACR

Baseline GFR

Baseline ACR x GFR

Type 1 vs. Type 2

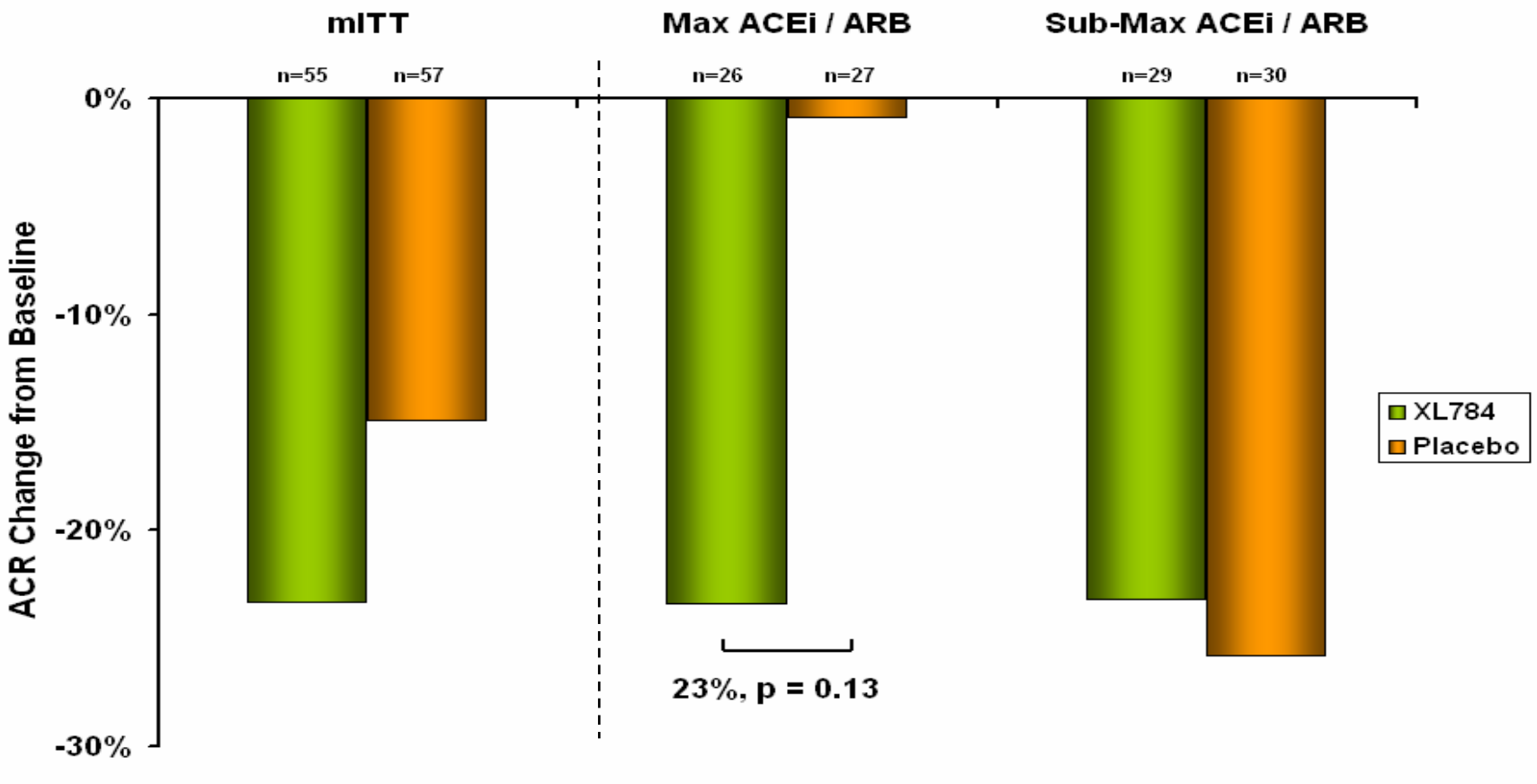
African American vs. Other

Exploratory

Maximal ACEi / ARB therapy

Maximal ACEi / ARB and Baseline HbA1c ≤ 7.8%

Max vs. Sub-Max ACEi/ARB dose (mITT)



Adverse Events

XL784: 200 AEs reported amongst 48 (77%) subjects

Placebo: 258 AEs reported amongst 53 (85%) subjects

AEs reported by more than 10% of subjects in either group

Number (%) of patients	XL784 N=62	Placebo N=62
Nausea	8 (13%)	4 (6%)
Diarrhea	6 (10%)	6 (10%)
Cough	3 (5%)	9 (15%)
Edema peripheral	3 (5%)	8 (13%)
UTI	3 (5%)	7 (11%)
Pitting edema	2 (3%)	6 (10%)

Source: Appendix Table 16.1

Summary

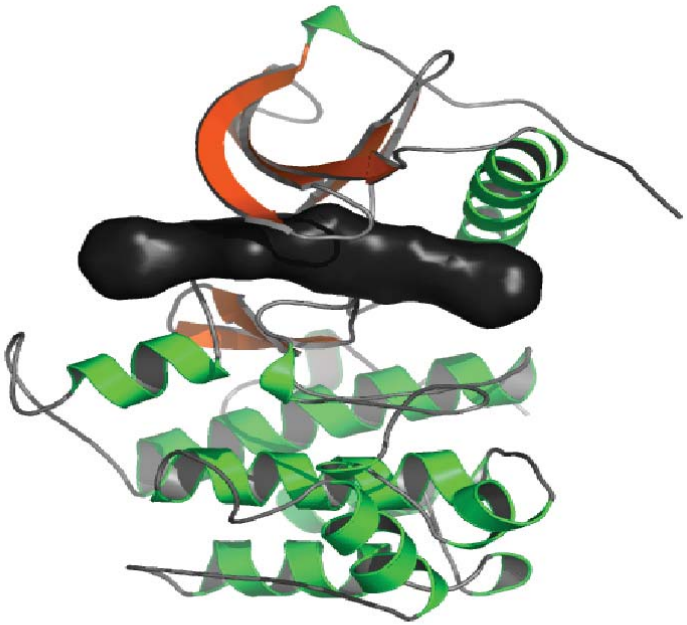
EXACT Study

- Failed to meet primary endpoint
- Generally well tolerated
- Encouraging results in subgroup of subjects treated with maximal doses of ACEi / ARBs

PoC package delivered to GSK; decision expected mid-Jan 2008

Further development plan under consideration

XL820 Overview



XL820 inhibits wild-type and mutationally activated human KIT, PDGFRβ, FLT3 and KDR

Pharmacodynamics:
In vivo effects on tumor vasculature and tumor cell proliferation

Preclinical efficacy models:
Potent antitumor activity against solid tumors in rodents

Clinical Profile:
Evaluated daily and BID dosing in Phase 1
Generally well tolerated
PK: exposure increases less than proportional with daily dosing
PD: Inhibition of target molecules in surrogate tissue (hair) and plasma

XL820: Advancing to Phase 2

Phase 2 study initiated in GIST

- Patients with progressive disease or intolerance to Imatinib and/or Sunitinib

Phase 2 study starting in Malignant Melanoma

- Arising in sun damaged skin
- Acral melanoma
- Mucosal melanoma
- Stratified by KIT amplification of mutation

Rationale for Targeting RAF/MEK/ERK: Major Signaling Pathway Downstream of RTKs

B-RAF activating mutations

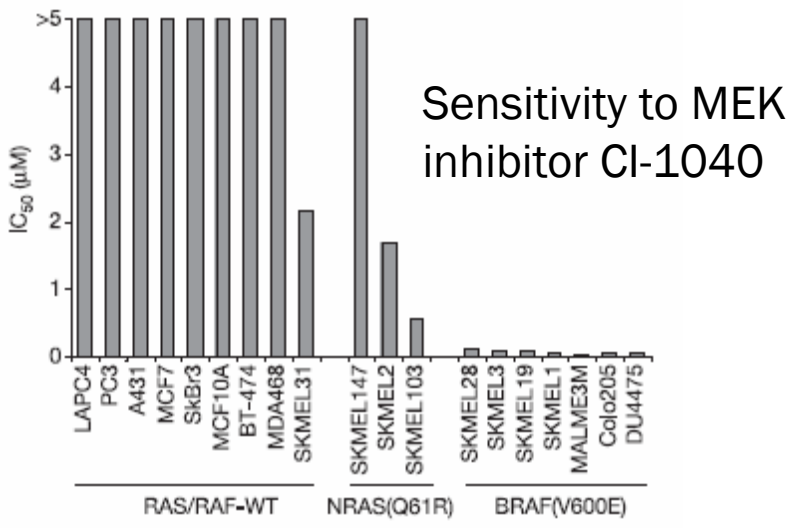
- 66% of malignant melanomas
- Also prevalent in papillary thyroid carcinomas, low-grade ovarian serous carcinoma, 5-10% of colorectal tumors

RAF1/C-RAF- increased expression and activity

- 55%-100% of renal cell carcinomas, high grade ovarian cancers
- HER2-driven breast tumors and androgen-insensitive prostate cancer

B-RAF mutation predicts sensitivity to MEK inhibition

- Solit et al. (2006)



Sensitivity to MEK inhibitor CI-1040

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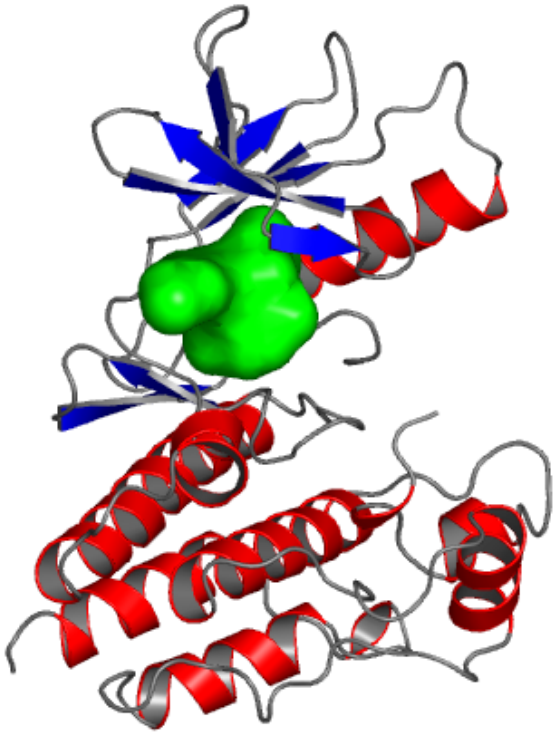
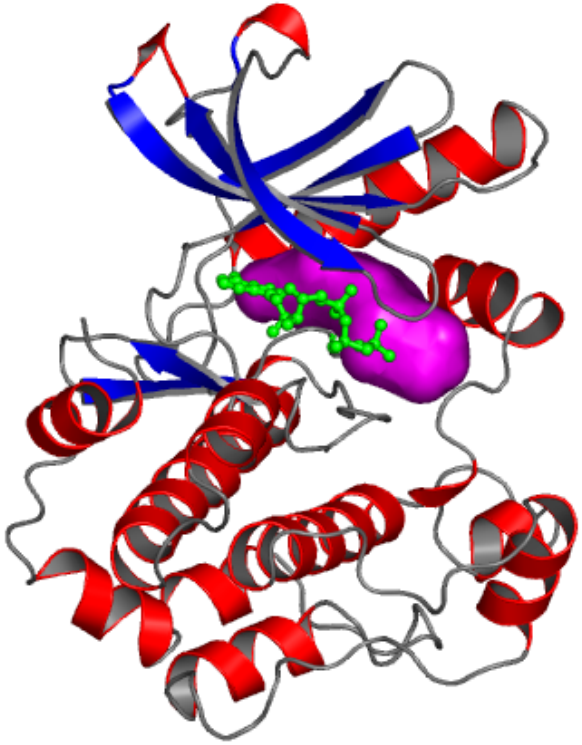
RAF/MEK/ERK Pathway Modulators

XL281:
Highly specific, ATP-competitive RAF kinase inhibitor



XL518:
Highly specific, Non ATP-competitive MEK kinase inhibitor



Both highly specific: different binding modes

Raf Inhibitor XL281

Kinase	IC_{50} (nM)
C-RAF	3.6
B-RAF	4.5
B-RAF V600E	5.0

Phase 1 study ongoing in patients with solid malignancies

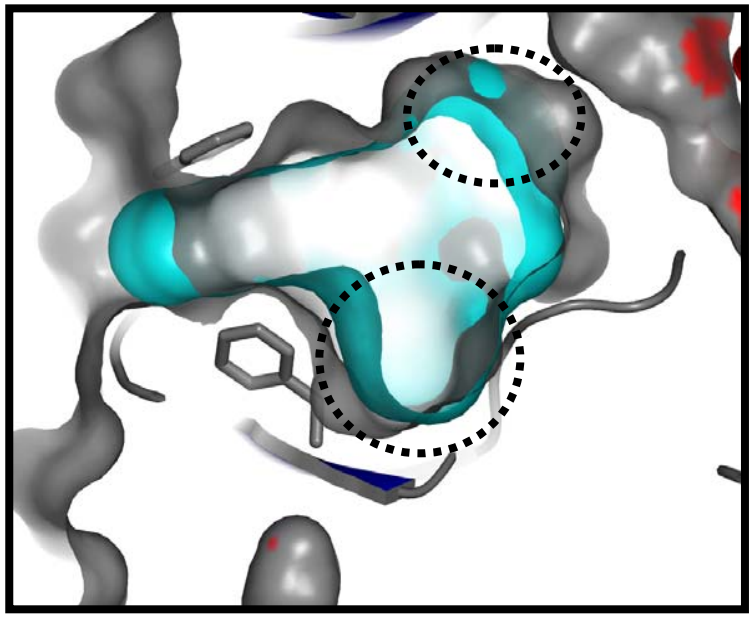
ATP competitive, reversible, highly selective

XL281 binds the active conformation of B-RAF

Good DMPK properties

Dual inhibition of p-MEK & p-ERK in vivo after oral dosing

Anti-tumor efficacy in five xenograft cell lines



MEK Inhibitor XL518

Specific inhibitor of MEK

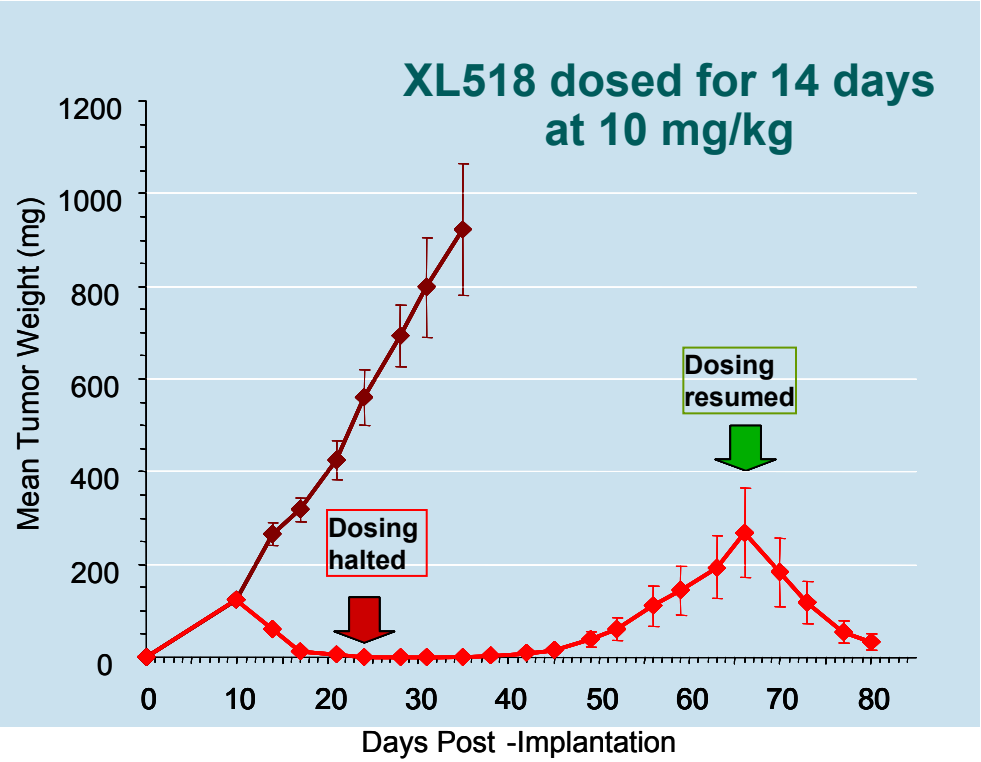
- IC_{50} = 0.4nM, highly selective
- Inhibits ERK phosphorylation in mutant BRAF and RAS cell lines
- Complete regression in some xenograft models

Attractive PK:

- Orally available
- No food effect in dogs
- No substantial brain exposure

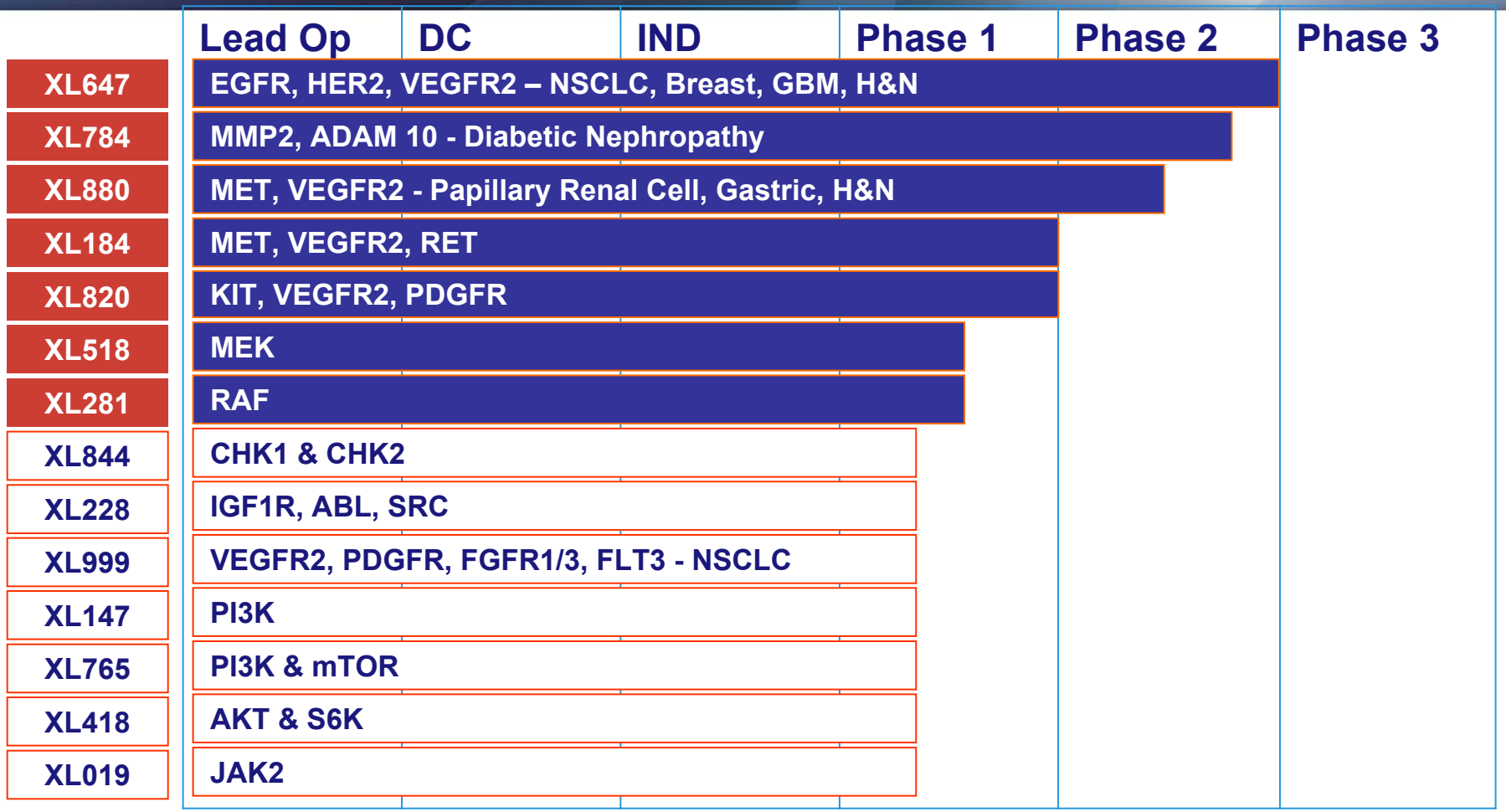
Clinical Status:

- **Phase 1 study ongoing**
- **Substantial in vivo target inhibition with paired tumor biopsies**



Colo205 model (BRaf V600E)

Exelixis Pipeline



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XL844

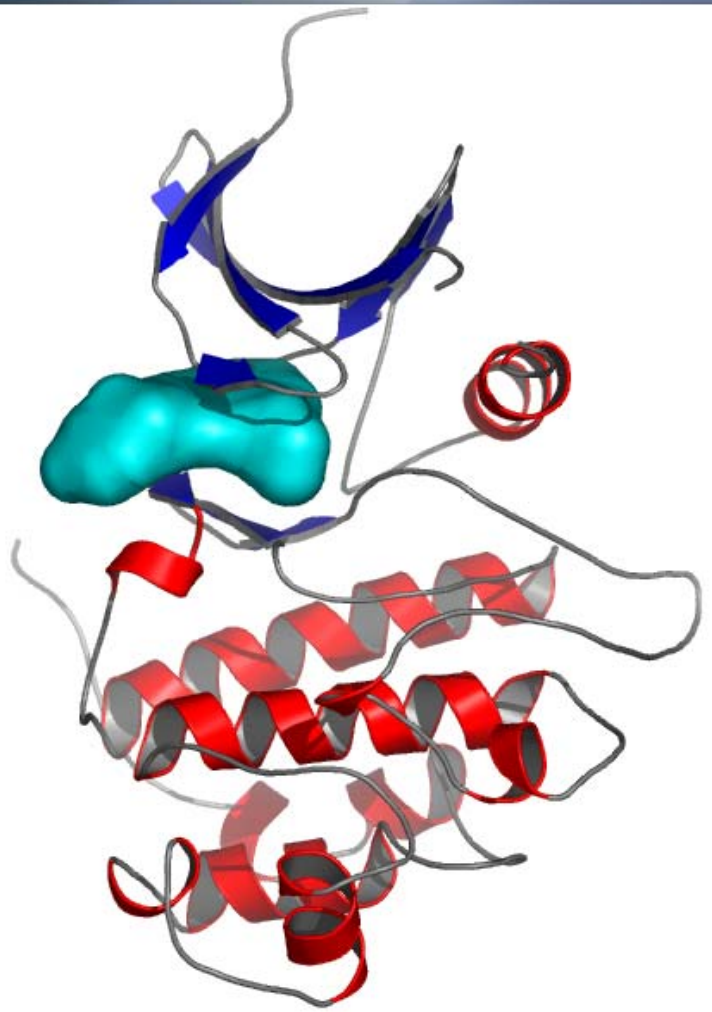
Potent inhibitor of Chk1 and Chk2 kinases

Abrogates DNA-damage checkpoints in vitro and in vivo

High aqueous solubility, metabolic stability, PK and tolerability

Synergistically potentiates activity of genotoxic agents

First specific Chk1/Chk2 inhibitor to advance into the clinic



Phase 1 solid tumors in combination with gemcitabine ongoing

Therapeutic Opportunity for PI3K Pathway Inhibitors

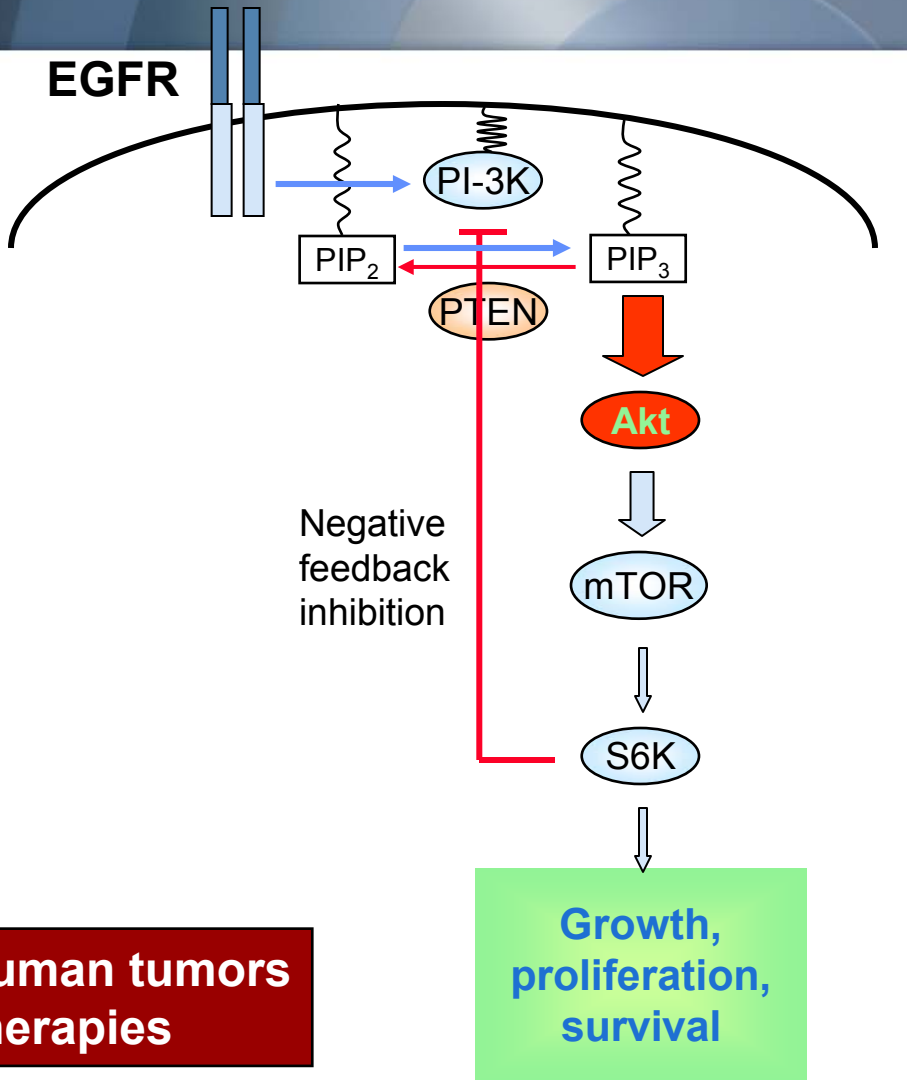
PI3K pathway abnormalities are common:

- PI3Kα mutation
- PTEN deletion, down-regulation
- RTK activation: growth factor expression, mutation, amplification
- RAS mutation

PI3K activation in resistance:

- Chemotherapy and radiotherapy – triggers activation of pathway, promotes cell survival
- Targeted therapy – failure to downregulate PI3K pathway signaling correlates with lack of response (e.g. rapa + EGFRi)

Pivotal pathway activated in majority of human tumors
Inadequately addressed by current therapies



XL147, a Potent and Selective PI3K Inhibitor

PI3Kα is a key component of the most frequently deregulated pathway in human tumors

- PI3Kα activating mutations/amplification
- PTEN down-regulation

Potent, selective PI3K inhibitor

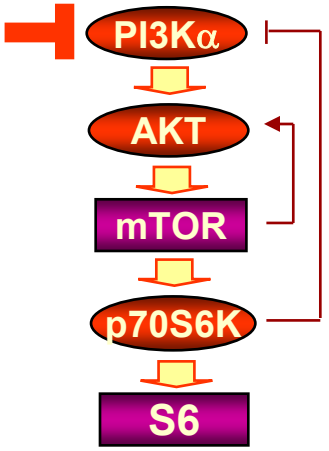
- No activity vs mTOR, or S/T/Y kinases
- Active against WT and mutant PI3Kα

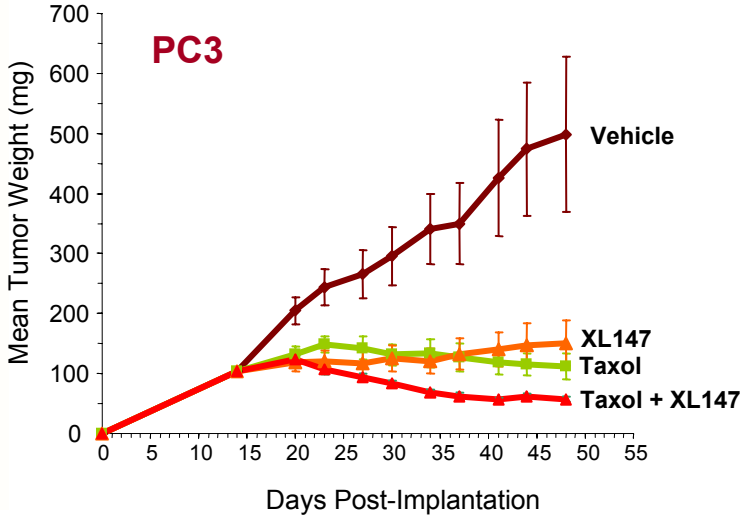
Excellent PK/PD properties

- Good oral bioavailability in 4 species
- Robust inhibition of AKT phosphorylation in multiple tumor models, >24 hr duration of action

Efficacious in xenograft tumor models

Single agent and combinations

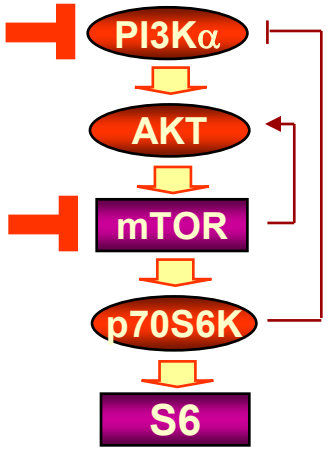




XL765, a Dual Inhibitor of PI3K and mTOR

mTOR is a pivotal node in the PI3K pathway

- Activated by PI3K dependent and independent mechanisms (amino acids, energy)
- Implicated in complex feedback regulation of the PI3K pathway



Dual PI3K/mTOR inhibitor

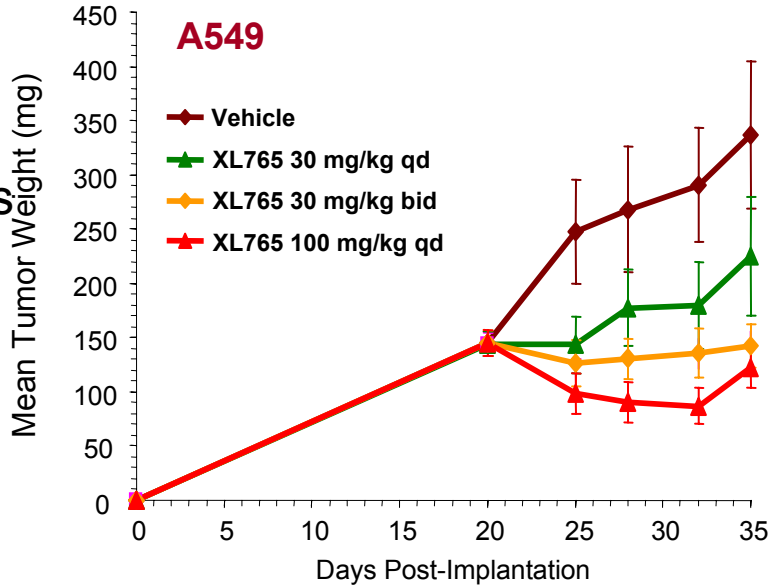
- WT and mutant PI3Kα
- No activity vs S/T/Y kinases

Excellent PD, good duration of action

- Robust inhibition of pAKT and pS6 in tumors
- 24 hr duration of action

Efficacious in xenograft tumor models

Single agent and combinations

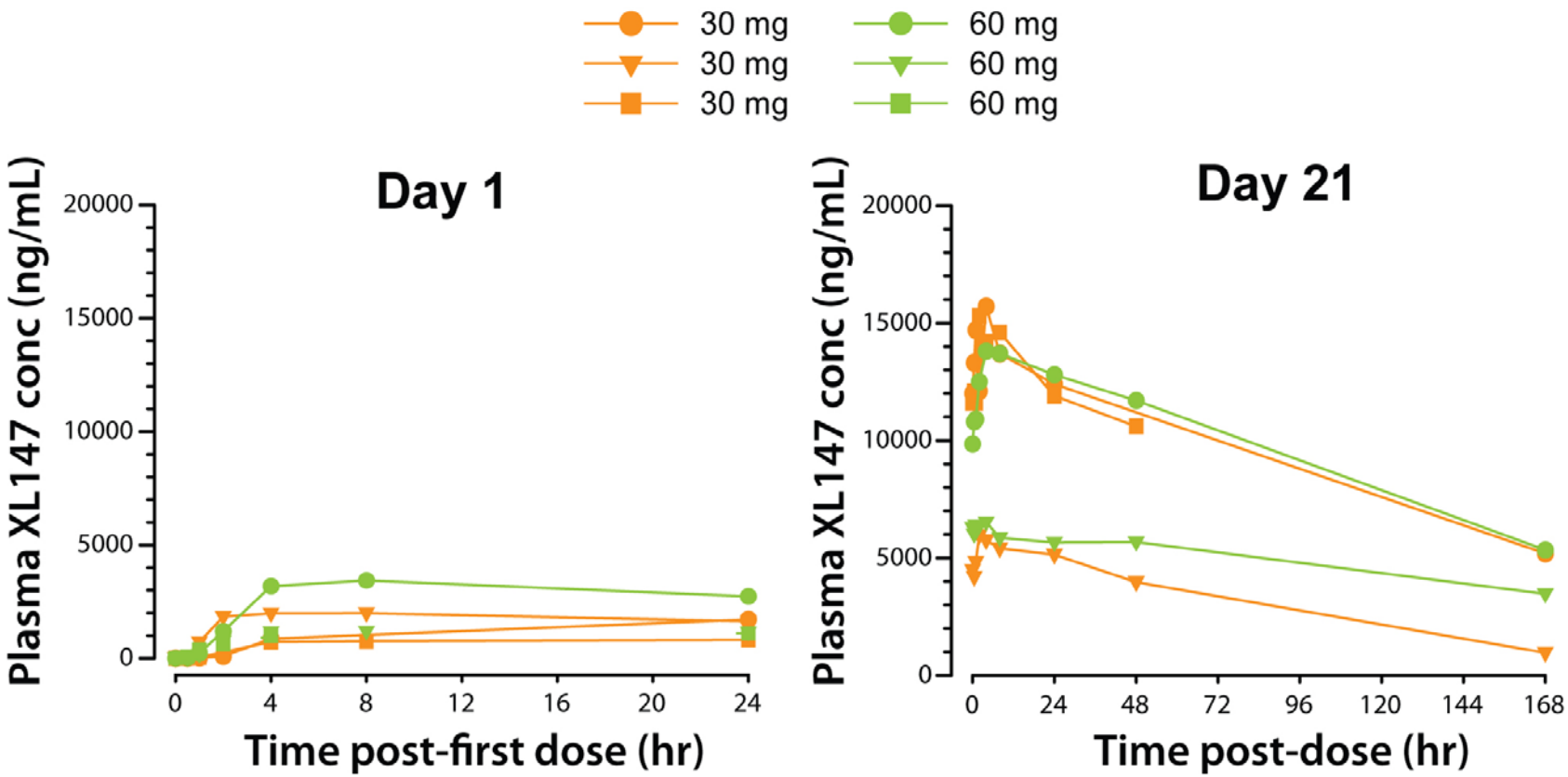


EXELIXIS™

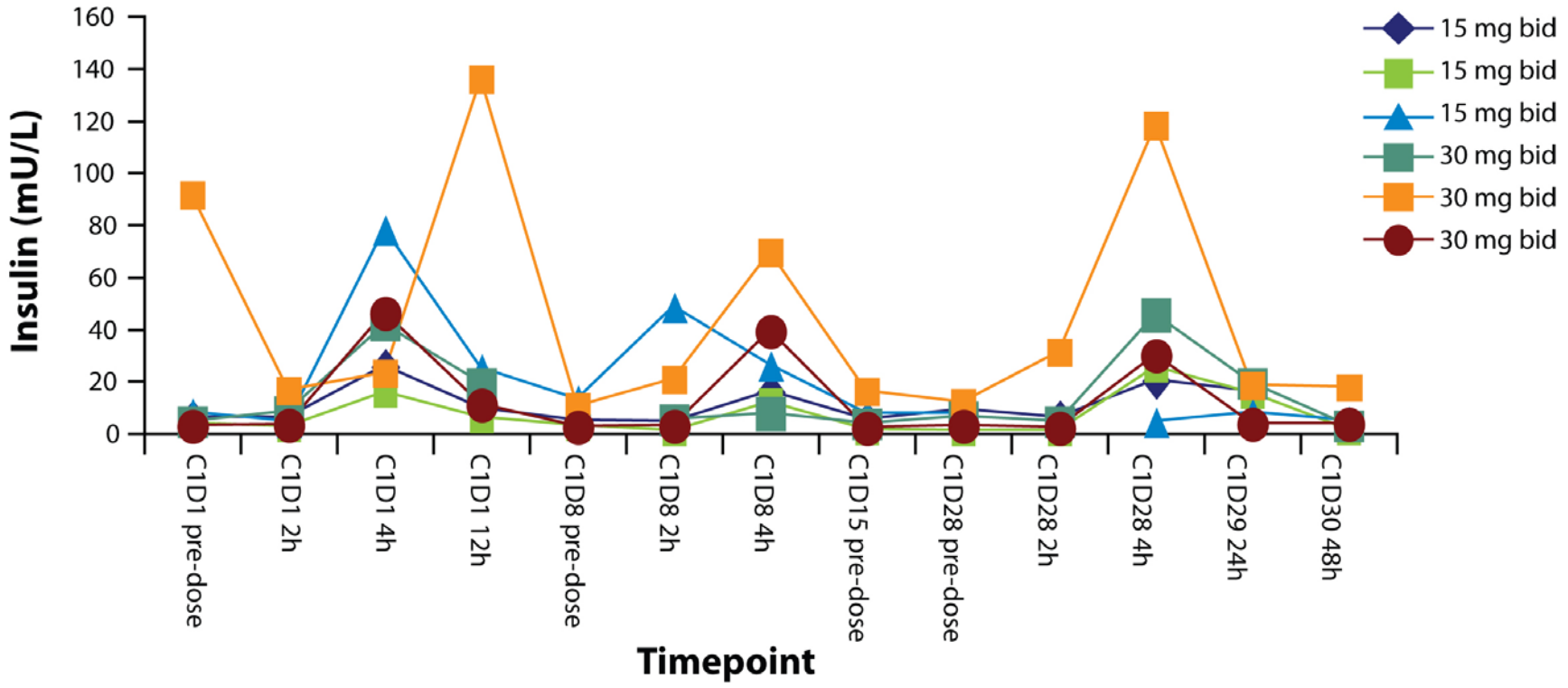
Targeting Aberrant PI3K Pathway Signaling With XL147 and XL765, Potent Inhibitors of PI3K and PI3K/mTOR

C205

Plasma Concentration–time Plots for XL147 Dosed at 30 and 60 mg

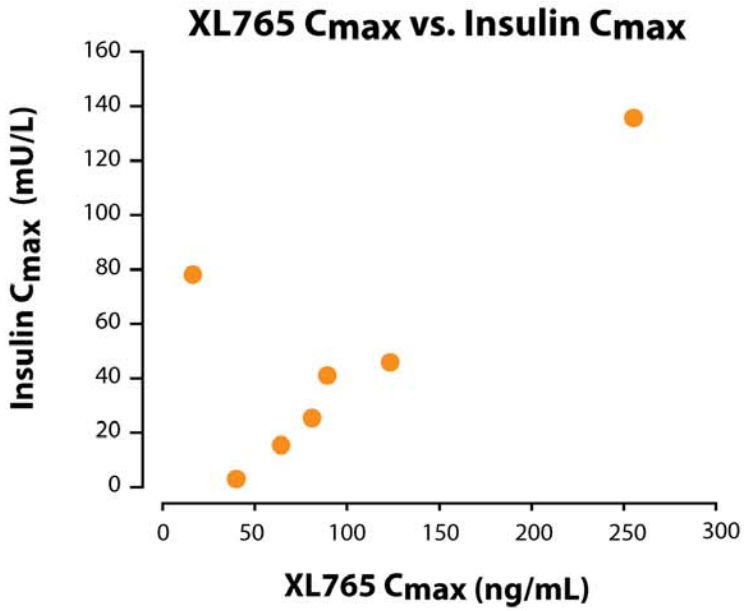


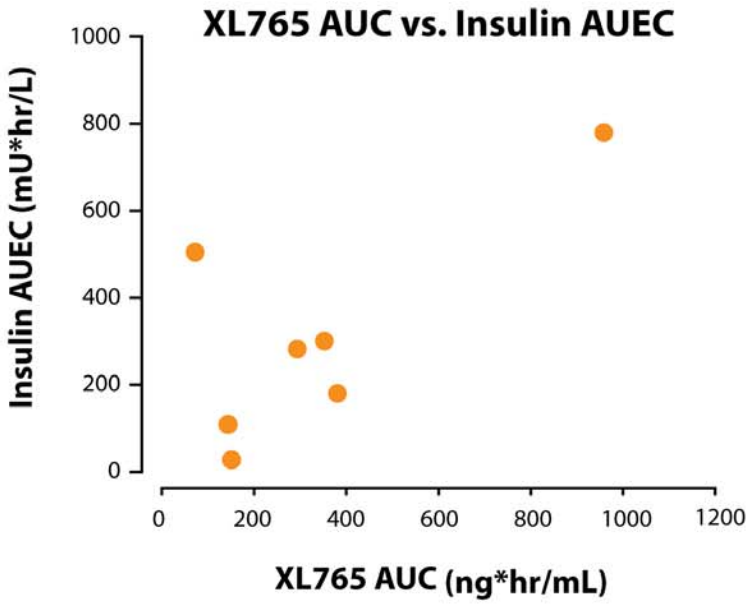
Plasma Concentrations of Insulin Following Twice-daily Dosing With XL765 at 15 and 30 mg BID



Plasma Insulin Plotted Against Selected XL765 Plasma Exposure Parameters

Patients from Cohorts 1 and 2 and a patient from Cohort 3 (n = 7 total)





PI3K/mTOR Inhibitors - Conclusions

XL147 and XL765 are potent, orally bioavailable inhibitors of PI3K and PI3K/mTOR, respectively

- Robust pharmacodynamic and efficacy data in preclinical models

PK from early cohorts in the Phase 1 clinical trials is encouraging for both compounds

- Plasma insulin levels tracked as a PD marker – early evidence of dose dependent effects for XL765

Dose escalation continues

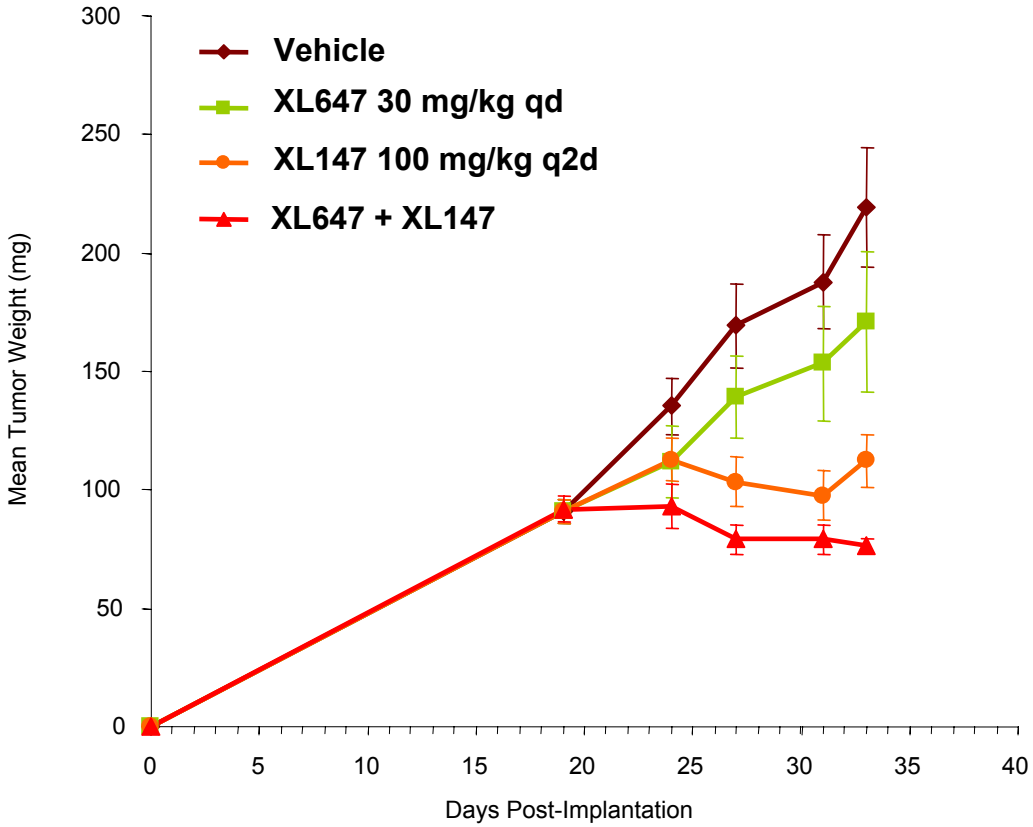
Exciting potential for combination with targeted therapies (XL880, XL184, XL647) and with chemotherapy

XL647/ XL147 Combination Data

Rational combination strategy for XL compounds

- -XL647 EGFR/HER2/VEGFR
- -Combination has superior efficacy in xenograft model

MCF7 breast carcinoma model
EGFR positive, p110α E545K



Broad Phase 2 Plan in Various Tumors

Single agent evaluation

Combination approaches

- XL647
- Erlotinib
- Other targeted agents including Sorafenib, Herceptin, Lapatinib…
- Chemotherapeutic agents

XL228 Summary

Potent dual inhibitor of IGF1R & SRC

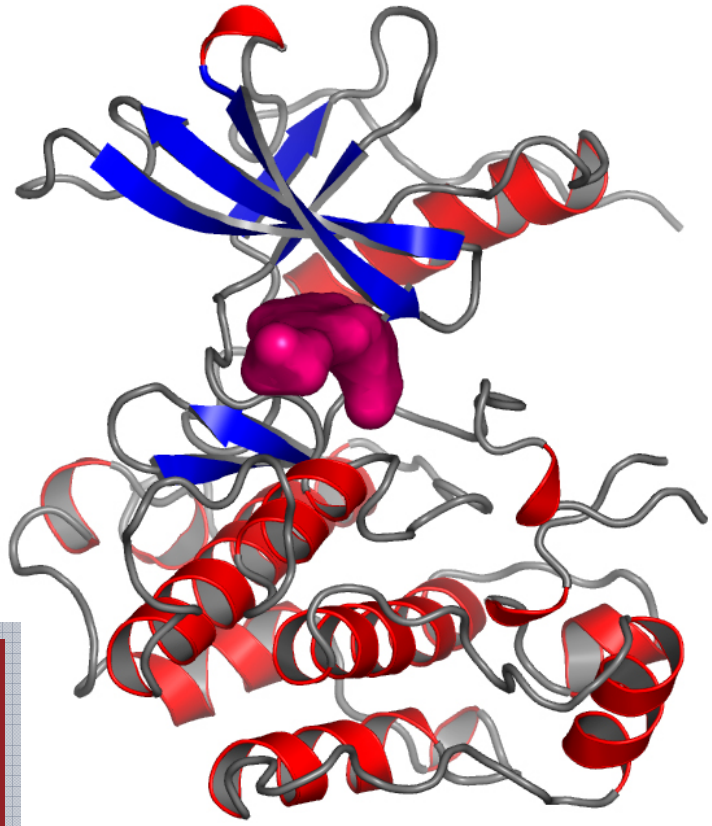
- IGF1R enhances cell growth and survival
- SRC cooperates with FAK to promote motility and invasion

Potent inhibitor of wt & mutant Abl

- Effective against Imatinib and Dasatinib resistance mutations

Significant in vivo PD effects on IGF1R and SRC pathways

- Efficacious in multiple xenograft models



Phase 1 studies ongoing in patients with

1. **CML (Imatinib and Dasatinib resistant)**
   - **Data presentation at ASH (12/10/07)**
2. **Advanced Malignancies**

XL019 – Potent & selective JAK2 Inhibitor

Excellent pharmaceutical properties

- Oral bioavailability; good preclinical PK
- Excellent CYP profile & low microsomal oxidation

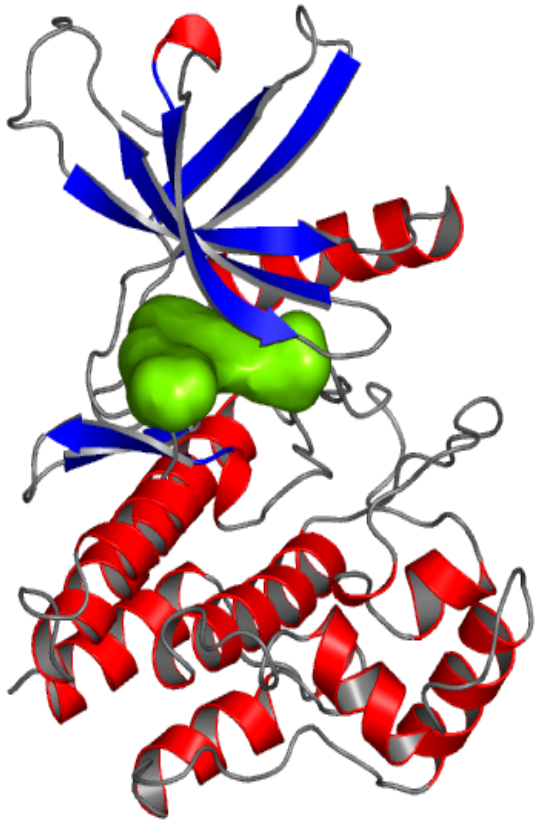
Demonstrated in vivo activity

- Downregulation of pSTATs in JAK2-driven model
- Inhibition of tumor growth in JAK2-driven model

1. Phase 1 dose escalation study in patients with myelofibrosis

- **Data presentation at ASH – 12/10/07**

2. Phase 1 dose escalation study in patients with polycythemia vera starting in Dec. 07



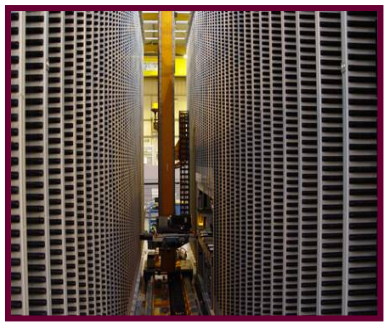
Discovery Platform

Peter Lamb, PhD, SVP & CSO

Discovery Platform

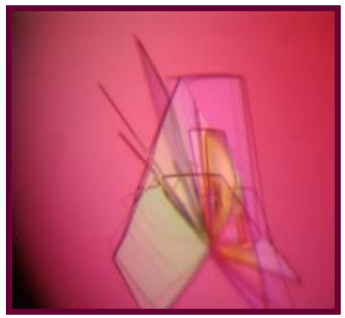
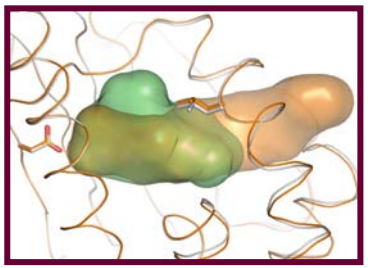
Exelixis has built a leading small molecule discovery platform capable of a sustained output of 3+ INDs / year

- Large (4.6M) compound library and a fast, flexible HTS platform




Potent Leads
Multiple Scaffolds
Multiple Target Classes

- Fully integrated structural biology capabilities

Detailed structural information guides lead optimization

Discovery Platform

Intensive parallel optimization of in vitro DMPK, cellular and in vivo parameters

In vivo target modulation (PD) is a key optimization parameter

Leads with appropriate PD advance into efficacy and tolerability studies

- Attrition during lead op due to target validation questions, SAR constraints, toxicity concerns

Robust HTS and lead validation pipeline and data driven decision making to focus resources on programs with best chance of success

Discovery Pipeline Overview

Three DCs identified in 2007

- Hedgehog Antagonist, XL139
- HSP90 inhibitor, XL888
- LXR partial agonist, XL652 (BMS-788)

On track for 3 additional DCs in 1H08

- CDC7 inhibitor (Oncology; EXEL-5413 is preDC)
- 11βHSD inhibitor (Metabolic disease; EXEL-5465 is preDC)
- Selective mTOR inhibitor (Oncology)

Robust early stage pipeline in oncology and metabolic disease

Expanded capabilities in metabolic disease, translational medicine and target ID / validation

Hedgehog Signaling and Cancer

Production and processing of HH ligands plays a key role in developmental processes

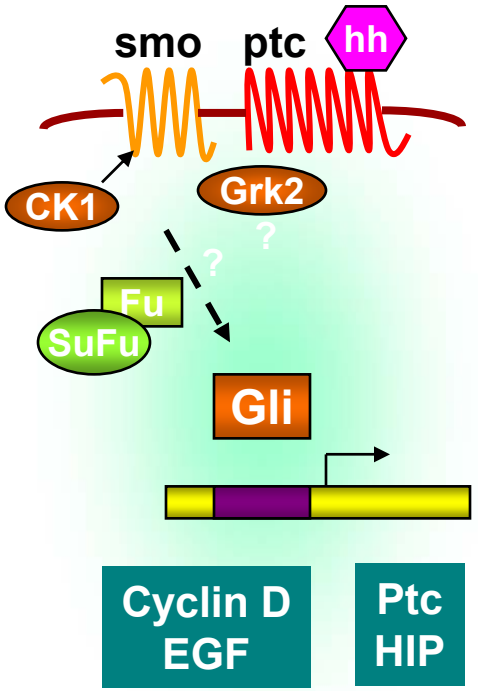
- SHh, IHh and DHh
- Renewal of tissue stem cells

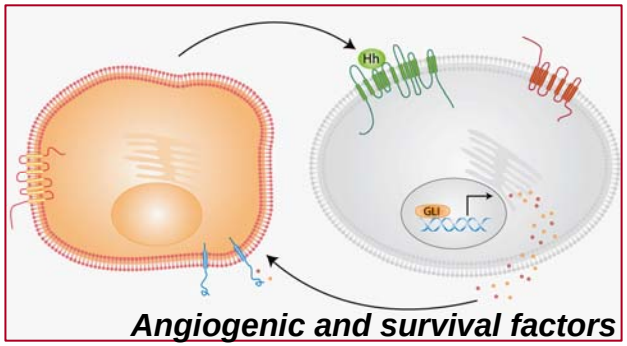
Inappropriate activation of the HH signaling pathway occurs in a variety of tumors

- Genetic activation (loss of repressors, activating mutations)
- Over-expression of ligands

HH ligand production can result in pathway activation in both tumor and stromal cells

- Activation in tumor cells promotes proliferation
- Activation in the stroma promotes production of angiogenic and survival factors





Hedgehog – Potential Indications

Tumors that harbor pathological mutations in pathway components

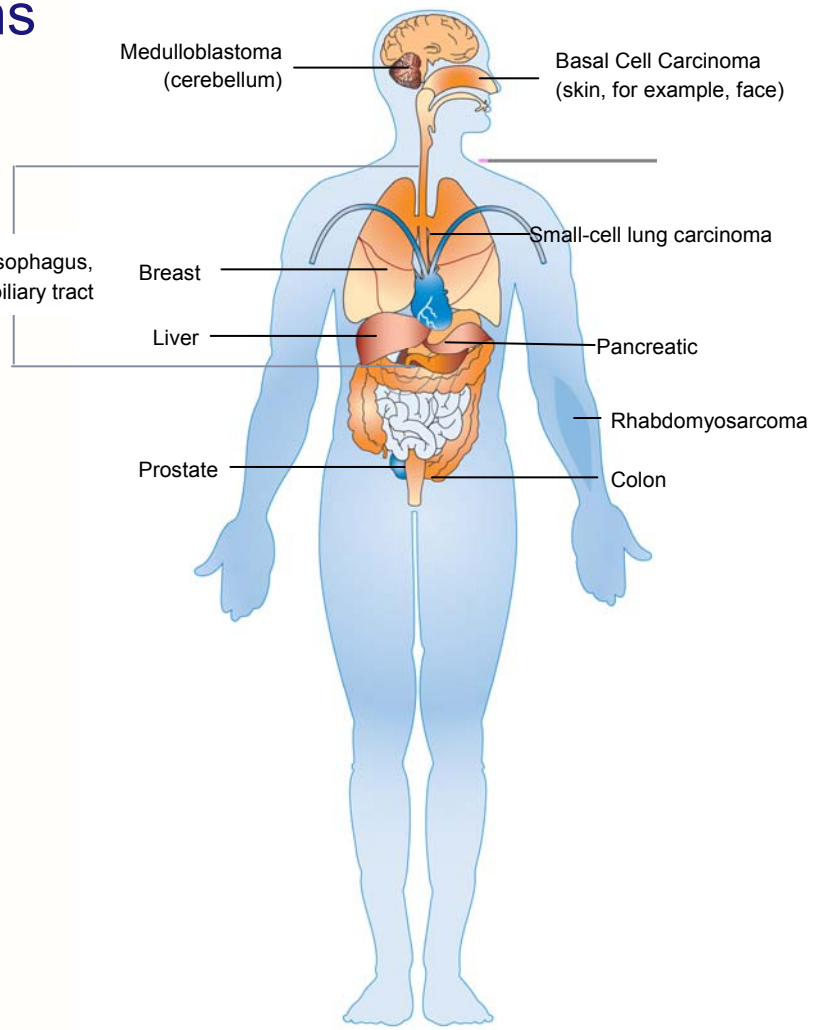
- Examples: BCC, medulloblastoma, rhabdomyosarcomas

Ligand-expressing tumors

- Autocrine/paracrine stimulation of the tumor/stroma
- Examples: Pancreatic, prostate, SCLC, digestive tract tumors

Cancer stem cells

- Examples: Glioblastomas, pancreatic, breast tumors and MM
- Strong rationale for combination with chemo and radio therapy



EXEL-1139 Overview

EXEL-1139 is a novel, potent antagonist of HH signaling

- Binds to smoothened

	3T3 Gli Reporter	Gli1	Smo S533N	Smo W535L
IC50 nM	6	7	27	35

BCC activating mutations

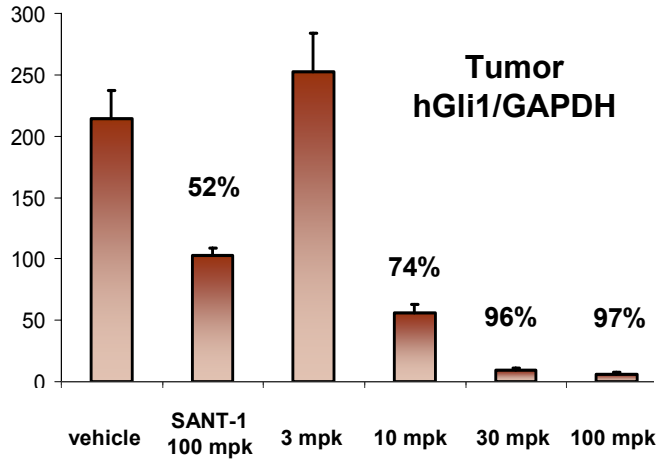
EXEL-1139 has excellent PK and PD properties

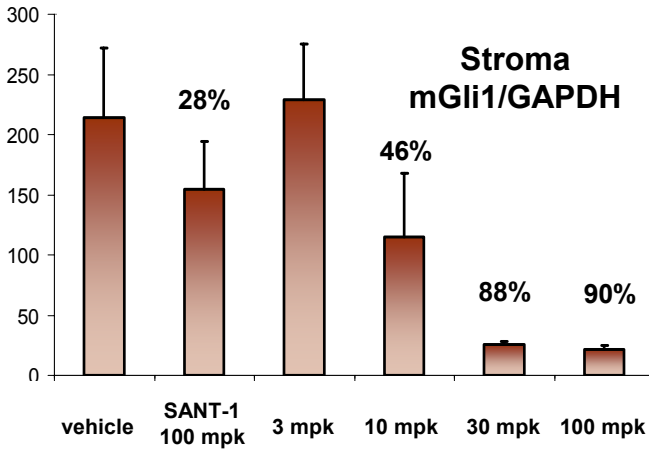
- Excellent oral bioavailability
- Minimal CYP interactions
- Profound downregulation of HH signaling in tumors and stroma

EXEL-1139 Inhibits Tumor and Stromal HH Pathway Activation in a Medulloblastoma Model

EXEL-1139 is a potent inhibitor of HH pathway activation in a medulloblastoma model expressing high levels of SHh

- SHh drives pathway activation in tumor and stroma

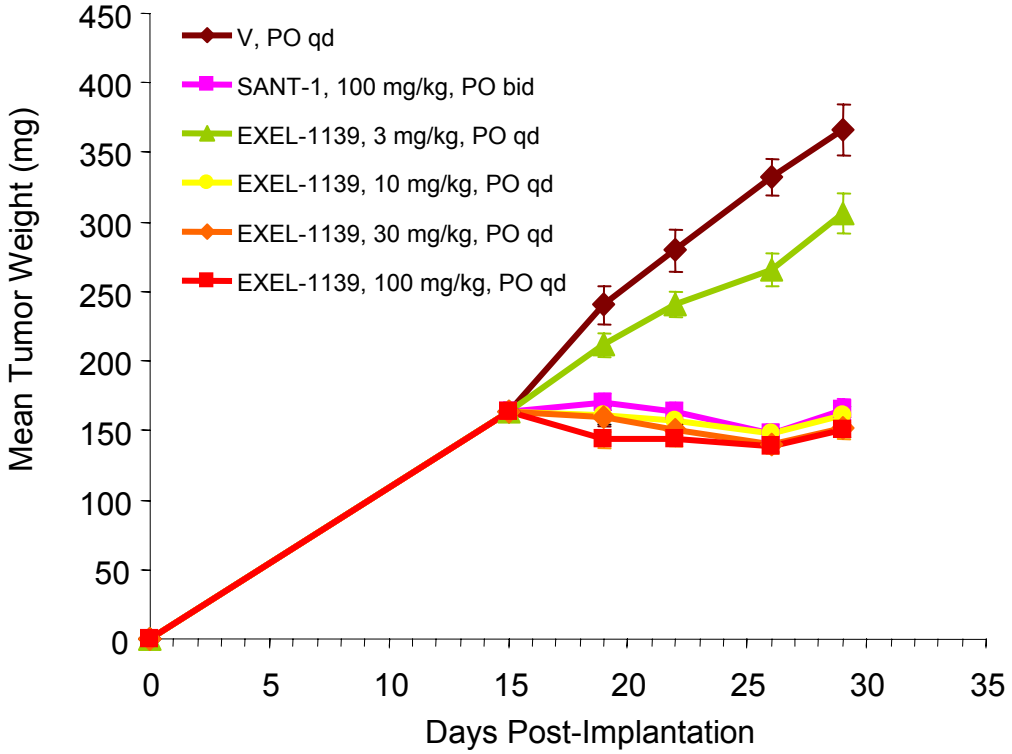




EXEL-1139 Efficacy

Highly efficacious in a human medulloblastoma model

- Well tolerated at all doses



EXEL-1139 (XL139) Summary

Novel, potent inhibitor of HH signaling

- Excellent preclinical PK and PD properties

Active in xenograft models

- Impact on tumor and stromal cells

Strong rationale for combination with chemo and radio therapy

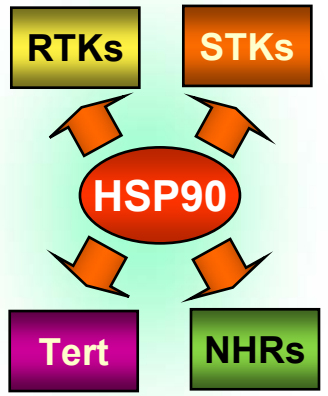
- Exploit potential to inhibit survival of chemo and radio resistant tumor stem cell component

XL139 scheduled to enter clinical trials 2Q08

EXEL-4888, Novel HSP90 Inhibitor

Chaperone that stabilizes multiple proteins deregulated in human tumors

- ErbB2, AKT, Raf, Src, CDK4, AR
- Key role in stabilizing mutant proteins
- Overexpressed in human tumors



First generation inhibitors in clinical development

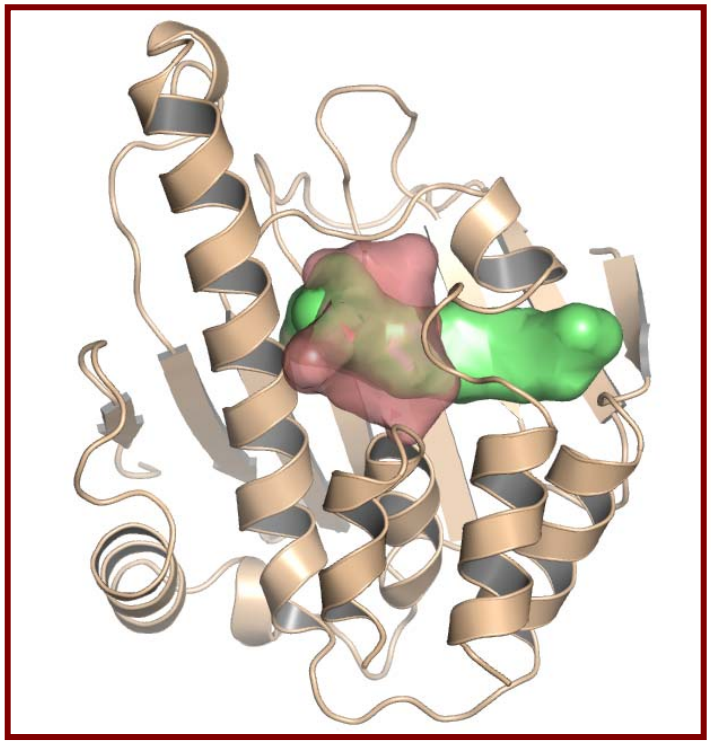
- Geldanamycin analogues, poor DMPK properties
- Inconsistent tumor exposure due to metabolism
- Hepatotoxicity – intermittent dosing schedules limit efficacy

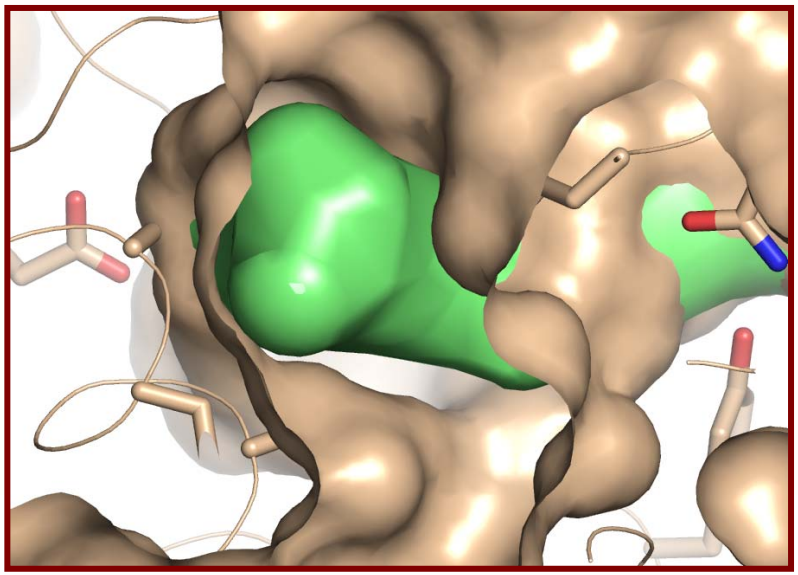
Opportunity for 2nd generation compounds with improved DMPK

EXEL-4888, Novel HSP90 Inhibitor

EXEL-4888 is a highly novel scaffold and accesses interactions not described by other HSP90 inhibitors to date

- **EXEL-4888 (green) has distinct binding mode from clinical competitor 17-AAG (pink)**

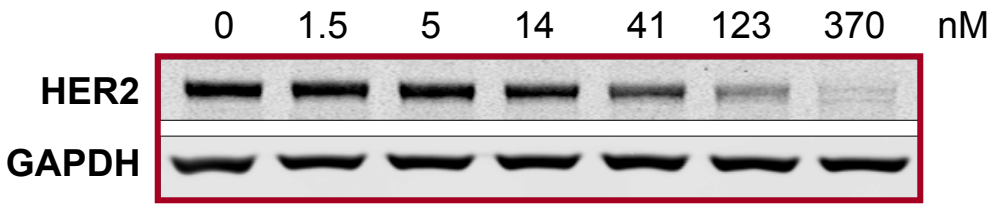




EXEL-4888 In Vitro Potency Profile

EXEL-4888 has equivalent or superior potency to 17-DMAG in client degradation and proliferation assays

- low – sub nM anti-proliferative activity

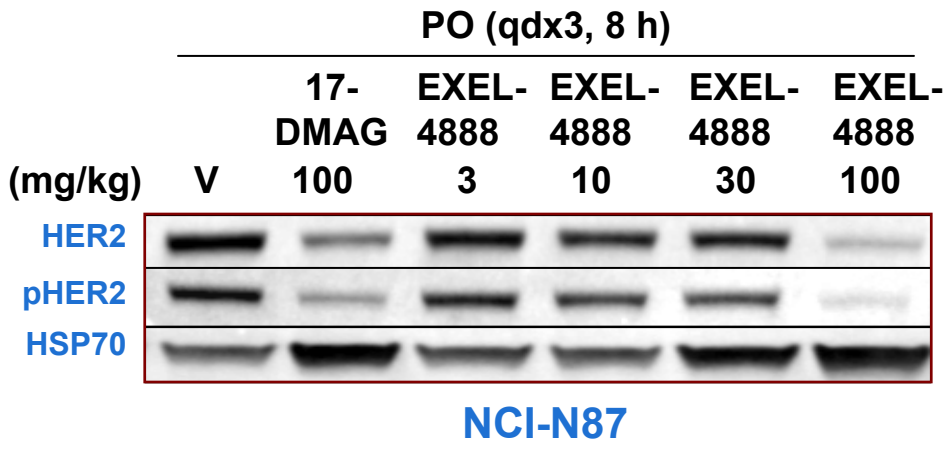


	Proliferation IC_{50} nM				
	BT-474	Colo-205	HN5	NCI-N87	MCF7
17-DMAG	0.3	15	10	19	8
4888	0.1	12	6	28	15

EXEL-4888 Down-regulates Multiple HSP90 Clients In Vivo

Oral administration of EXEL-4888 results in HSP90 client protein reduction in 3 xenograft models

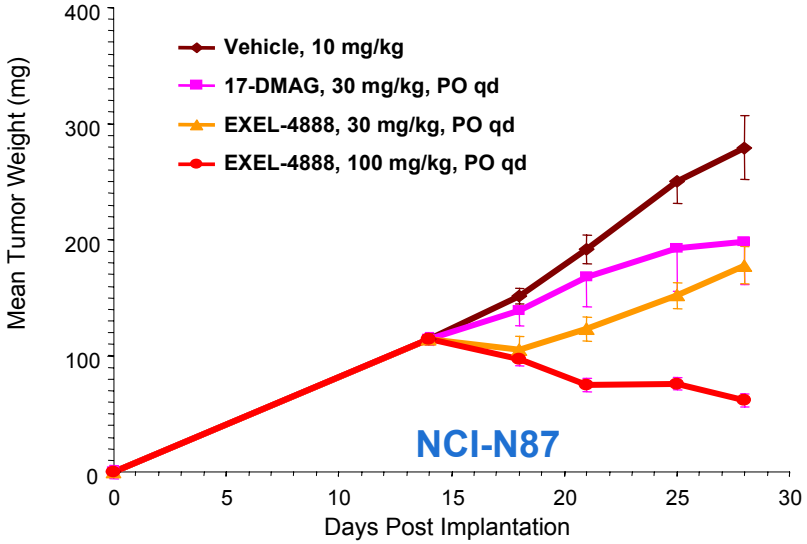
- HSP70 upregulation also observed

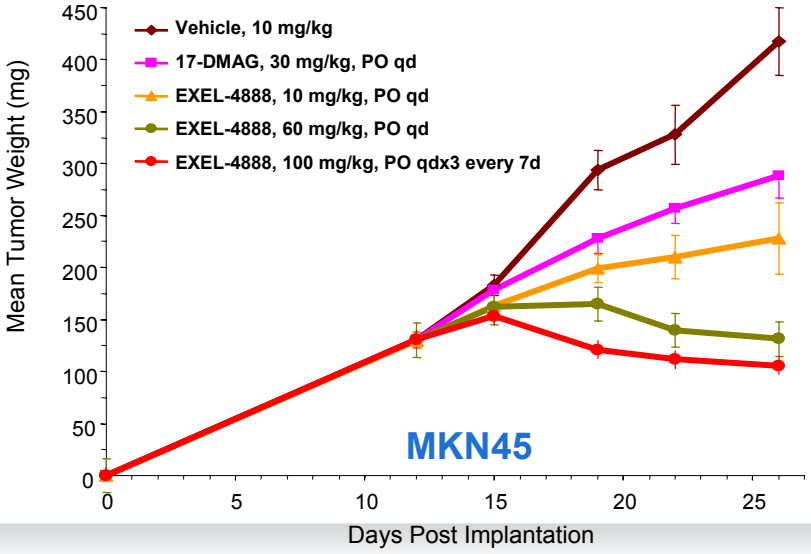


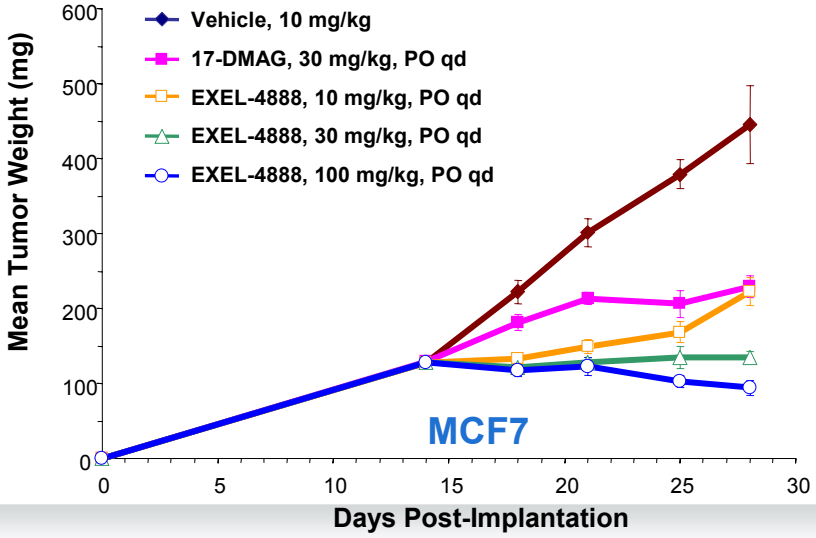
EXEL-4888 is Efficacious in Multiple Xenograft Models

EXEL-4888 demonstrates excellent efficacy in MCF, NCI-N87 and MKN45 xenograft models

- **Superior efficacy vs 17-DMAG at MTD**







EXEL-4888 (XL888), Novel HSP90 Inhibitor

EXEL-4888 is a novel, potent HSP90 inhibitor

- Highly active in cellular client protein degradation and proliferation assays
- Excellent in vivo pharmacodynamic activity in multiple tumor models

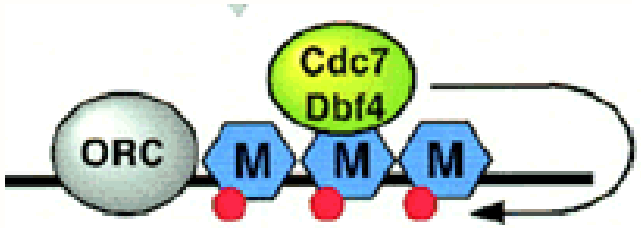
Advantages over 17-AAG and derivatives

- Good oral bioavailability
- Activity independent of quinone oxidoreductase (diaphorase) activity
- Improved tolerability of EXEL-4888 results in superior preclinical efficacy at MTD vs 17-DMAG
  - Potential to allow for more continuous dosing schedules - intermittent schedules required for geldanamycin analogues likely to limit efficacy

CDC7 Rationale

Serine/Threonine kinase that regulates initiation of DNA replication and progression through S-phase

- Phosphorylates and regulates MCM proteins (putative DNA helicase)
- Kinase activity is required for firing of origin of replication (ORC) complexes



CDC7 is over-expressed in many tumor types

- Potential for broad spectrum activity

Inhibition of CDC7 in tumor cells is associated with aberrant S-phase progression followed by apoptosis

- p53 independent
- Normal cells enter reversible arrest

Novel approach to inhibiting tumor cell proliferation with potential for reduced side effects

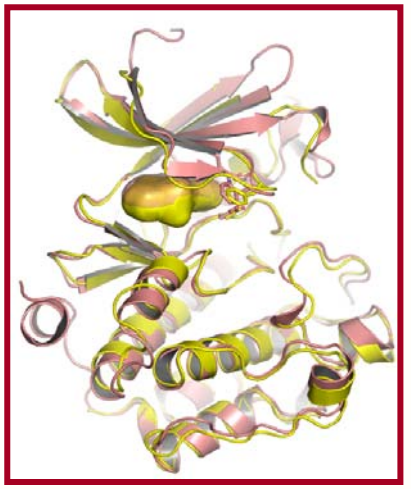
EXEL-5413: Selective CDC7 Inhibitor

EXEL-5413 is a novel, potent and selective inhibitor of CDC7

- CDC7 biochemical IC50 4 nM

EXEL-5413 is highly active in a cell-based assay monitoring CDC7 dependent phosphorylation of MCM2

- IC50 191 nM



Attractive in vitro DMPK profile and in vivo PK

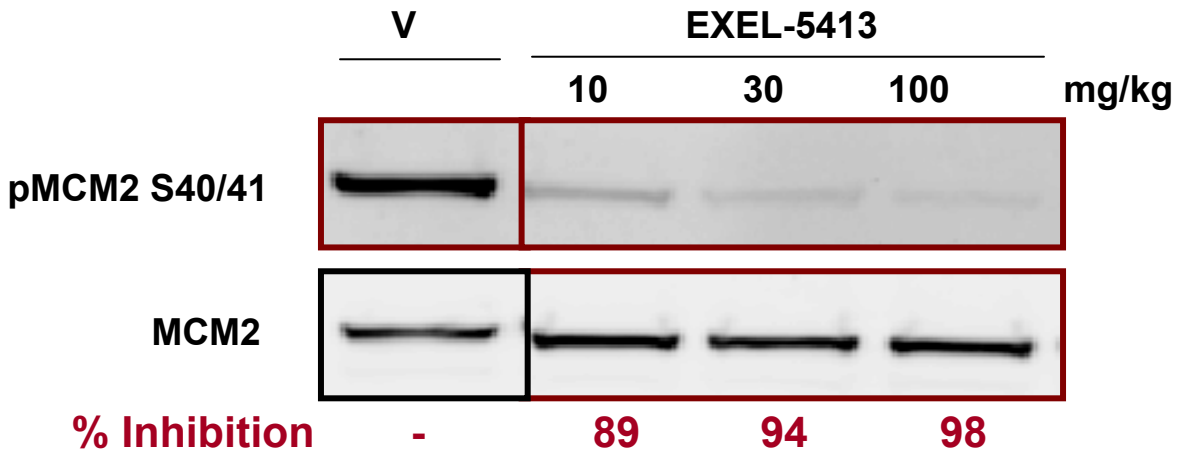
- Limited interaction with CYPs
- No inhibition of hERG
- High exposure following oral dosing

Encouraging activity and tolerability in preclinical tumor xenograft models

EXEL-5413: Selective CDC7 Inhibitor

EXEL-5413 is highly active against CDC7-specific PD readout in vivo

- phosphorylation of MCM2 by CDC7 is significantly reduced in a xenograft tumor model following a single oral dose of EXEL-5413

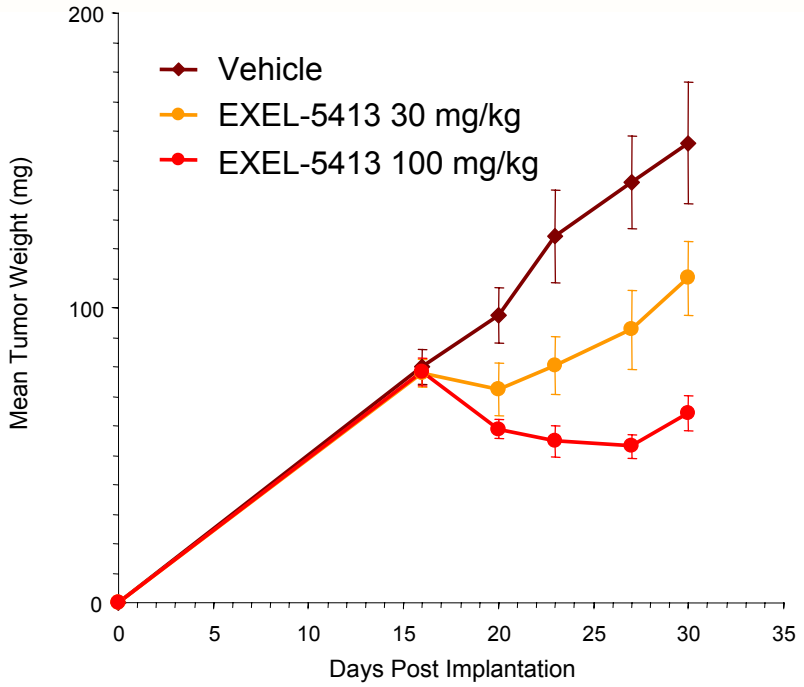


A431 human tumor xenograft

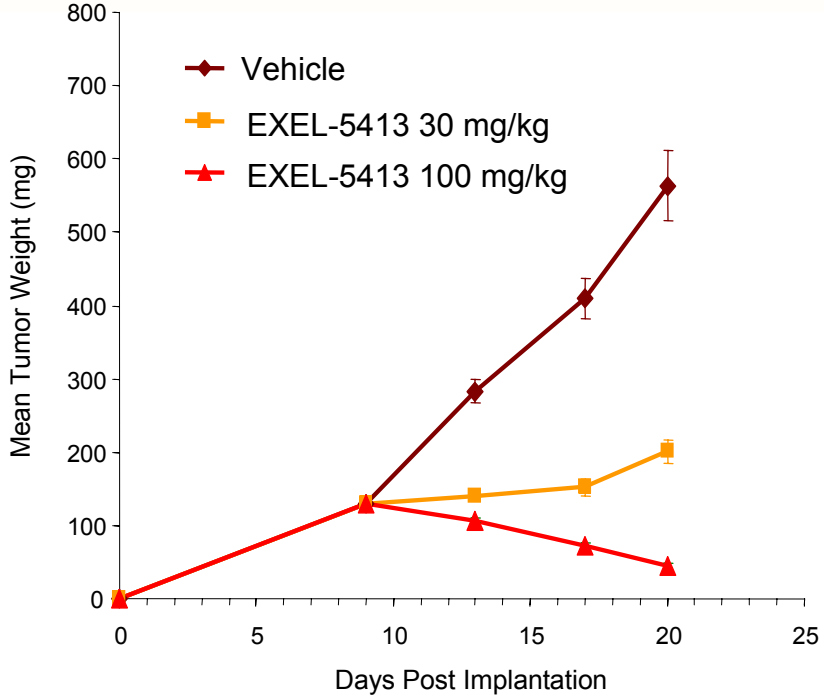
EXEL-5413 Efficacy in Xenograft Tumor Models

EXEL-5413 shows significant TGI in tumor xenograft models

- A549 and A431 models
- Well tolerated at 30 and 100 mg/kg



A549



Colo205

EXEL-5413 Summary

EXEL-5413 is a selective inhibitor of CDC7

- Novel scaffold
- Attractive in vitro ADME profile
- Excellent oral bioavailability

Robust PD activity vs CDC7 readout in tumors in vivo

Encouraging efficacy in preclinical xenograft models

- Well tolerated

EXEL-5413 may also be very potent in combination with genotoxic chemotherapy

- Documented role of CDC7 in protection from genotoxic stress

EXEL-5413 is a preDC – DC declaration 1Q08

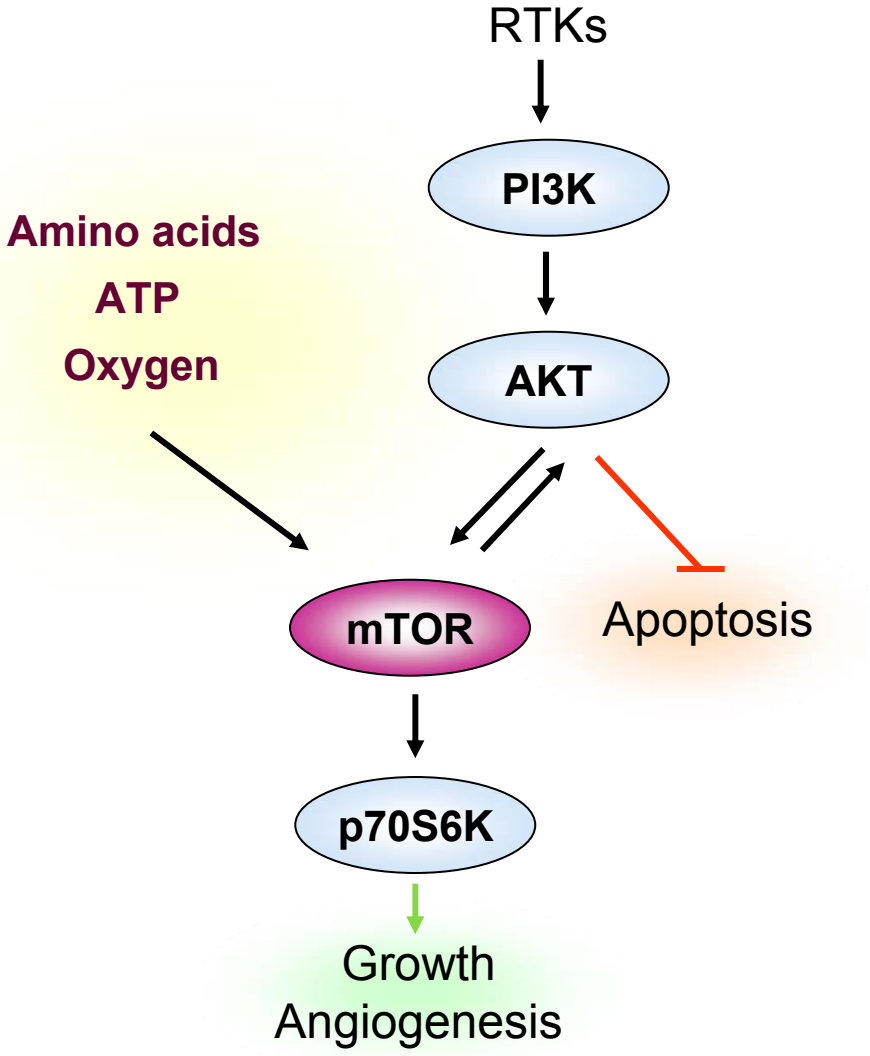
Selective mTOR Inhibitor

mTOR is an S/T kinase that is a pivotal node in the PI3K signaling pathway

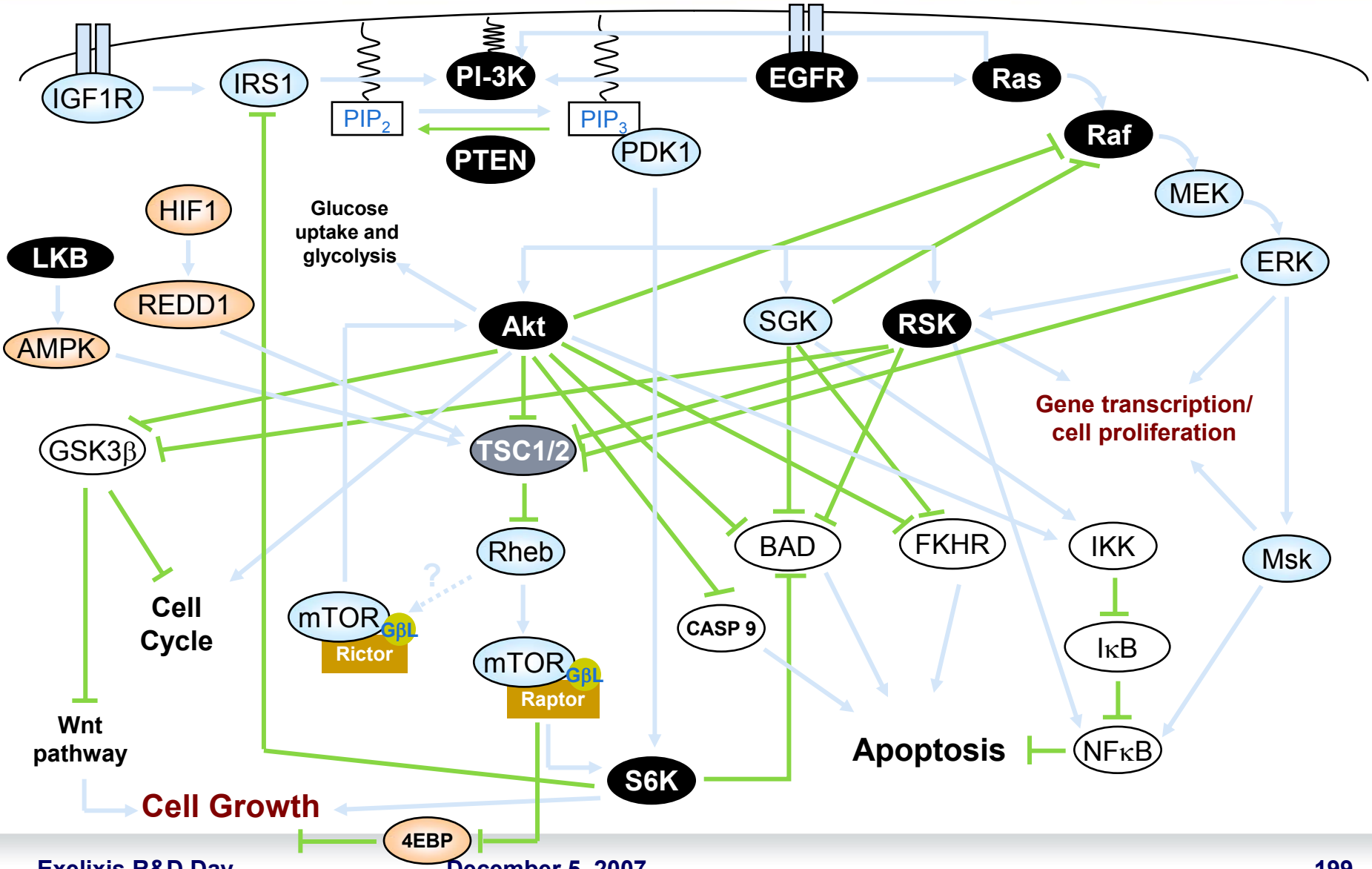
- Promotes growth
- Promotes angiogenesis
- Inhibits apoptosis

mTOR plays a highly conserved role in integrating cellular growth signals with cellular metabolic status

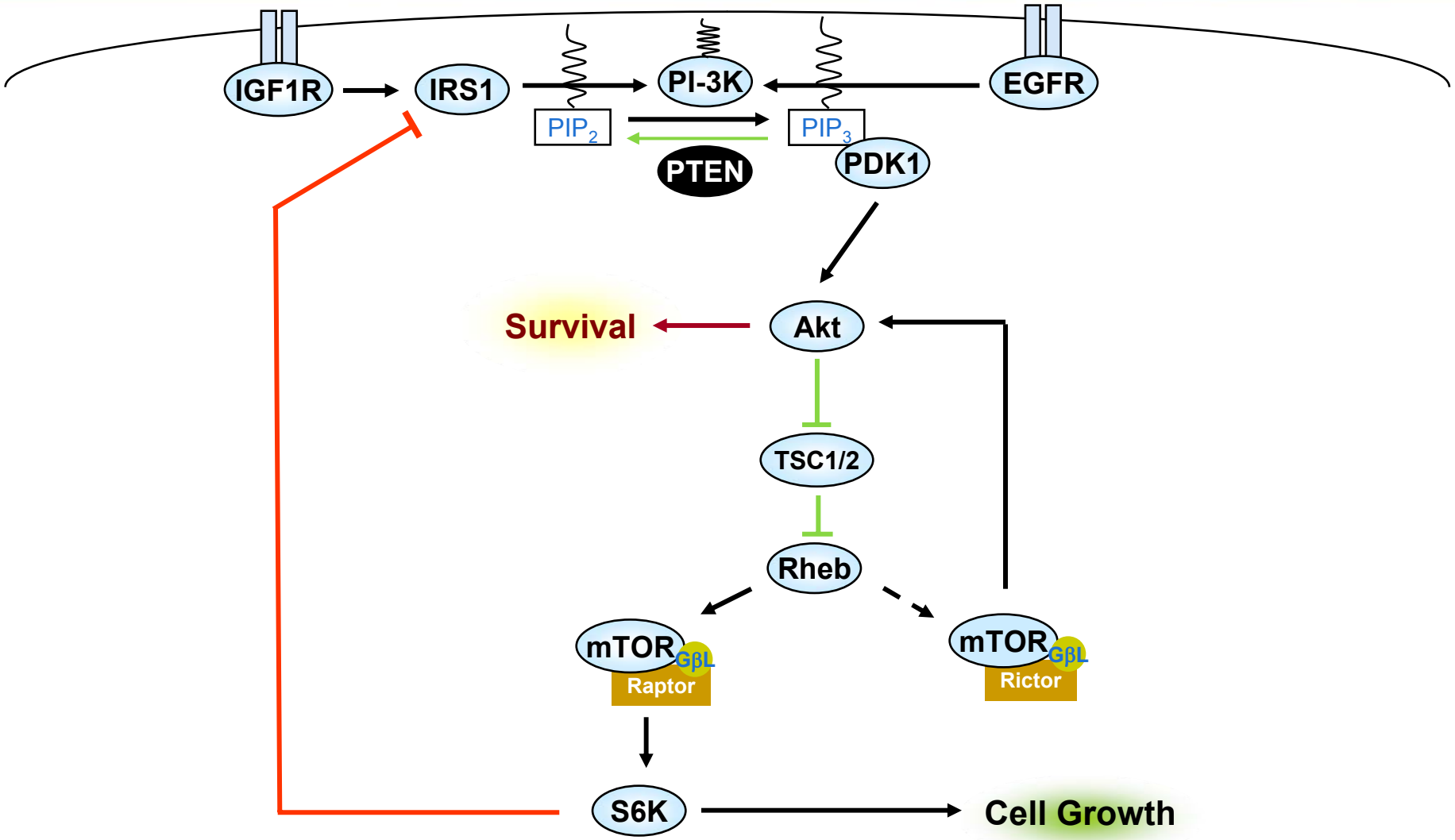
- Cellular energy levels
- Amino acid availability
- Oxygen levels



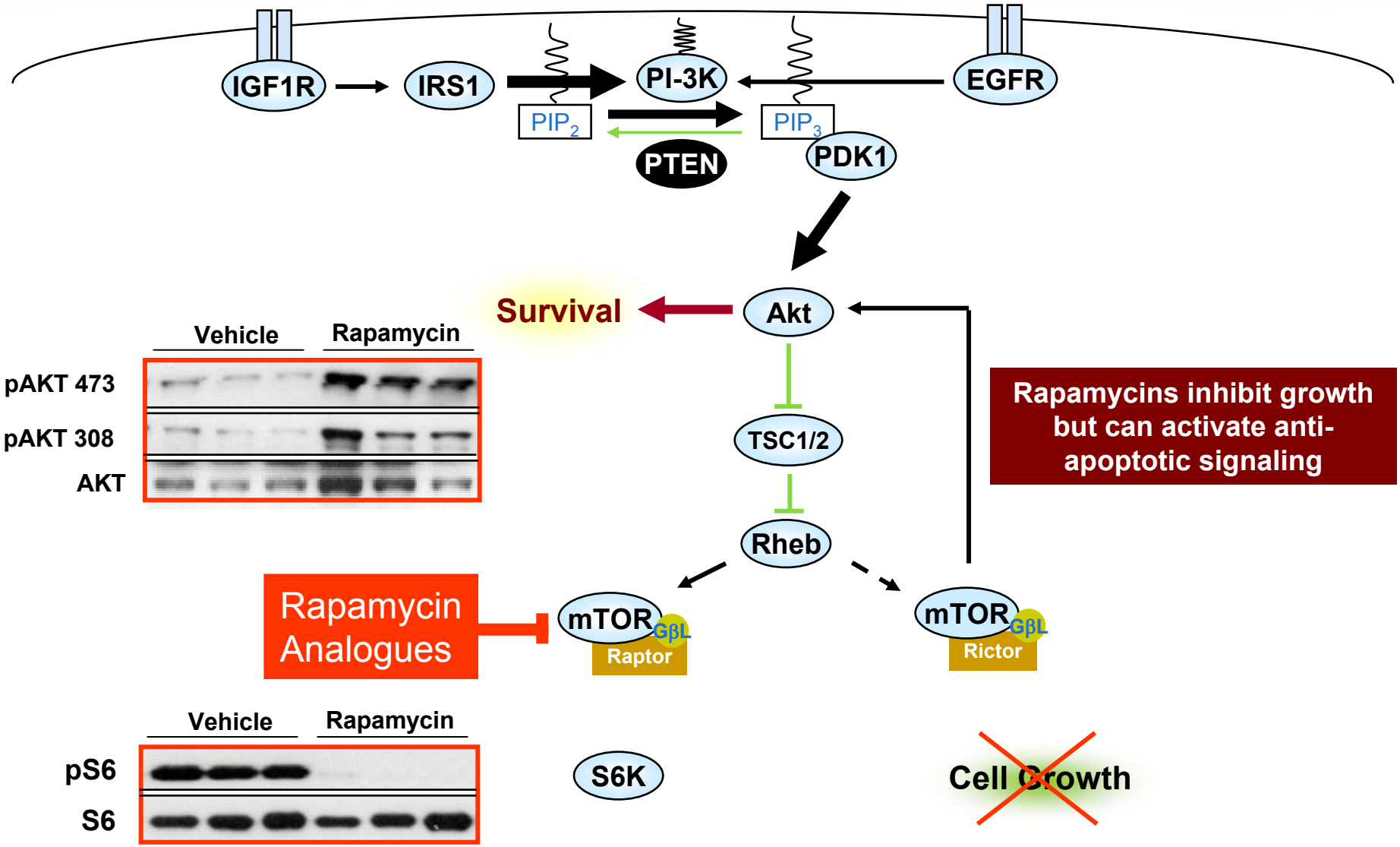
Selective mTOR Inhibitor



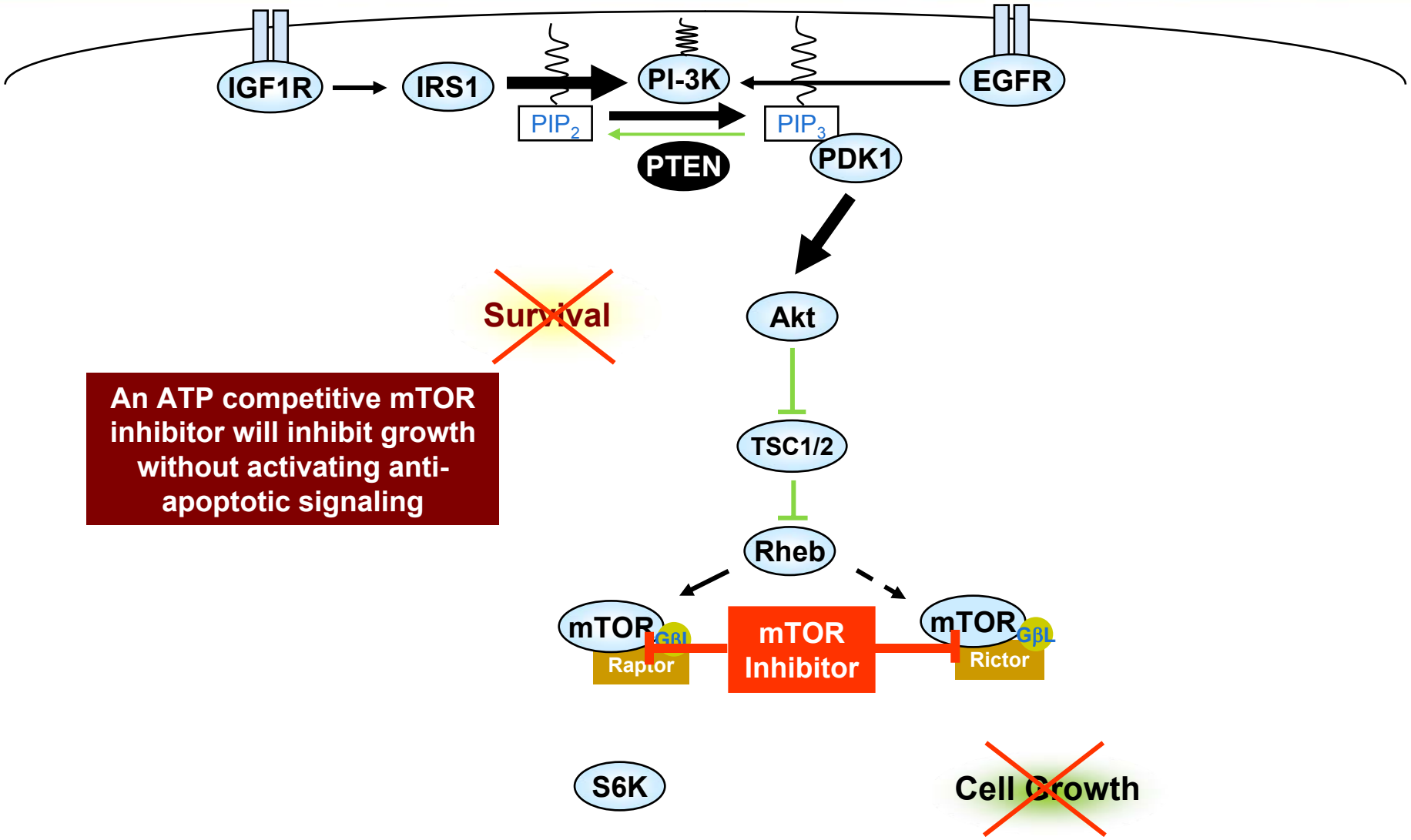
Selective mTOR Inhibitor



Selective mTOR Inhibitor



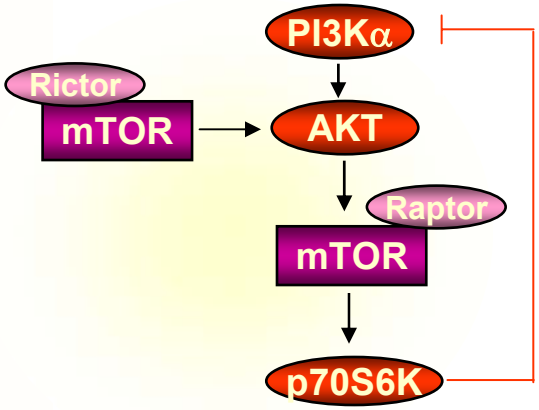
Selective mTOR Inhibitor



Selective mTOR Inhibitor

An ATP competitive mTOR inhibitor will inhibit both mTOR / Raptor and mTOR / Rictor

- Inhibit p70S6K and AKT activity
- Distinct from and superior to rapamycins



HTS completed using recombinant mTOR

- Focus on a single scaffold – ATP competitive
- Multiple additional scaffolds identified

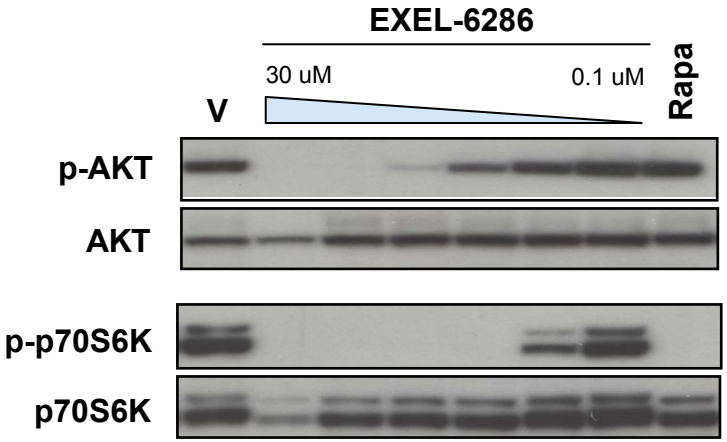
SAR has yielded highly potent selective leads

- <1 nM vs mTOR / Rictor complex
- <100 nM in cellular mechanistic assays (pS6)
- >100x selective vs PI3K

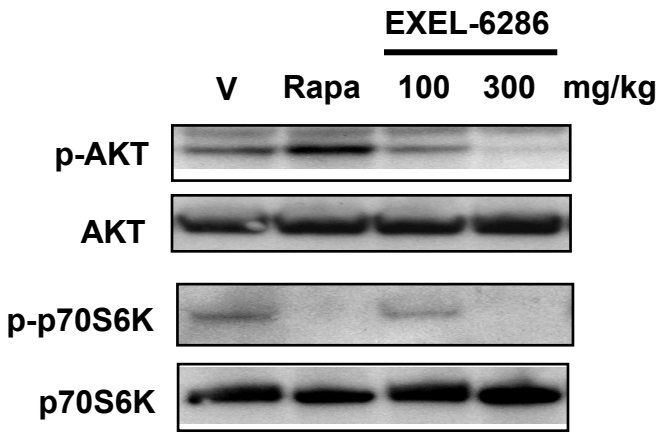
Selective mTOR Inhibitor

Balanced inhibition of both p-p70S6K and pAKT S473 in PC3 cells

- In vitro and in vivo



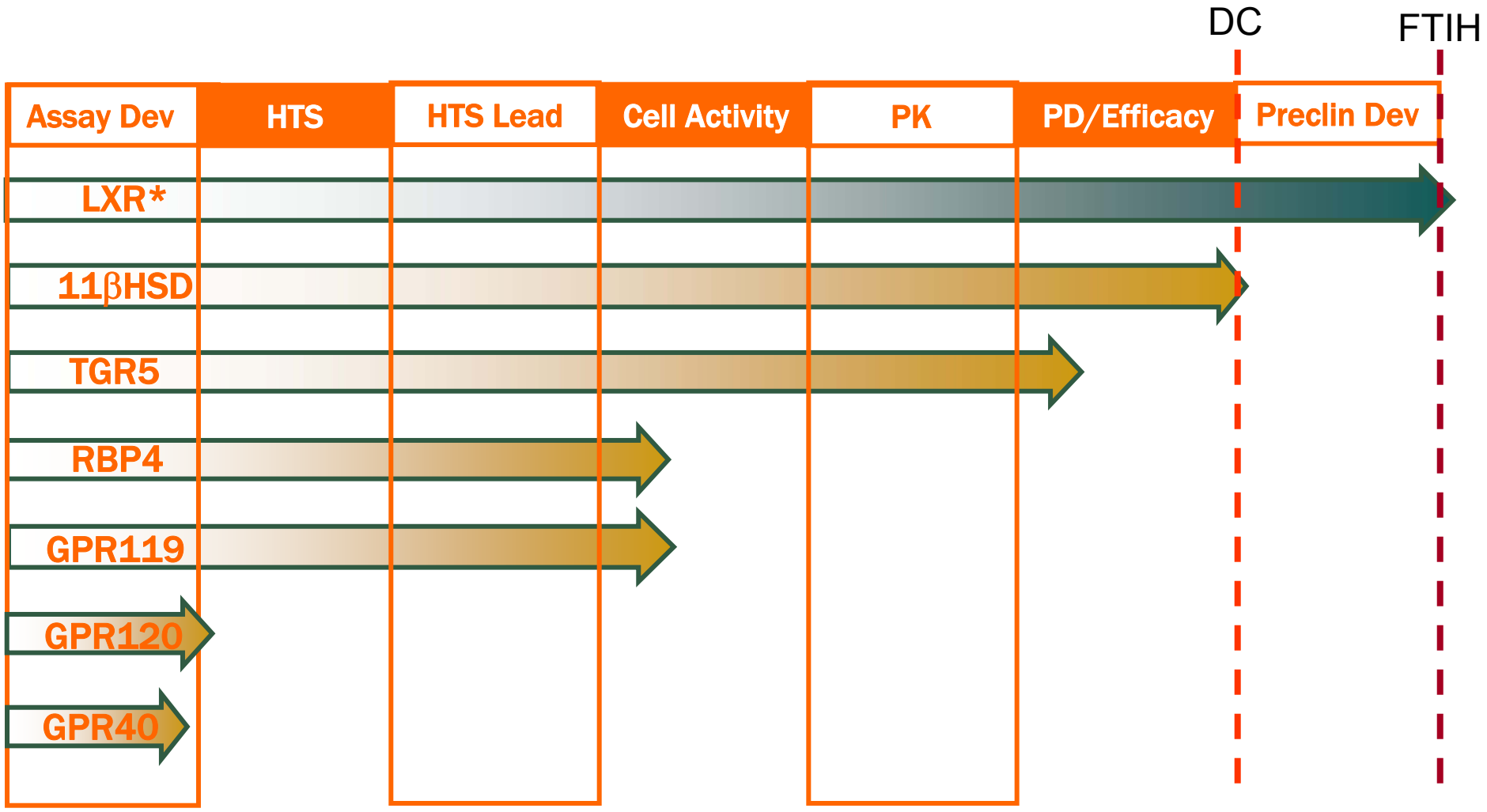
In Vitro – PC3 Cells



In Vivo –PC3 Tumor xenografts

Advanced leads with in vivo PD activity – DC selection 1Q08

Discovery Pipeline – Metabolic Disease

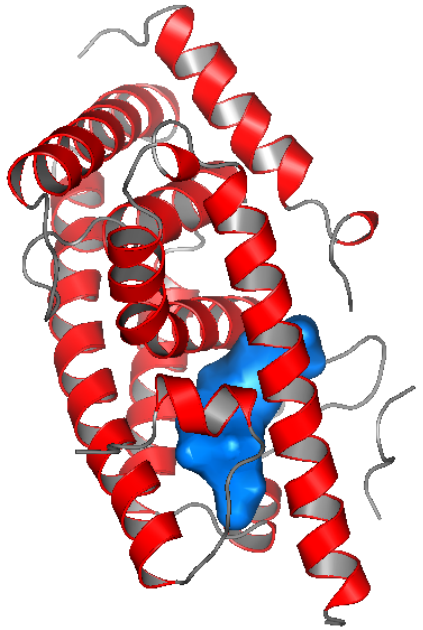


* BMS collaboration target

LXR and Atherosclerosis

Atherosclerosis

- Primary mechanism of CAD and number one leading killer in the US
- Unmet need for therapy that effects disease pathology
  - Macrophage inflammation
  - Plaque stability and regression



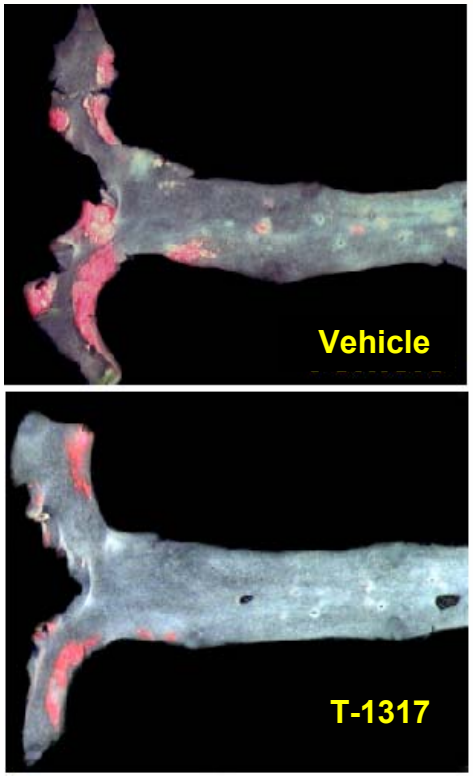
LXR Agonists

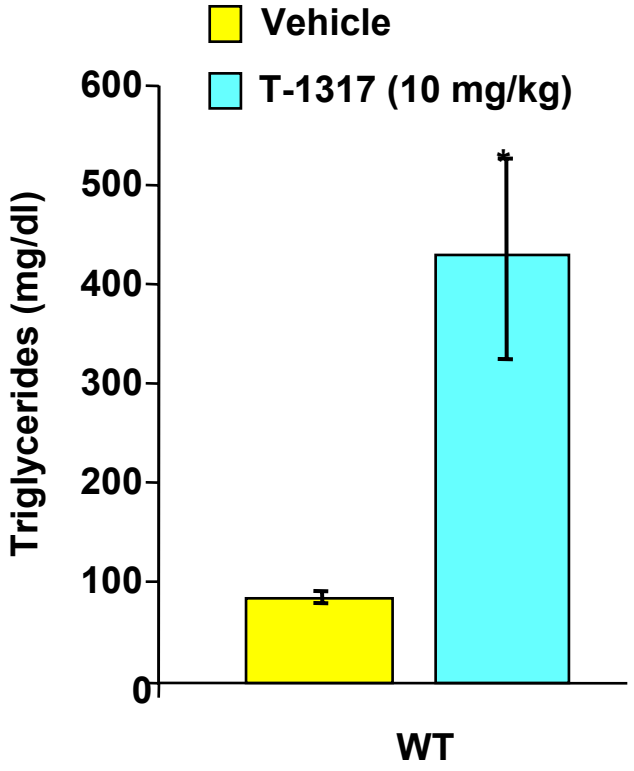
- Increase ABCA1 and reverse cholesterol transport
- Inhibit inflammation at vessel wall
- Are anti-atherogenic in established lesions

Challenge for an LXR Drug is to Maintain Anti-Atherogenic Effects in Macrophage and Minimize TG elevation in Liver

First Generation LXR Agonists in Atherosclerosis Studies

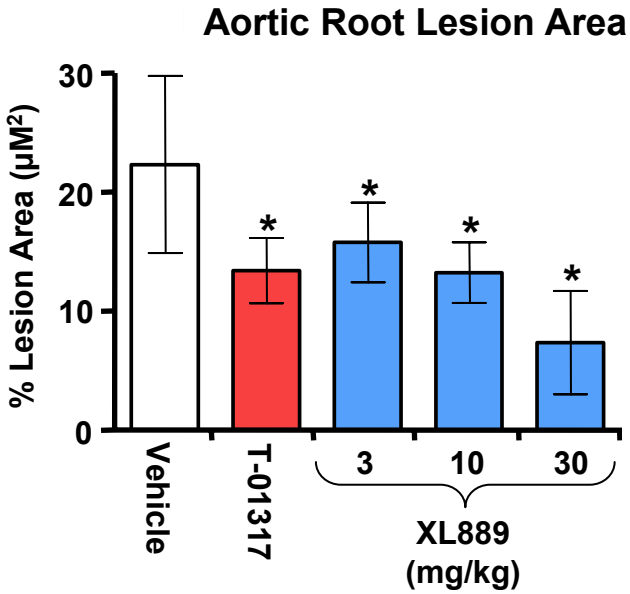
Prototype compound is efficacious in atherosclerosis model but raises serum and hepatic triglycerides

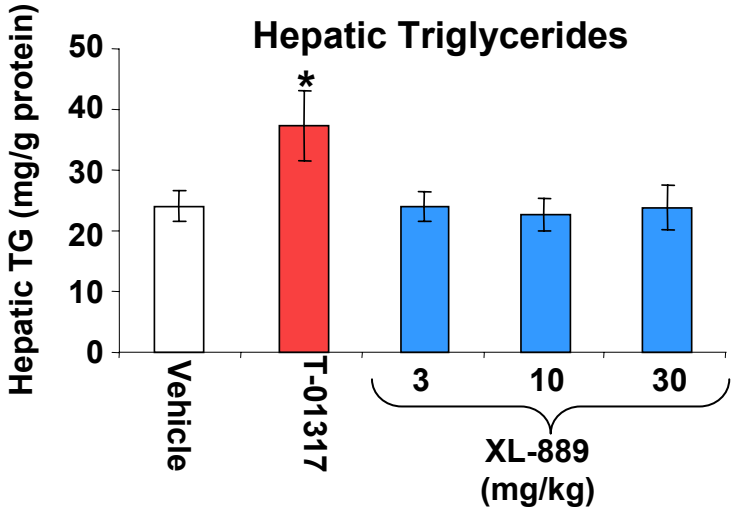




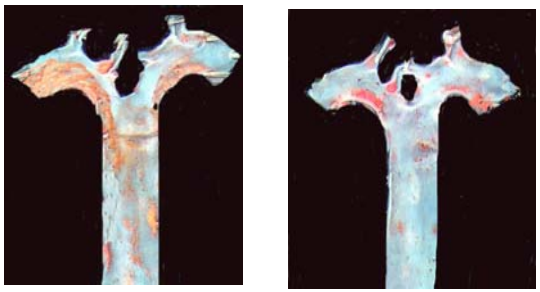
Minimize impact on LXRα in the liver while maintaining upregulation of RCT in the periphery

Exelixis LXR Agonists Show Separation of Anti-Athero Efficacy from Lipid Elevation Side-Effects





Significant Reduction in Lesion Area



No Effect on Hepatic TG

LXR DC XL652

XL652 is a potent LXR partial agonist, discovered as part of our ongoing collaboration with Bristol-Myers Squibb

XL652 shows high LXRβ binding affinity and potent cellular activity in multiple target assays

XL652 potently induces ABCA1 in PD studies in the cynomolgus monkey

- 10X therapeutic window for ABCA1 induction vs. effects on lipids (triglyceride and LDL elevation)

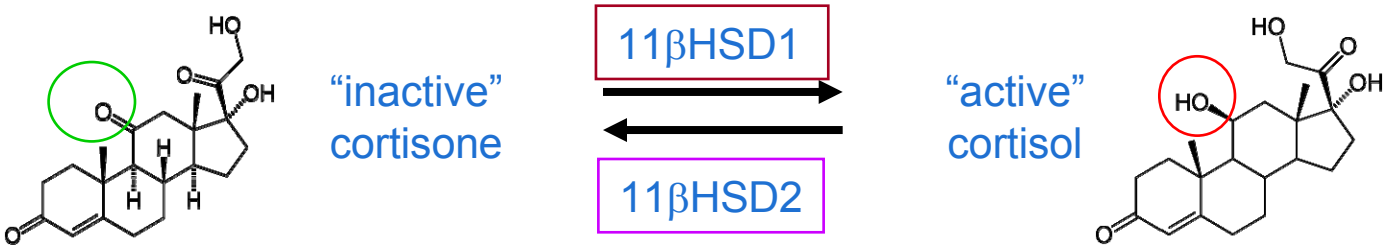
XL652 shows >70% oral bioavailability in non-clinical species

First patient dosing imminent

Advanced backup compounds identified

11βHSD Rationale

Link between stress and diabetes/obesity



Converts cortisone to cortisol in adipose tissue, liver and skeletal muscle

Promotes gluconeogenesis, insulin resistance and adiposity

11βHSD1 is upregulated 3-5 fold in obese humans

11βHSD1 Rationale

Adipose-specific overexpression of 11βHSD1 in transgenic mice results in a human type 2 diabetic phenotype

- visceral obesity
- glucose intolerance and insulin resistance
- dyslipidemia (increased TG and FFAs)

Small-molecule inhibitors of 11βHSD1 reduce intracellular cortisol levels in rodents

- Reverse multiple factors contributing to metabolic syndrome including obesity, diabetes, dyslipidemia and atherosclerosis

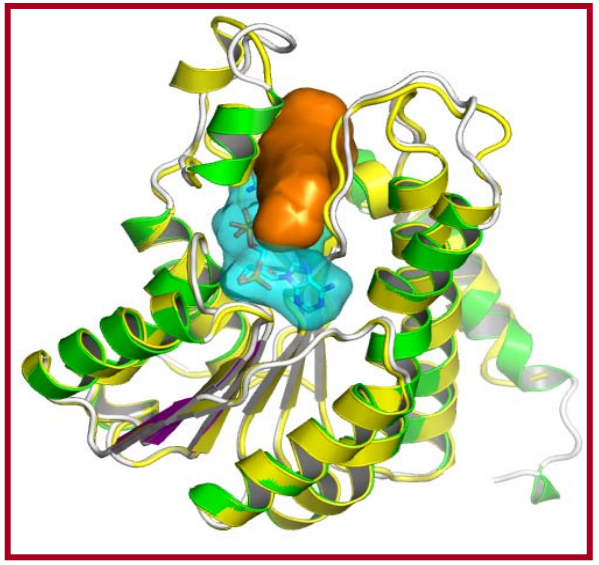
Non-specific inhibitors cause hypertension by activation of MR

- HSD inhibition by glycerrhizic acid underlies licorice-induced hypertension

11βHSD1 Program

Human 11βHSD1 preDC identified (EXEL-5465)

- Highly selective for HSD1 vs. HSD2
- Novel scaffold – backup scaffolds identified
- Excellent oral bioavailability
- Minimal interaction with CYPs



IC50s	hHSD1 nM	hHSD2 nM	mHSD1 nM
EXEL-6343	10	>10000	14
EXEL-5465	15	>10000	497

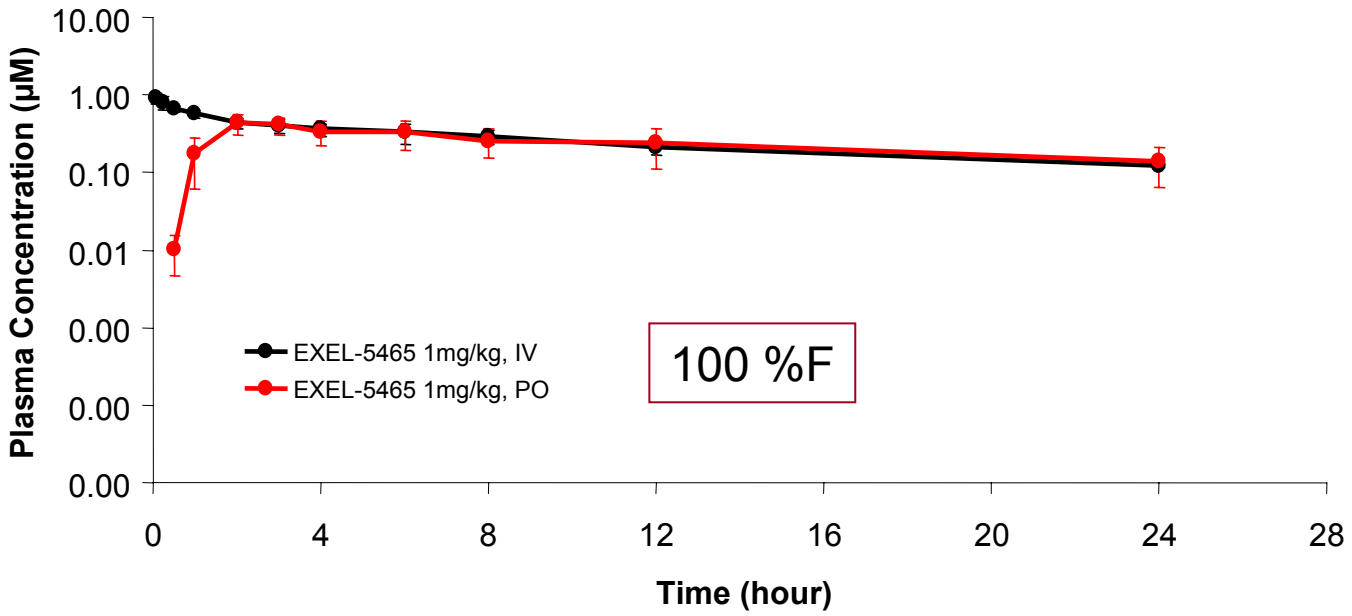
Structure of lead scaffold in mHSD1 solved at 2.6 A

Lead scaffold binds to enzyme-NADPH complex

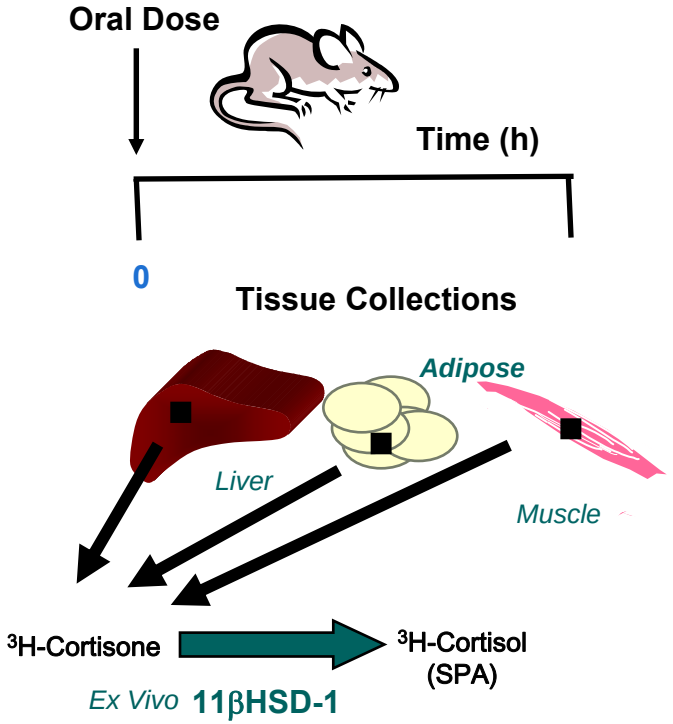
11βHSD1 Program

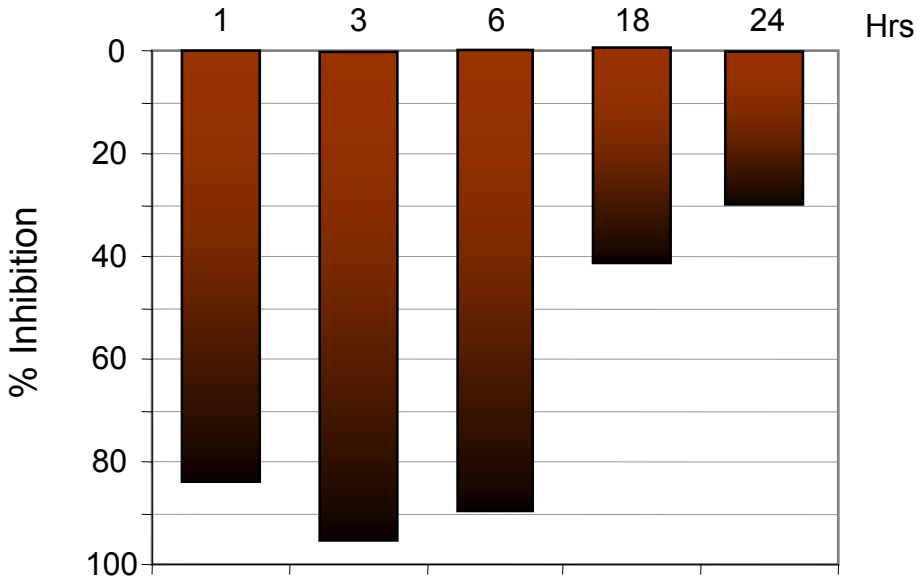
EXEL-5465 has excellent pharmacokinetic properties in rodent and non-rodent species

- High oral bioavailability
- Good exposure
- Half life compatible with once daily dosing



EXEL-6343 Ex-Vivo HSD1 Inhibition
Time-course in WAT (White Adipose Tissue)





Potent and sustained inhibition following a single dose of EXEL-6343

11βHSD1 Program Summary

EXEL-5465 is a novel, potent HSD1 inhibitor

- Highly selective for HSD1
- Inhibitory potency and activity similar to or better than published compounds

No significant interaction with CYPs or hERG

Excellent PK

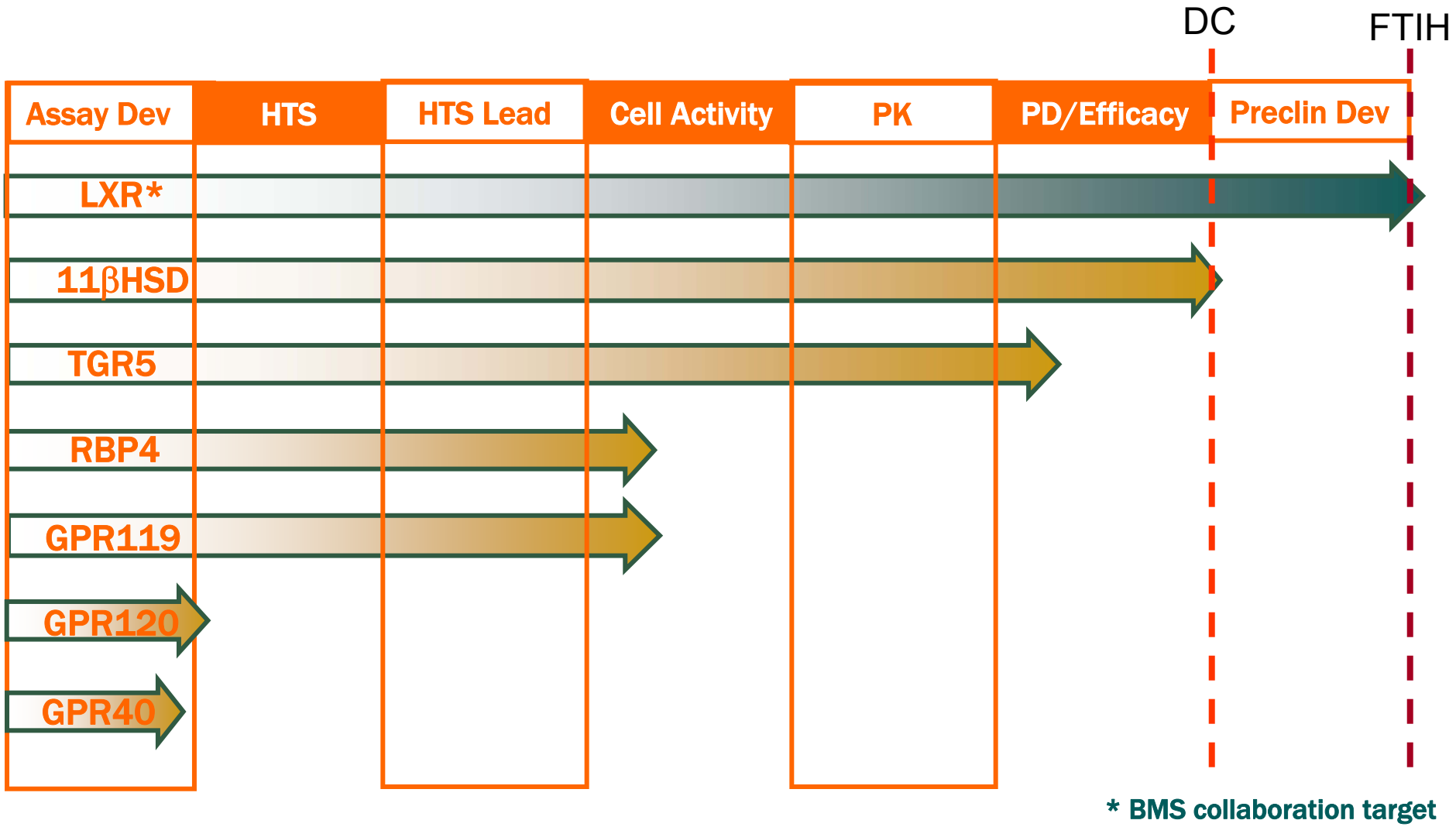
- High oral bioavailability
- Good distribution of compound into target tissue

Lead series demonstrates potent inhibition of liver, fat and muscle HSD1 following a single oral dose

- Extended duration of action: inhibition observed up to 18 h

EXEL-5465 is a pre-DC, DC nomination 1Q08

Discovery Pipeline – Metabolic Disease



* BMS collaboration target

Discovery Summary

Highly productive and flexible Discovery platform

- Success optimizing DCs against multiple target classes (kinases, NHRs, enzymes)
- Oncology and metabolic disease

Three new DCs advanced in 2007, advanced lead op programs will provide additional DCs in 2008

- Robust early stage pipelines in oncology and metabolic disease and increased target validation capabilities will provide continued flow of DCs
- Growth / replenishment of Exelixis development pipeline
- Additional partnering opportunities

Expanded capabilities for Translational Medicine support

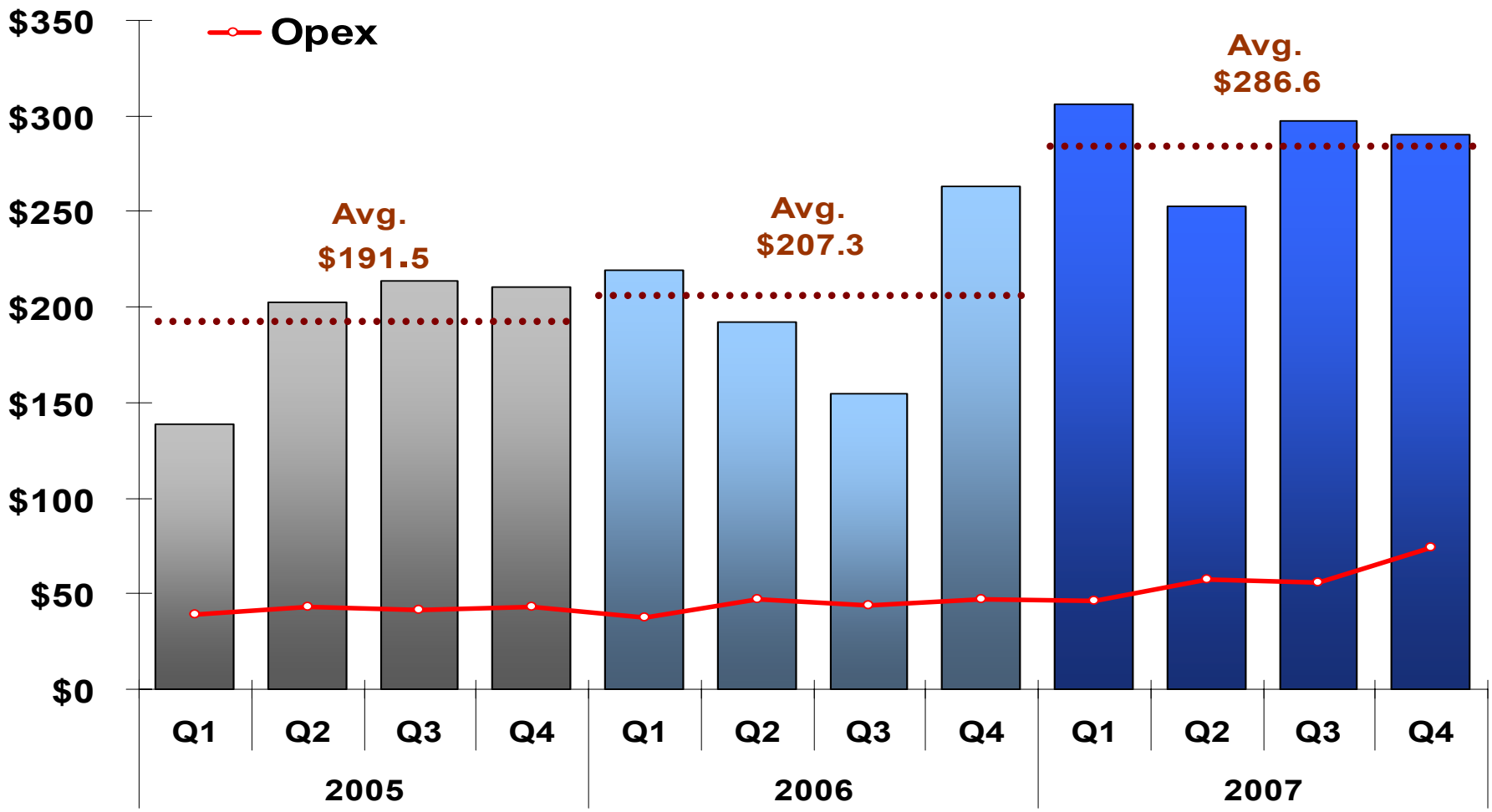
- Tumor genotyping, plasma biomarkers, tumor and surrogate tissue target modulation

Financial Overview
Frank Karbe, EVP & CFO

Finance Topics

1. Review of Financing History
2. Outlook for 2008
3. Update on Collaborations

Quarterly Cash Balance
2005 to 2007 *($ in MM)*



Cash Balance & Estimated Burn

($ in MM)

EXELIXIS

	Actual		Forecast
	2005	2006	2007
Beginning Balance	171.2	210.5	263.2
Ending Balance	210.5	263.2	> 290.0
Difference	39.3	52.7	> 26.9
Equity Proceeds	(49.6)	(90.5)	(71.9)
Net Burn	(10.3)	(37.8)	< (45.0)

Financing History – 4 Pillar Strategy
2005 to 2007 *($ in MM)*

	Total Cash Raised	2005	2006	2007	Total	(%)
❶	Existing Partnerships:	63	37	44	144	23%
❷	New Partnerships:	35	151	-	185	30%
❸	Clinical Financing:	40	40	-	80	13%
❹	Equity Markets:	50	91	72	212	34%
	TOTAL	187	318	116	621	100%
	Divestitures:	-	-	*38*	*38*	
	Total Cash Raised	187	318	154	659	

Exelixis Funding Outlook – Summary

Healthy Cash Balance

- > $340M in cash and committed funding at Year-End 2007
- > $1Bn in contingent funding

Potential substantial milestones from collaborations in 2008

- BMS - Oncology
- Genentech (MEK) - Oncology
- GSK - Oncology
- Sankyo (MR) - Metabolism

Multiple high value assets for potential new collaborations

- XL647
- XL765 & XL147
- XL019
- XL784
- New DCs (Oncology & Metabolism)

Financial Outlook 2007 & 2008

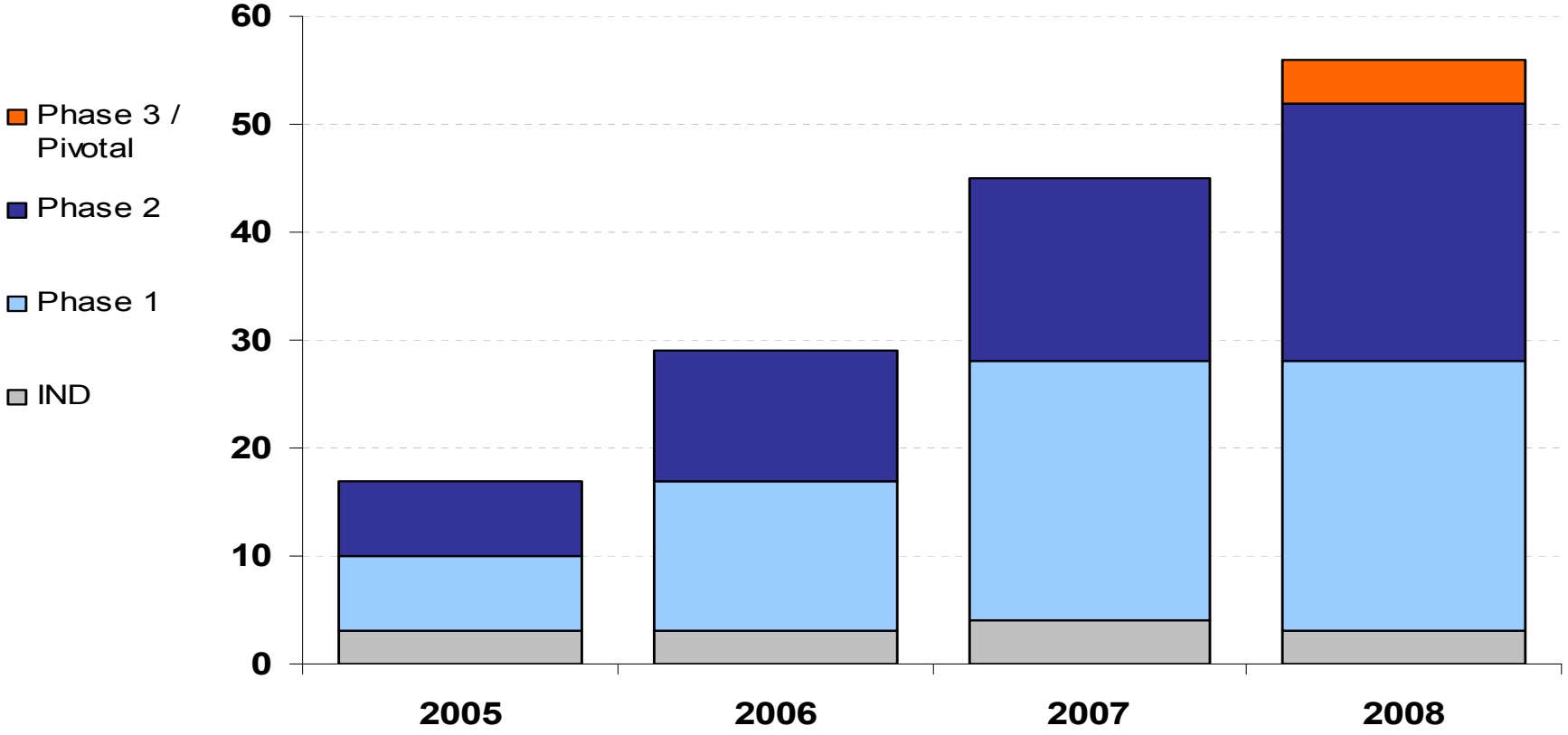
($ in MM)

	2007	2008	
	Guidance	Outlook	Comments
Revenue	**110 - 120**	↑	**Milestones** **New Business Development**
Discovery *Development* *G&A*		→ ↑ →	
Operating Expense	**260 - 290**	↑	**Driven by Development**
Cash	**> 290**		

- 2007: Revenue & Opex Guidance Confirmed; Cash Guidance Increased
- 2008: Financial Guidance to be provided at Q4 Earnings Call

R&D Productivity

Number of Trials per Phase per Year



R&D Expense *	**$135.5**	**$166.8**	**$219.0**	**TBD**

*** Excludes Artemis & Exelixis Plant Sciences**

Managing Burn

Focusing Our Efforts

- Discontinued XL999 and XL418
- Divested non-core assets
- Reviewing options for XL784

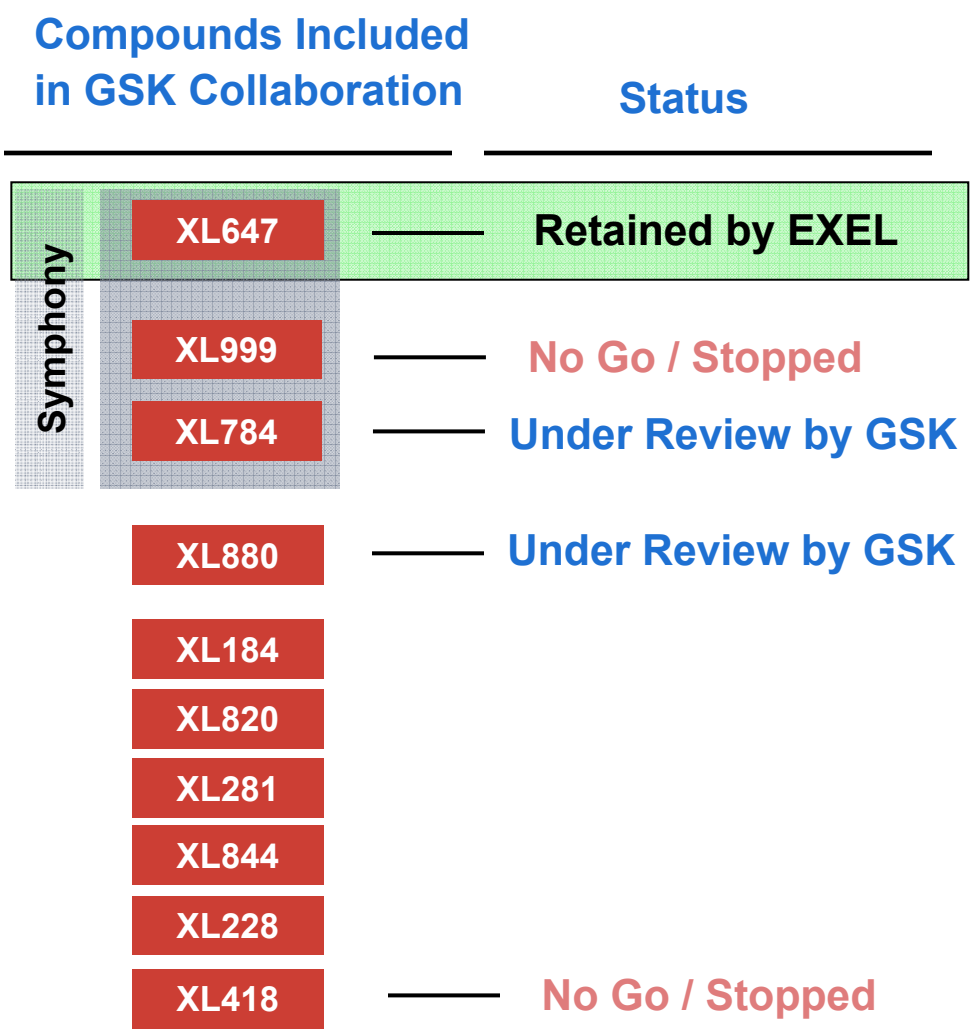
Leverage Partnering Strategy

- Expect multiple opt-in decisions in 2008
  - Receive milestones
  - Off-load future development expenses
- Continue selective partnering

Financial Discipline

- Transparency / insight
- Informed / smart decision making

GSK Collaboration
Overview and Status Update



GSK has right to select two compounds; all others stay with EXEL

- **GSK could obtain 3rd pick if collaboration were extended**
- **Extension triggers additional payments to EXEL**

GSK decisions at PoC

- **PoC defined as either a pre-determined level of activity or**
- **Completion of a pre-determined body of work**

Substantial downstream economics to EXEL if GSK selects

- **Milestones**
- **Double digit royalties**
- **Co-promotion rights**

GSK Selection Milestones

($ in MM)

EXELIXIS

Contract Year 6 through Oct '08

# of Picked	Milestones
1	35.0
2	55.0
3	75.0
Total Milestones	**165.0**

- First milestone is reduced by $36M to pay off 2005 milestone advance → XL880 milestone would be cash and revenue neutral
- Milestones on Non-Symphony compounds are applied towards paying down the GSK loan
- Milestones on Symphony compounds are increased by 25% and are not applied towards paying down the GSK loan

GSK Loan

($ in MM)

- Total Loan Principal Amount equals $85M
  - Accrues 4% interest per annum
  - Accrued interest YE 2007 totals $13.6M
- Loan matures in 3 tranches beginning one year after the collaboration ends
  - Currently first payment due Oct ’09 but could be later if GSK extends collaboration
  - GSK selection milestone for non-Symphony Compounds are applied towards loan
  - Remaining amounts are repayable in cash or stock at EXEL’s discretion

Collaborations Review

BMS – 3 IND Ready Compounds

- $20M milestone per compound at opt-in ($60M received at signing)
- Share clinical world wide development cost 65% (BMS) 35% (EXEL)
- 50/50 US profit share; Royalties and milestones ex US

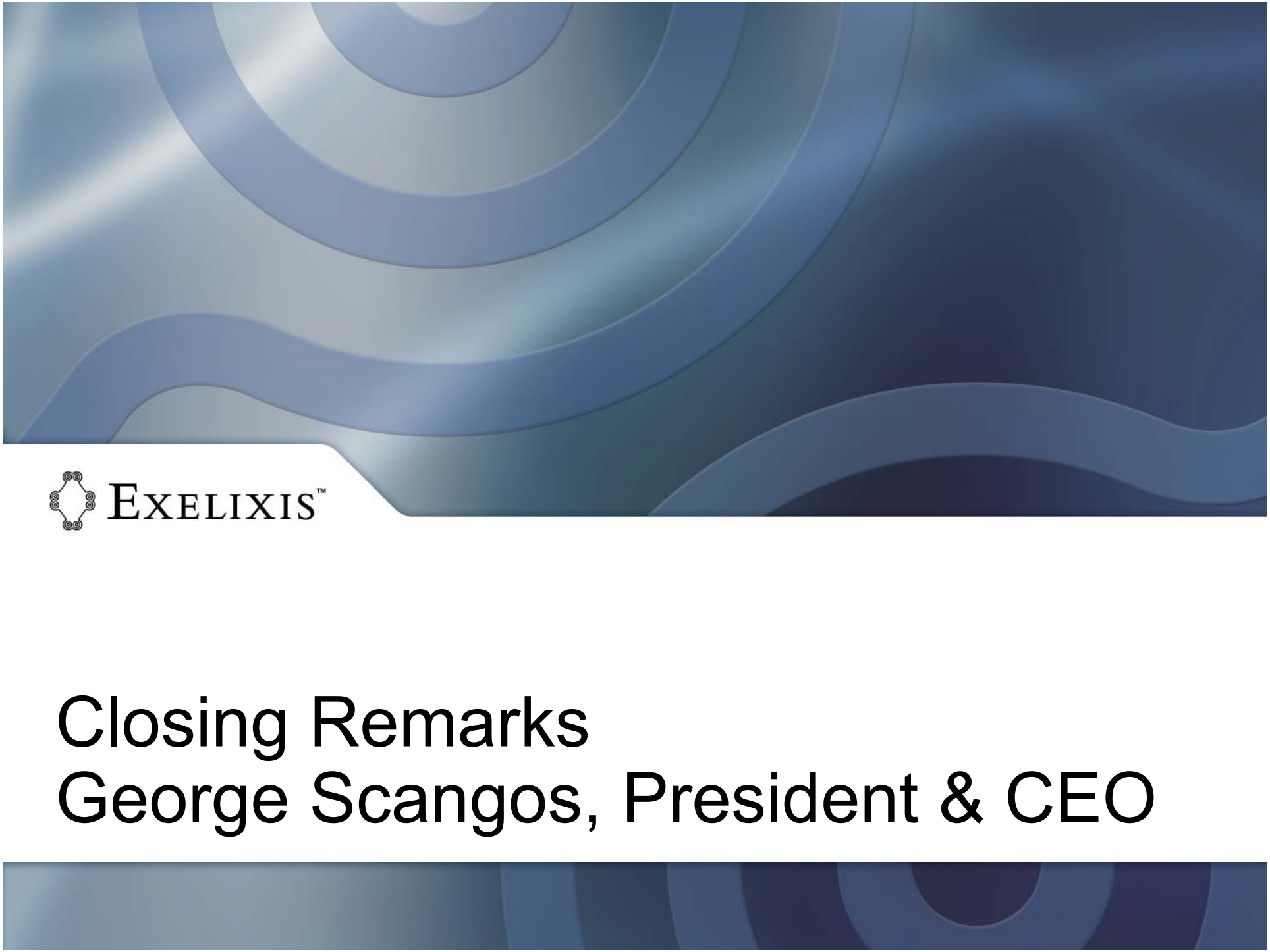
Genentech – XL518 (MEK)

- Milestone at opt-in ($40M received at signing)
- Genentech to pay all development costs post opt-in
- 30 – 50% US profit share; Double digit royalties ex US

GSK – Up to 3 Compounds at PoC

- Opt-in milestones
- GSK to pay all costs post opt-in
- Double digit royalties, milestones and co-promotion rights for North America

Up to Five Opt-in Decisions in 2008



Closing Remarks
George Scangos, President & CEO

Exelixis 2007 Accomplishments

XL880 (Met, VEGFR)

- Positive phase 2 data in PRC
- Submitted POC package to GSK early, in September

XL184 (Met, VEGFR, Ret)

- Impressive data in medullary thyroid cancer
- On track for NSCLC phase 2 by Q1 2008

XL647 (EGFR, Her2, VEGFR)

- Positive phase 2 data in first line NSCLC
- Retained rights post GSK decision

XL147 (PI3K), XL765 (PI3K, mTOR)

- First clinical data for PI3K inhibitors
- PD markers indicative of target modulation
- Potential blockbuster drugs

XL019 (JAK2)

- Phase 1 data (ASH, 12/10)
- Leading JAK2 inhibitor
- Rapid, low-risk route to market

Exelixis 2007 Accomplishments

5 Additional phase 1 programs moved forward

- XL844 (Chk1, Chk2)
- XL518 (MEK)
- XL281 (Raf)
- XL228 (IGF1R, Abl, Src)
- XL418 (Akt, S6K)

XL784 (Adam 10, MMP2)

- Completed phase 2 study in patients with diabetic nephropathy
- Missed end-point, considering options

Filed 4 INDs

Advanced 5 compounds into development

Exelixis Partnership Accomplishments

3 POC packages submitted to GSK

- XL647 rights to Exelixis
- XL880 decision by mid-December
- XL784 decision by January

Significant progress in XL518 collaboration with Genentech

BMS initiated clinical studies on XL652 (LXR agonist)

Wyeth initiated clinical studies on XL335 (FXR agonist)

Exelixis 2007 Business Accomplishments

Signed newest collaboration with BMS

- $60 million upfront
- $60 million in near-term milestones
- 50% ownership of 3 compounds

Divested majority of assets of Exelixis Plant Sciences

- Total deal worth up to $60 million
- $22.5 million guaranteed ($18.5 mm in 2007)

Sold 80.1% of Artemis

- $20 million upfront
- Retained preferred customer status and 19.9%

$70 million equity financing

Three POC compounds presented to GSK

- XL647 - rights retained by Exelixis
- XL880 – decision in mid-December
- XL784 - decision in January

Managed cash

- Year end 06 cash $260 million
- Year end 07 cash >$290 million

Pipeline Decisions

Stop development of XL999

- Mechanism-related toxicity

Focused PI3K pathway assets on XL765 and XL147

- Stop development of XL418 - low exposure after oral dosing
- XL765 and XL147 look encouraging in phase 1

Hold development XL784

- Failed to meet primary endpoint, but encouraging data
- Awaiting GSK decision
- Exploring options
- No near-term expenditures

Spend resources on other compounds in pipeline with excellent data sets

- XL647, XL765, XL147, XL019, XL184, XL820, XL844, XL518, XL281, XL228
- New compounds into development
- Balance partnered and wholly-owned compounds

Exelixis Assets

"You have partnered away all your assets and have given away the future of the company"

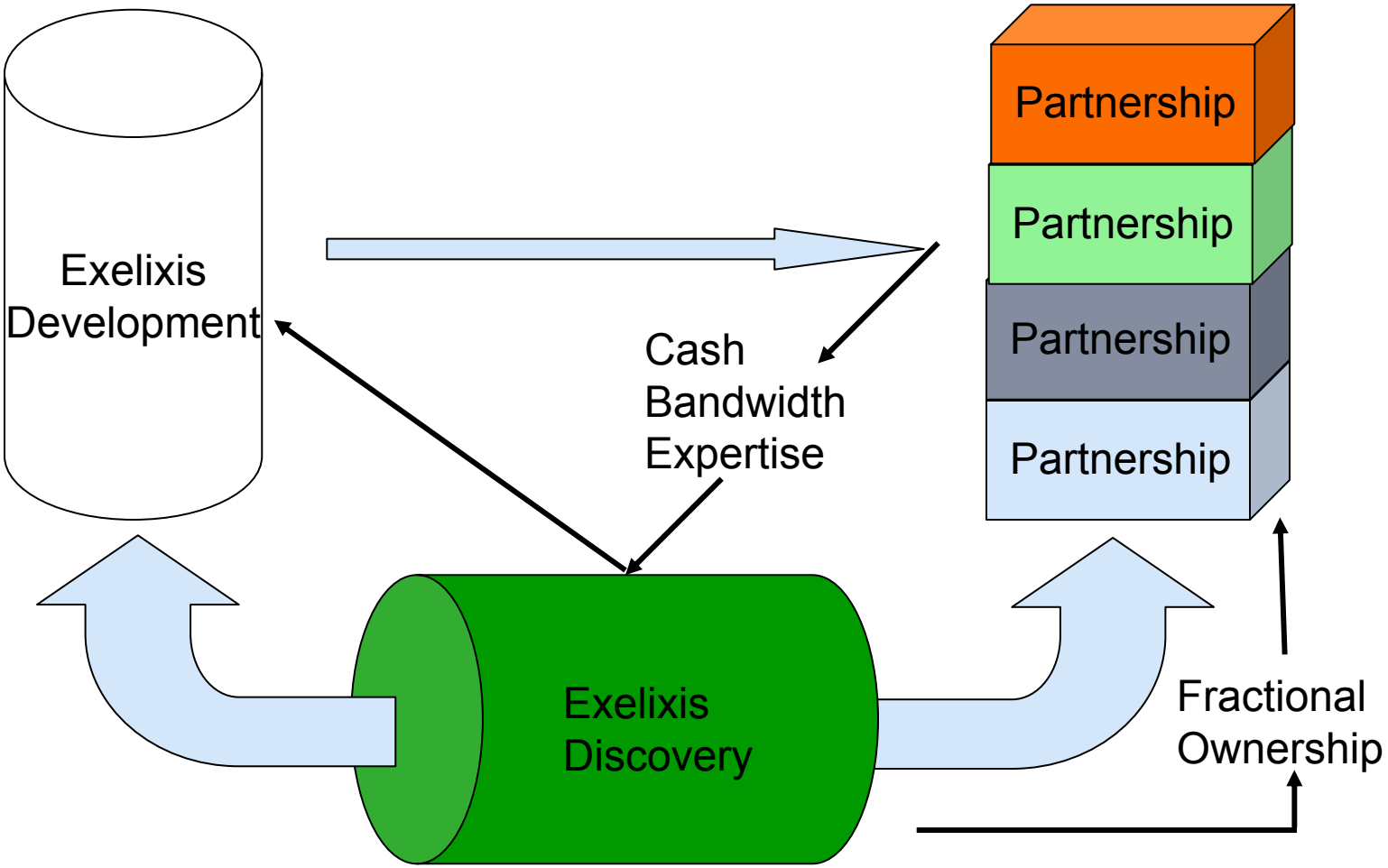
We have a lot of proprietary assets:
XL647, XL147, XL765, XL019, XL139, XL888, GSK compounds, DCs

"You have so many assets, how are you going to prioritize and pay for them all?"

We have done a lot of partnering/licensing:
GSK, BMS metabolism, BMS oncology, Genentech MEK, Genentech Notch, Wyeth, Sankyo. Others in discussion.

We have a lot of assets.
We can partner many compounds and retain many others.

Exelixis Compound Strategy



Large Drug Discovery Capacity

Dynamic pipeline: Ability to cull out weak compounds and invest resources in high potential compounds

Short term cash: Partnerships bring more cash than the actual cost of assets and offset cost of proprietary programs

Long term revenues: Fractional ownership of compounds that we otherwise wouldn't have

Bandwidth: Transferring clinical development responsibility to partner frees up internal resources

Expertise: Many pharma companies have substantial expertise in oncology development and translational medicine

Large discovery capacity does not increase cash utilization and allows Exelixis to retain fractional ownership of compounds that it otherwise wouldn't have.

Exelixis Cash Management

Year	Beginning Cash	Equity Offering Proceeds	Ending Cash	Cash Difference
2004	$242	$0	$171	
2005	$171	$50	$210	-$10
2006	$210	$90	$263	-$37
2007	$263	$72	>$290	<-$45

Ownership of Exelixis Assets

Compound	Projected Phase by mid 08	Ownership
XL647	2/3	97% (subject to Symphony)
XL765	1/2	100%
XL147	1/2	100%
XL019	2/3	100%
XL518	1/2	100% (30-50% if Genentech opts in)
XL880	2	Up to 3 subject to milestones and royalties if GSK opts in; balance 100% (XL784 subject to Symphony)
XL784	2	
XL184	2	
XL820	2	
XL218	1	
XL281	1	
XL844	1	
XL652	1	Milestones, double digit royalties
XL335	1	Milestones, double digit royalties

Symphony

Symphony provided $80 million to finance XL999, XL784, XL647

- $40 million in mid 05
- $40 million in mid 06
- In return for cash, IP on the compounds was transferred to Symphony
- Exelixis can reacquire compounds by repaying $80 million plus 25% IRR
- XL647 is main driver of value today

At end of Q3, $38 million left in vehicle

- Expect to use for advancement of XL647
- Phase 2 daily dosing first line NSCLC trial
- Phase 2 trial in patients who responded to and then failed another EGFR antagonist
- Data available mid-year 2008

Partnering discussions ongoing

- Positive clinical data will increase upfront value (especially relapsed EGF-antagonist responders)
- Upfront payment can be applied to Symphony

Risk remains with Symphony

- XL647 looks good, but this is drug development after all
- Unexpected compound failure will be at Symphony's expense

Exelixis Development Portfolio in 2008: Potential Pivotal Trials

XL647	→	Non Small Cell Lung Cancer (NSCLC)
XL784		
XL880		
XL184	→	Medullary Thyroid Cancer (MTC)
XL820		
XL518		
XL281		
XL844		
XL228		
XL999		
XL147		
XL765		
XL418		
XL019	→	Myelofibrosis (MF)

The compounds XL647, XL784 and XL999 have been out-licensed to Symphony Evolution, Inc. and are subject to a repurchase option. Pursuant to a product development and commercialization agreement between Exelixis and GlaxoSmithKline, GlaxoSmithKline has the option, after completion of clinical proof-of-concept by Exelixis, to elect to develop up to three compounds in Exelixis' product pipeline, including XL784 and XL999, but excluding XL647, XL518, XL147, XL765 and XL019. Finally, the compound XL518 is the subject of a co-development collaboration between Genentech and Exelixis.

Exelixis Development Portfolio in 2008: Potential Phase 2 Compounds

XL647	Breast, GBM, H&N
XL784	
XL880	PRC, Gastric, H&N
XL184	NSCLC, GBM
XL820	GIST, Melanoma, AML
XL518	
XL281	Solid tumor (potentially CRC, NSCLC, melanoma)
XL844	Pancreatic + Gem
XL228	CML and/or solid tumor
XL999	
XL147	Single Agent & Combination Phase 2 with Chemotherapeutics & Targeted Agents
XL765	
XL418	
XL019	Polycythemia Vera

The compounds XL647, XL784 and XL999 have been out-licensed to Symphony Evolution, Inc. and are subject to a repurchase option. Pursuant to a product development and commercialization agreement between Exelixis and GlaxoSmithKline, GlaxoSmithKline has the option, after completion of clinical proof-of-concept by Exelixis, to elect to develop up to three compounds in Exelixis' product pipeline, including XL784 and XL999, but excluding XL647, XL518, XL147, XL765 and XL019. Finally, the compound XL518 is the subject of a co-development collaboration between Genentech and Exelixis.

Exelixis 2008 Goals

Opt-in decisions from GSK, BMS, Genentech

Additional substantial partnership(s)

Three new INDs

Three new Development Candidates

Build out commercial infrastructure

Manage cash, maximize output/dollar

Exelixis Senior Management Q&A Panel

George Scangos, PhD, CEO

Michael Morrissey, PhD, President R&D

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Gisela Schwab, MD, SVP and CMO

Peter Lamb, PhD, SVP and CSO

EXELIXIS™

Exelixis 3rd Annual R&D Day

December 5th 2007

Mandarin Oriental Hotel

New York, NY